# The aetiology and epidemiology of faecal incontinence

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***Faecal incontinence is experienced by at least 2% of the population and 7% of those over 65 years of age. The true incidence is probably much higher because of the stigmata of the affliction leading to underreporting. The common causes of faecal incontinence are discussed.***

Faecal incontinence is an embarrassing and distressing condition. It interferes with personal wellbeing and may lead to social isolation. It is a condition that is 'not discussed' in polite circles. Faecal incontinence has therefore been termed the silent affliction (Johanson and Lafferty, 1996). As a consequence, the true incidence is thought to be underestimated. The reported incidence in the general population ranges from 2.2 to 7.1% (Nelson et al, 1995; Johanson and Lafferty, 1996). Faecal incontinence in residents in nursing homes is much higher and approaches 33% (Denis et al, 1992). In the United States, more than $400 million each year are spent on faecal incontinence appliances alone (Lahr, 1988).

Faecal continence is the normal state and is defined as the ability to retain solid or liquid stools or flatus not only in various positions but also during physical exercise, coughing and sneezing. Continence therefore requires intact sensation and motor innervation, and an anatomically intact sphincter complex. Failure of these continence mechanisms will lead to incontinence.

## CONTINENCE MECHANISMS

In order to appreciate the pathogenesis of incontinence, an understanding of the mechanisms that maintain continence is paramount:

1. The anorectal angle is responsible for gross continence. The puborectalis sling creates an acute angulation forward between the rectum and anal canal. At rest this is approximately 90–105°, 60–90° during voluntary contraction and opens out to 120–180° during defaecation. Incontinence may result if the angle is too wide
2. The sphincter complex is divided into an internal smooth muscle component and external striated muscle component. The internal sphincter is responsible for at least 60% of the anal resting pressure, ranging from 65–85 mmHg. The external sphincter complex reflexly responds to increases in intra-abdominal pressure and rectal distension as a voluntary defaecation delay mechanism. It results in the squeeze pressure generating an increase of at least 50–100% above resting pressure. Damage to the anatomical structure of the sphincters or nerve supply will result in incontinence
3. Rectal compliance is under the control of the anorectal inhibitory reflex of Debray. This allows the rectum to dilate and act as a reservoir. Inflammatory conditions of the rectal wall interfere with this reflex with decreased compliance and storage capacity
4. Sensory innervation of the anal canal must be intact in order to detect stool within the canal and activate sphincter contraction and continence
5. The anal cushions are responsible for fine continence, the so-called sampling reflex that distinguishes stool from flatus. They fill with blood and provide the final seal of the anal canal.

Impairment or destruction of the above mechanisms will result in incontinence. Incontinence may be classified according to the damaged mechanisms rather than the causes themselves. *Table 1* summarizes the aetiology of incontinence.

## SENSORY

The most common cause of faecal incontinence is faecal impaction. This is usually confined to the elderly, especially those in institutions. Incontinence is secondary to diminished anorectal sensation and persistent internal sphincter relaxation as a result of continued rectal distension from the impacted faeces. Treatment is through disimpaction and with laxatives.

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Spurious diarrhoea will cause incontinence to those with impaired mobility who are unable to overcome the urgency. This is in the presence of normally functioning sphincter and continence mechanisms.

Destruction of the sensory receptors as a result of surgery, fibrosis or malignancy will lead to incontinence.

## SPHINCTER COMPLEX

The majority of patients referred to the colorectal clinic will have incontinence as a result of obstetric injury. Direct injury to the sphincter complex may occur as a result of uncontrolled second stage delivery, epidural anaesthesia, instrumental delivery and episiotomy (*Figure 1*). Prolonged labour is also associated with pudenal nerve neuropathy (Sultan et al, 1993, 1994, 1998; Kamm, 1994; Poen et al, 1998). The risk of a third-degree tear during childbirth varies from 0.5–5%. Occult injuries to the sphincter in the presence of an intact perineum at the time of delivery are more common (Sultan et al, 1998), and may present many years after childbirth.

