Faecal incontinence in adults

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Abstract | Faecal incontinence, which is defined by the unintentional loss of solid or liquid stool, has a worldwide prevalence of ≤7% in community-dwelling adults and can markedly impair quality of life. Nonetheless, many patients might not volunteer the symptom owing to embarrassment. Bowel disturbances, particularly diarrhoea, anal sphincter trauma (obstetrical injury or previous surgery), rectal urgency and burden of chronic illness are the main risk factors for faecal incontinence; others include neurological disorders, inflammatory bowel disease and pelvic floor anatomical disturbances. Faecal incontinence is classified by its type (urge, passive or combined), aetiology (anorectal disturbance, bowel symptoms or both) and severity, which is derived from the frequency, volume, consistency and nature (urge or passive) of stool leakage. Guided by the clinical features, diagnostic tests and therapies are implemented stepwise. When simple measures (for example, bowel modifiers such as fibre supplements, laxatives and anti-diarrhoeal agents) fail, anorectal manometry and other tests (endoanal imaging, defecography, rectal compliance and sensation, and anal neurophysiological tests) are performed as necessary. Non-surgical options (diet and lifestyle modification, behavioural measures, including biofeedback therapy, pharmacotherapy for constipation or diarrhoea, and anal or vaginal barrier devices) are often effective, especially in patients with mild faecal incontinence. Thereafter, perianal bulking agents, sacral neuromodulation and other surgeries may be considered when necessary.

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Faecal incontinence (FI) is defined by the unintentional loss of solid or liquid stool in people aged ≥4 years[1,2]. However, the aetiology of FI in children aged ≥4 years differs from that in adults; thus, this Primer focuses on FI in adults. Anal incontinence includes leakage of gas and/or faeces but the uncontrolled passage of flatus alone, without faeces, is not defined as FI. The Rome criteria, revised over time, define FI as the recurrent uncontrolled passage of faecal material for ≥3 months, which aims to exclude self-limited conditions[3]. Many patients misrefer to FI as diarrhoea. At least 7% of adults living in a community setting have FI[4]. Bowel disturbances, particularly diarrhoea, anal sphincter trauma (obstetrical injury or previous surgery), rectal urgency and chronic illness, are the main risk factors for FI; other causes include neurological disorders, inflammatory bowel disease (IBD), and pelvic floor anatomical disturbances[1]. These conditions cause FI through bowel disturbances, typically diarrhoea and/or anorectal sensorimotor dysfunctions. The latter comprises weakness of the anal sphincter and/or levator ani muscle, a small and/or stiff rectal reservoir, and decreased or increased rectal sensation[5]. Severe constipation with faecal impaction may predispose to overflow FI[1]. Initial management approaches include investigations if prompted by red-flag symptoms (such as bloody stool or weight loss), simple measures such as fibre supplementation for bowel disturbances, management of haemorrhoids, and prompt surgical referral for selected indications such as full-thickness rectal prolapse or rectovaginal fistula[6]. Especially in patients with moderate or severe symptoms, anorectal structure and function should be characterized by diagnostic tests, the results of which may guide therapy. For these patients, management follows a stepwise approach that begins with non-surgical options (diet and lifestyle modification, behavioural therapy, pharmacotherapy for constipation or diarrhoea, and anal or vaginal barrier devices), followed by pelvic floor biofeedback therapy[6–8]. Standard advice improved symptoms in 50% of patients in a controlled trial[9]. If symptoms persist, sacral neuromodulation (SNM) or perianal biomaterial injection, or, in patients with an external anal sphincter (EAS) defect, anal sphincteroplasty may be considered[7].

