DEFINITION

Faecal incontinence has been previously defined as ‘the involuntary or inappropriate passage of faeces’. This definition, however, is considered to be incomplete as it does not include incontinence to flatus. Therefore many adopt the term anal incontinence to include flatus. The current recommended definition of anal incontinence is the involuntary loss of flatus, liquid or solid stool that is a social or hygienic problem.

PREVALENCE OF ANAL INCONTINENCE

As a result of inconsistencies in definition and cohort bias, the precise prevalence of reported anal incontinence remains to be established. The estimated community prevalence of faecal incontinence is 4.2 per 1000 in men aged 15–64 years, 10.9 in men aged 65 or over, 1.7 per 1000 in women aged 15–64 and 13.3 per 1000 in women aged 65 or over. In residential homes for the elderly, the prevalence was found to be 10.3% but may approach 60% in the elderly. Although the incidence of faecal incontinence in 45-year-old women is eight times higher than in men of the same age, its prevalence has not been accurately assessed in the younger age groups. Even a minor degree of faecal incontinence can be very distressing and is a cause of great embarrassment and therefore very few admit to it and seek medical assistance. Leigh and Turnberg found that half the patients referred to a gastro-intestinal clinic complaining of diarrhoea were incontinent but fewer than half of them volunteered this information. Moreover clinicians may also not inquire or document this symptom and therefore the true prevalence of faecal incontinence can be underestimated. Unfortunately, little data are available about the community prevalence of anal incontinence (including flatus) but one study in Wisconsin, USA reported an incidence of 2.2%.

ANATOMY

The puborectalis and the external anal sphincter (EAS)

The levator ani muscle (pelvic floor) is often described as a funnel shaped structure but in vivo imaging has revealed that it is in fact more horizontal. It is subdivided into the pubococcygeus, ileococcygeus and coccygeus. The pubococcygeus arises anteriorly from the posterior aspect of the pubic bone and from the anterior portion of the arcus tendineus (white line) which is a condensation of the obturator internus fascia. The most medial fibres of the pubococcygeus that pass round the rectum at the anorectal junction form the puborectalis muscle.

Proximally the EAS lies in contiguity with the posterior half of the puborectalis (Fig. 1); distally it merges with the perianal skin. Like the levator ani it is primarily composed of striated muscle. There is lack of consistency in the literature with regard to the structural subdivisions of the EAS. Some authors describe it as one structure while others have subdivided it into two or even three components: the subcutaneous and deep EAS as annular muscles not attached to the coccyx and the superficial EAS (middle layer) as being elliptical with fibres running antero-posteriorly from the perineal body to the coccyx and the ano-coccygeal raphe. The deep EAS has anterior fibres that crossover to the opposite side and combine with the superficial transverse perinei attaching to the ascending ramus of the ischium. As these subdivisions are not identified during surgery they do not appear to be clinically relevant. However, a clear understanding of normal anatomy and variants are important during imaging (ultrasound and magnetic resonance imaging) of the anal sphincter in order to avoid misinterpretation. The motor supply of the puborectalis is via direct branches of the sacral nerves (S3 and S4) and that of the EAS is via the pudendal nerve (S2, S3, S4). This supports the contention that the puborectalis is part of the levator ani and separate from the anal sphincter. Nevertheless, close cohesion between these muscles would seem to be essential because without it peristalsis would pull the rectum upwards and over its contents without expelling them through the anus.
The longitudinal muscle (LM)

The LM of the anal canal is a direct continuation of the smooth LM of the rectum. Between the lower border of the internal anal sphincter and the upper border of the subcutaneous EAS it attaches to the anal skin to form the anal intermuscular septum. Many of the fibres then pass outwards traversing the subcutaneous EAS to insert into the perianal skin to form the ‘corrugator cutis ani’. The functional significance of this muscle is unknown.

The internal anal sphincter (IAS)

The IAS is continuous with the inner circular smooth muscle of the rectum and terminates in a sharply defined thickened rounded lower margin, separated from the subcutaneous EAS by the anal intermuscular septum. The IAS measures about 3 cm in length and 5 mm in thickness. The IAS is tonically active and under autonomic control; namely, excitatory (sympathetic L1-L2) and inhibitory (parasympathetic S2-S4).