Sphincter injuries may result from anal surgery, most commonly from the surgical treatment of fistulae-in-ano, notably high complex fistulae. The insertion of a loose seton* has been associated with a success rates approaching 78% in high transsphincteric and suprasphincteric anal fistulae, but with alteration of continence in 36% of patients (Kennedy and Zegarra, 1990). The tight seton has been recommended whenever the fistula encircles more than 30% of the sphincter complex (Goldberg and Garcia-Aquilar, 1996). The seton is inserted, and areas of sepsis laying outside the sphincter complex

*A seton is a thread which is passed through the fistula tract. The seton may be composed of inert prolene or nylon suture material or specially prepared from multiple layers of agents derived from plants with a high pH of 9.5.

**TABLE 1.**
**Aetiology of faecal incontinence**

| | | |
|---|---|---|
| Sphincter complex | Obstetric injury | |
| | Surgery | Fistula-in-ano sphincterotomy |
| | | Haemorrhoidectomy |
| | | Imperforate anus |
| | Trauma | |
| | Muscular degeneration | |
| Neurological disorders | Lower motor neuron | |
| | Pudendal nerve neuropathy secondary to childbirth | |
| | Neuropathy | Diabetes |
| | Upper motor neuron | Strokes, trauma, malignancy, dementia |
| | Ganglia | Absence or degeneration |
| Sensory | Destruction of sensory receptors | Surgery |
| | | Fibrosis |
| | | Malignancy |
| | Continuous stimulation | |
| | Overdistension: faecal impaction | |
| Alteration of compliance | Surgery | Anterior resection, rectocele repair |
| | | Ileo-anal pouch |
| | Fibrosis, inflammation, adenomas | |
| Congenital | Spina bifida, Hirschsprung's disease, atresia | |
| Psychological | | |
| Miscellaneous | Encopresis | |
| | Prolapse | |
| | Rectoceles | |
| | Intussusception | |
| | Diarrhoea and marked urgency | |

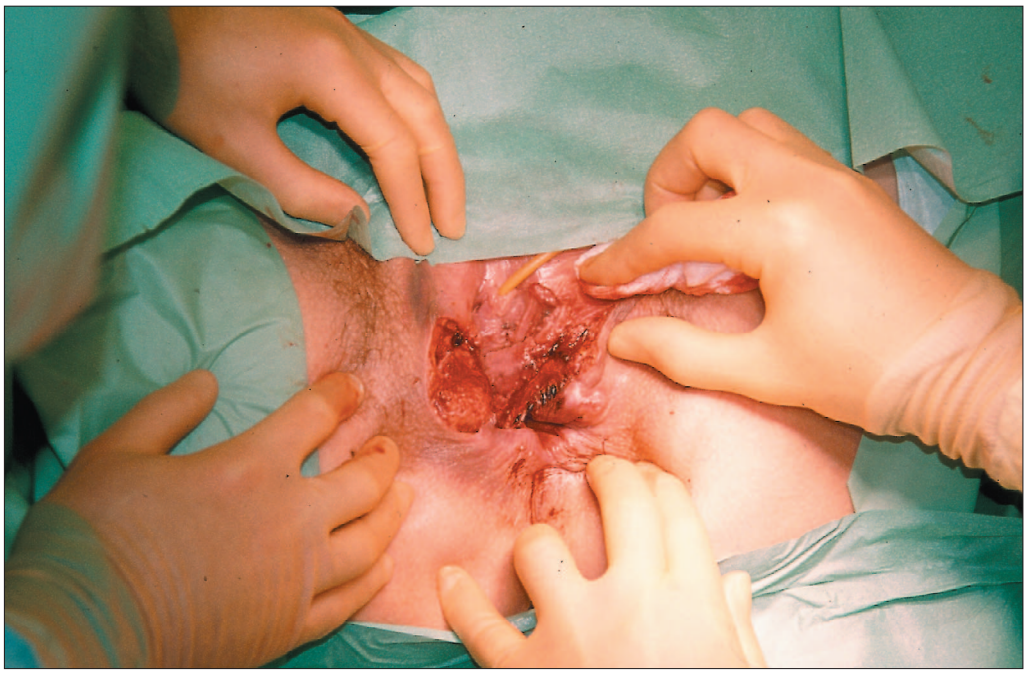

*Figure 1. Third degree obstetric anal sphincter injury.*

are laid open. Once sepsis has resolved, the seton is tightened at weekly intervals until the seton cuts through the sphincter muscles. Incontinence rates have been reported ranging from 54% to 62%, with some 29% requiring daily pads (Bennet et al, 1963; Goldberg and Garcia-Aquilar, 1996). Following simple fistulotomy of low fistulas, 9% of patients will experience incontinence.