MECHANISM OF DEFECATION

The main reservoir for faeces is the transverse colon and the rectum is usually empty. The defecation cycle begins when the sigmoid colon becomes distended and contractions are initiated and the stool is propelled into the rectum. Depending on various factors such as gut motility and stool consistency colonic contents are delivered at a variable rate to the rectum. The first sensation of rectal filling occurs at a volume of 50 mL, with a maximum tolerated volume of 200 mL. Unlike the anal canal, the rectum does not have rich sensory innervation. Although individuals experience a perception of rectal distension, this sensation persists in patients who have an ileal pouch created following rectal excision, supporting the hypothesis that there are external receptors probably in the pelvic floor.

Following rectal distension either with faeces or flatus, the internal sphincter relaxes to allow sampling of rectal contents to take place by the specialised sensory epithelium of the anal canal (Fig. 2). This relaxation is mediated via myenteric connections modulated by the autonomic nervous system.
nervous system and is known as the rectoanal inhibitory reflex. If it is socially convenient, the puborectalis and external sphincter relax and evacuation occurs. However, in order for evacuation to take place, the pressure within the rectum must exceed that within the anal canal. This is achieved by increasing the intra-abdominal pressure and in effect performing the Valsalva manoeuvre. Relaxation of the puborectalis also allows the anorectal angle to increase from about 90° up to 140°. 2 If the time for evacuation is inappropriate, voluntary EAS contraction extends the period of continence to allow the compliance mechanisms within the colon to make adjustments in order to accommodate the increased rectal volume. Thereafter the stretch receptors are no longer activated and afferent stimuli are abolished together and the sensation of faecal urgency subsides.

ANAL CONTINENCE MECHANISMS

Anal continence involves complex integration between sensory input and somatic and visceral muscle function. It is affected by various factors such as mental function, lack of a compliant rectal reservoir, changes in stool consistency and volume, diminished anorectal sensation and enhanced colonic transit. However, anal continence can be maintained as long as anal pressure exceeds rectal pressure and the ultimate barrier to rectal contents is provided by the puborectalis sling and anal sphincters (Fig. 2). An increased volume of liquid stool coupled with rapid colonic transit may overwhelm the compliant rectal reservoir. Therefore if the rectum is able to function effectively as a rectal reservoir and in the presence of normal stool consistency, faecal incontinence can usually be attributed to defective function of the anal sphincter complex. The physiological role of various components of this complex in maintaining continence will be considered separately.

THE PUBORECTALIS MUSCLE AND THE ANORECTAL ANGLE

The anorectal angle is formed by the anteriorly directed pull of the puborectalis. The angle varies from 60° to 105° at rest and during defecation the angle straightens allowing the rectum to empty.

Two theories have been proposed to explain how the anatomical angulation at the anorectal junction may contribute to maintain continence. The first is the ‘flutter’ valve, which is created as the rectum passes through the slit-like aperture in the pelvic floor caused by the forward pull of the puborectalis; a rise in intra-abdominal pressure would create a high pressure zone and result in apposition of the rectal walls at the anorectal angle. The second is the flap-valve theory in which contraction of the puborectalis creates an acute anorectal angle and intra-abdominal forces compress the anterior rectal wall against the upper anal canal. However, both these theories have lost credibility; for a flutter valve to produce such a high pressure zone, intra-abdominal forces would have to be applied below the pelvic floor and radiographic studies have failed to demonstrate a flap valve. Moreover, both theories would account for rectal pressures in excess of anal canal pressures without evacuation of rectal contents. As rectal pressures have been shown to be consistently lower than anal pressures in healthy subjects, continence must be sphincteric and not valvular.

The puborectalis is considered by some to be the most important muscle in maintaining continence. In children with congenital anomalies and absence of the anal sphincter a high degree of continence can be maintained with the puborectalis. However, posterior division of the puborectalis in the treatment of chronic constipation made no difference to the anorectal angle and was not associated with incontinence of solid stool. Furthermore, following successful postanal repair for faecal incontinence no significant change was observed in the anorectal angle. The role of the anorectal angle in maintaining continence therefore remains controversial.