Other common anal surgical procedures that can damage the sphincter complex are haemorrhoidectomy, lateral internal sphincterotomy and manual dilatation of the anus for the treatment of anal fissures. Incontinence following haemorrhoidectomy may approach 26% (Bennet et al, 1963). Faecal incontinence after lateral internal sphincterotomy is rare, at <1% (Lund and Scholefield, 1996). In a series of 829 patients who had sphincterotomy for chronic anal fissure, 35% had lack of control for flatus, 22% soiled and 5% had major episodes of incontinence (Khubchandani and Reed, 1989). Injudicious manual dilatation is associated with incontinence (up to 20%) (MacIntyre and Balfour, 1977). Long-term follow-up of patients subjected to an anal dilatation has shown incontinence rates of 52% at 17 years (Konsten and Baeten, 2000).

## INTERNAL SPHINCTER DYSFUNCTION

Isolated degeneration of the smooth muscle of the internal anal sphincter affects both men and women, often in middle age. It is a common cause of soiling (Vaizey et al, 1997). The resting anal pressure is low, and endosonography often shows that the internal sphincter is thin and fibrotic. Pudendal nerve damage, previously believed to be responsible for most cases of idiopathic faecal incontinence, is rare. Weak or structurally damaged smooth muscle tissue of the internal anal sphincter cannot be treated surgically. Often these patients respond to treatment with loperamide.

Patients with progressive systemic sclerosis (Engel et al, 1994) or chronic idiopathic intestinal pseudo obstruction, and those who have had radiotherapy for cervical or other pelvic neoplasms, may develop degeneration and fibrosis of the internal anal sphincter and sensory receptors, leading to passive faecal incontinence. The problem is sometimes compounded by diarrhoea.

## NEUROLOGICAL DISEASE

Faecal incontinence may impair quality of life as much as the primary disorder. In patients with neurological disease, other factors such as damage during childbirth, side-effects of drugs or coexistent behavioural disorders need to be identified.

Faecal incontinence occurs in about 50% of patients with multiple sclerosis. In 25%, it occurs at least weekly (Hinds et al, 1990). In a series of patients with spinal injuries, 61% had faecal incontinence, and in 11% it occurred at least weekly (Glickman and Kamm, 1996). Patients rated their bowel symptoms not far below the loss of mobility in terms of distress.

Faecal incontinence also occurs in patients with long-standing neuropathy secondary to diabetes mellitus. Faecal incontinence is a major social disability in 90% of patients with spina bifida, and about 50% soil regularly (Malone et al, 1994).

## CONGENITAL DISORDERS

Incontinence occurs in 50–80% of adolescents and adults treated surgically for anal atresia (Hassink et al, 1996), regardless of the type of operation (Mulder et al, 1995). In 60 children who had been operated on for Hirchsprung's disease at a mean of 9 years previously, 53% had serious faecal soiling and 27% less severe soiling. The prevalence did not seem to decrease with age (Catto-Smith et al, 1995).

## ALTERATION OF COMPLIANCE

Compliance of the rectum is affected by surgery, most notably following anterior resection and ileal pouch procedures. Loss of compliance results in reduced reservoir capacity and a tendency for incontinence. Urgency is also a feature. A similar pattern is noted in patients with rectal inflammation, e.g. Crohn's disease and radiation proctitis.

## MISCELLANEOUS

Children with a normal anal sphincter can pass stool inappropriately (encopresis), or they may leak faeces as a result of faecal impaction with overflow, which usually requires disimpaction and treatment with laxatives. Prolapse, intussusception and rectocoeles may also present with incontinence. Careful examination will help diagnose these conditions.

## CONCLUSION

Faecal incontinence is more common than one would expect. Patients are often initially embarrassed about discussing their symptoms. An understanding, empathic doctor is crucial to elicit an accurate history. The most common cause is faecal impaction in the elderly. Patients presenting to the surgical clinic usually have a structural defect in the sphincter complex secondary to childbirth. HM

### KEY POINTS

- Faecal incontinence is common and socially disabling.
- Faecal incontinence affects 2% of all adults and up to 33% of those in residential care.
- The true incidence is unknown and is thought to be underreported.
- Faecal incontinence is commonly caused by faecal impaction and sphincter damage following childbirth.
- It is often multifactorial in aetiology.

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