The puborectalis muscle functions in concert with the external anal sphincter and it is probable that if damage occurs to one muscle the other may compensate functionally. Faecal incontinence may ensue if in addition, other factors in the continence mechanism (see below) are compromised, or if the remaining muscle cannot compensate adequately.

IAS

There are conflicting opinions on the role of the IAS in maintaining continence. As 70% of the resting tone is contributed by the IAS it is a major contributory factor in keeping the anal canal closed at rest. This is supported by the finding that symptoms of incontinence can develop in up to 40% following lateral internal anal sphincterotomy. Faecal incontinence has also been reported following anal dilatation with sonographic evidence of internal sphincter disruption and a reduced resting pressure.

Ultrastructural changes have been identified in the morphology of the internal sphincter of patients suffering from neurogenic faecal incontinence. Although these changes are probably not the primary cause of faecal incontinence, they may have some relevance to IAS function. In addition, abnormalities of adrenergic innervation with a diminished sensitivity of the IAS to α adrenergic agents in vitro have been demonstrated in patients with idiopathic faecal incontinence. These changes could be attributed to an intrinsic degeneration of the muscle and its receptors or to simultaneous direct injury to the striated muscle of the pelvic floor.
THE EAS

The EAS is inseparable from the puborectalis posteriorly and both muscles appear to function as a single unit electrophysiologically. It has been shown that while contraction of the puborectalis accentuates the anorectal angle, it does not increase the intraluminal pressure of the anal canal.

The EAS, similar to the IAS, is in a state of tonic contraction even at rest and the activity is reflexly raised when intra-abdominal pressure is increased, e.g. when coughing, laughing or lifting. Activity is maximally raised when the EAS is contracted voluntarily but contraction can only be maintained for 1–2 min. Stimulation of the perianal skin also results in a reflex EAS contraction via the pudendal nerve called the cutaneo-anal reflex. Electrical activity usually decreases during straining and when defecation is attempted, although a variable response was seen in some subjects.

The EAS contributes up to 30% of the resting pressure and the increment of the squeeze pressure above the resting pressure reflects predominantly EAS function. The maintenance of tone is, however, also dependent on a sensory input as it is lost if the sensory roots are destroyed, e.g. tabes dorsalis.

THE ANAL CUSHIONS

The anal cushions consisting of epithelium, subepithelium and the underlying haemorrhoidal plexuses can contribute up to 15% of resting pressure. The anal sphincters cannot obliterate the lumen completely without the sealing effect of the anal cushions. The thickened cushions may account for the increased resting pressures seen in patients with haemorrhoids. The fall in resting pressure following haemorrhoidectomy might explain the development of minor anal incontinence although inadvertent damage to the sphincter, particularly the internal sphincter, has been observed using anal endosonography.

RECTAL COMPLIANCE

A compliant rectal reservoir that can accommodate large volumes of stool without significant increases in pressure is an important prerequisite for the effective function of barrier mechanisms of continence. Patients who have a reduction in rectal capacity as occurs in colitis and radiation proctitis often suffer from faecal urgency and incontinence.

ANORECTAL SENSATION

The epithelium of the anal canal is richly supplied with sensory nerve endings exquisitely sensitive to pain, heat and cold. The afferent nerve pathways for anal canal sensation is via the posterior inferior haemorrhoidal branches of pudendal nerve and anterior haemorrhoidal branches of the perineal nerve to the sacral roots of S2, S3 and S4, but in addition direct anal and urethral branches arise from S4 and S5. Sampling has been shown to occur less frequently in incontinent patients compared with controls.

CENTRAL CONTROL OF CONTINENCE

The frontal cortex is important for the conscious awareness of the need to defaecate and appropriate social behaviour. The upper motor neurones for the voluntary sphincter muscles lie close to those of the lower limb musculature in the parasagittal motor cortex. They communicate by a fast conducting oligosymptomatic pathway with the Onuf nucleus situated in the sacral ventral grey matter mainly S2 and S3. Disease affecting the upper neurone motor pathway usually results in urgency and urge incontinence and provided the lower motor pathway is still intact reflex defaecation will still be possible. Neurological diseases such as multiple sclerosis, Parkinson’s disease and disorders of the spinal cord or cauda equina can be accompanied by incontinence because the central pathways that control sphincter function are located in the vicinity of the corticospinal tracts. Patients suffering with diabetes mellitus can have an autonomic neuropathy and this can also lead to faecal incontinence.

The lower motor neurones innervating the striated pelvic floor and urethral and anal sphincters arise from the Onuf nucleus. The commonest cause of a lower motor neurone lesion in the adult is chronic stretching of the pudendal nerve usually as a result of chronic straining at stool and/or childbirth. Damage to the pudendal nerve results in progressive denervation and reinnervation of the pelvic floor–anal sphincter complex causing weakness and atrophy of these muscles (Fig. 3).

![Fig. 3. The mechanism of neurogenic anal incontinence.](image-url)
PATHOPHYSIOLOGY

In general, passive soiling and/or flatus incontinence is associated with internal sphincter dysfunction whereas urgency and/or urge incontinence is associated with external sphincter dysfunction. However, there is usually dysfunction in both sphincters giving rise to mixed symptoms. The development of anal incontinence may be due either to mechanical disruption or neuropathy but sometimes both may coexist. Obstetric trauma is a major cause and there is a double peak incidence of anal incontinence; the first is in the postpartum period and the second is in the perimenopausal years. The development of anal endosonography has revolutionised our understanding of anal incontinence and it has now been demonstrated that about one-third of primiparous women develop anal sphincter injury that is not recognised during vaginal delivery. However, even when it is recognised and repaired the outcome is suboptimal as one-third continue to suffer impaired continence. Attention is now being focused on improved training in anatomy and repair techniques. However, there are other factors such as the effect of ageing, collagen weakness, progression of pelvic neuropathy, oestrogen deficiency, concurrent irritable bowel syndrome, primary degeneration of the internal anal sphincter, severe constipation and uterovaginal/rectal prolapse that may contribute to a deterioration in anorectal function. It is therefore important to perform a full assessment of anorectal investigations before surgical intervention. However, as the results of surgery are not always optimal and there is a proven deterioration over time, conservative measures should always be explored first.

NONSURGICAL MANAGEMENT OF FAECAL INCONTINENCE

Although there are increasing arrays of surgical options for faecal incontinence, which allow an improvement in symptoms in a highly selected group of patients, the first line of therapy remains conservative. Surgical options include anterior overlapping sphincter repair, neo-sphincters, stomas, ante-grade continence procedures and more recently sacral nerve stimulation. Conservative treatments often rely on a combination of pharmacological, behavioural and physical treatments, all of which may also be used as adjuncts to surgical procedures.

Pharmacological

Constipating agents are often the first line of treatment — the rationale being that solid and less frequent call to stool will be easier to control with a deficient sphincter mechanism. Loperamide has a low side-effect profile, reduces small bowel motility and stool weight and can be titrated accurately against stool consistency by the patient using the syrup form (2 mg in 10 mL). By using a bowel diary with increasing amounts of loperamide a patient can learn how to ‘manipulate their bowels rather than their bowels manipulating their lives’. Codeine phosphate in doses of 30–120 mg daily in divided doses can be used in a similar way but its higher side-effect profile includes drowsiness and addiction and this limits its use. It is sometimes useful in combination with loperamide for unresponsive diarrhoea-induced incontinence.

Occasional patients respond well to fibre supplements, if they have loose stools; the fibre increases stool bulk and sometimes improves consistency. The side-effect is an increase in wind and the patients often have worsening symptoms. Indeed a reduction of fibre, to increase the consistence of the stools and reduce flatus, is usually preferable.

If elderly patients are incontinent secondary to constipation or faecal impaction, regular use of a laxative, such as movicol or lactulose, may lead to improved continence since a normal stool is passed on a regular basis leaving the rectum relatively empty and less likely to leak in between.

An alternative pharmacological approach is to facilitate an empty rectum between controlled evacuation by the judicious use of suppositories or enemas. These are particularly helpful in patients who suffer from incomplete emptying or postdefecatory soiling. Alternative treatments include retrograde washouts with tap water or phosphate solutions with a form of anal plug or balloon to limit spillage.

The evidence for benefit from these pharmacological approaches is limited. A recent Cochrane review looked at the only four randomised trials of antidiarrhoeal treatment vs placebo for incontinence and showed that people were better on active treatment. There were fewer episodes of incontinence, urgency and use of pads and more people achieved full continence. However, patients on the active drug also reported more adverse effects such as constipation, abdominal pain, headache and nausea. The focus of these trials was the treatment of diarrhoea, rather than incontinence, and there are no studies comparing drug treatment with any other treatment modality.

The internal sphincter is a smooth muscle and receives stimulatory α-adrenergic innervation. It was therefore supposed that by using an adrenergic agonist the resting pressure and hence faecal leakage might be reduced. Patients with intact anal sphincters but passive faecal incontinence have been shown to have improvement in their symptoms and an increase in resting tone by using topical phenylephrine. However, the local adverse events (stinging and dermatitis) have restricted its use.

One final pharmacological approach to consider is the role of hormone replacement therapy in post-menopausal women. Only one small prospective study of 20 post-menopausal women with demonstrable faecal incontinence has been conducted. After 6 months of oestrogen replacement therapy, five (25%) became asymptomatic and a further 13 (65%) were symptomatically improved in terms of flatus control, urgency and faecal staining.
**Behavioural therapy**

This approach includes simple measures such as low fibre diet and caffeine exclusion and biofeedback. A recent Cochrane review\(^\text{15}\) found that although there are over 40 published studies looking at biofeedback for incontinence, there is not enough evidence to judge whether these treatments are beneficial nor who will benefit the most.

Norton et al.\(^\text{16}\) attempted to address this issue by randomising 171 patients to four groups. These included a group who were offered standard advice, those given advice and sphincter exercises, a group offered hospital-based biofeedback and a final group who had hospital biofeedback and a home electromyelogram biofeedback device too. There was no significance difference between the four groups – all of them showed improvement in continence, quality of life and anal sphincter function and in the 60% who showed improvement, this was maintained at 1 year. It looks as though the main mechanism for improvement may be due to health seeking, general advice, medication or the development of a therapeutic relationship but not the exercises or biofeedback.

**Physical treatments**

Some patients are helped by the use of anal plugs – these are inserted into the anal canal, extend and create a watertight seal (Fig. 4). Patients often find them difficult to tolerate due to a persistent feeling of ‘something’ within the anal canal or they may have problems retaining them.

Electrical stimulation has shown some promising results. A fitted electrode (Fig. 5) is inserted into the anal canal and the muscles are stimulated twice a day for 15 min. This may improve continence in up to 60% of people by increasing the squeeze pressure by 23%. However, at present there are insufficient data to allow reliable conclusions to be drawn on the effect of electrical stimulation on faecal incontinence.\(^\text{15}\)

Initially, hopeful results of sphincter augmentation using bulking agents (autologous fat, PTFE paste, gluteraldehyde cross-linked collagen) have been disappointing as the benefits wear off with time. A larger molecule (Durasphere) has recently been shown in 18 patients to significantly improve continence in the majority of patients at 6 and 12 months.\(^\text{17}\) It appears to be safe but the mean follow-up was relatively short – only 28.5 months. Kenefick et al.\(^\text{18}\) have also recently reported the use of silicone biomaterial in six patients with a median follow up of 18 months. Five of the six had symptom improvement as well as a physiological increase in resting and squeeze pressures. All studies looking at injectable bulking agents have involved few patients and have relatively short follow-up. Longer and larger trials are necessary to confirm their use in passive incontinence.

**CONCLUSIONS**

The mechanisms that maintain anal continence are complex and therefore the precise diagnosis is often difficult. It is usual practice for a number of investigations to be performed before treatment can be instituted. Nonsurgical forms of treatment should be explored aggressively as the long-term outcome of surgery may be disappointing. The mainstay of treatment is to alter stool consistency by using constipating drugs with or without an evacuatory aid. This can be combined with simple dietary exclusions (caffeine and fibre) that may also reduce bowel frequency. A few patients may find benefit with a bulking agent or laxative. Exercises/biofeedback may have some role but the use may only be the development of a patient/doctor interaction and assessment of the problem. Newer therapies such as sphincter augmentation have shown some promising results...
but each of the studies are small with poor follow-up. The focus of future research should be directed towards preventative measures to minimise the social, physical and emotional stigma associated with anal incontinence.

References