In many patients with FI, the disorder affects nearly every aspect of daily life. The inability to manage this bodily process leads to the loss of confidence, self-respect, modesty and composure[10]. Since 1995, several epidemiological studies have shown that FI is common not only in nursing homes but also in the community. Our understanding of the risk factors and pathogenesis of FI, especially the interaction between bowel disturbances and anorectal dysfunctions, has also evolved. Controlled,

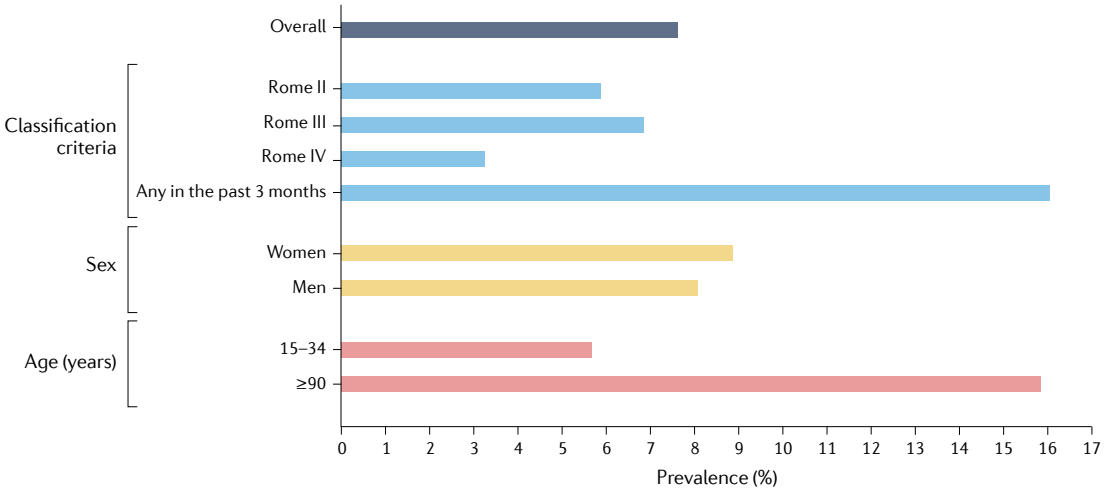

Fig. 1 | **Prevalence of FI.** The definition of faecal incontinence (FI), methods to ascertain the symptom, sex distribution and age distribution of surveyed participants vary considerably between studies. According to two systematic reviews, the prevalence is lower in younger than in older people[4] and when defined by Rome II criteria[20]. Variations in the Rome criteria over time also affect the observed prevalence of FI: the more restrictive Rome IV criteria have substantially reduced the prevalence[21]. Only few studies have assessed differences between men and women[4]. A comprehensive meta-analysis of the epidemiology of FI is necessary.

multicentre trials have demonstrated that pelvic floor biofeedback therapy is better than pelvic exercises alone in patients who did not respond to education and management of bowel disturbances[11]. However, SNM was predominantly evaluated in uncontrolled studies. The design, inclusion criteria and primary end points varied considerably among studies, and head-to-head comparisons are only now being performed[12]. The precise utility of managing bowel disturbances or their stepwise management is unknown. Even simple measures are not diligently applied in clinical practice.

In this Primer, we discuss the epidemiology, including relevant risk factors, and pathophysiological mechanisms of FI, describe the diagnostic workflow and consideration, summarize both non-surgical and surgical management options, and highlight the effects of the disorder on the quality of life (QoL) of patients.

Flatus
Passing of bowel gas.

Rome criteria
A set of diagnostic criteria defining functional bowel disorders.

Faecal impaction
Accumulation of faeces in the colon, especially in the rectum, which may cause blockage.

Prolapse
A bulging or falling out of a body part, such as the rectum or vagina, that may occur because of weakened supportive tissues and/or excessive straining.

Biofeedback therapy
Training by visual or auditory feedback signal of muscle action.

Epidemiology

Prevalence

It is challenging to precisely estimate the population prevalence of FI owing to several reasons. First, the definition of FI varies among studies. For example, not all studies include the involuntary passage of flatus, differentiate between incontinence for solid and liquid stools, and/or discriminate between soiling and gross stool leakage. Second, data sampling methods (for example, mailed or electronic surveys[13,14], phone[15] or face-to-face interviews[16]) vary among surveys. It is conceivable that some people may be reluctant to acknowledge that they have FI, which is an embarrassing symptom[17], during a phone interview. Third, some studies consider any accidental leakage of stool while others ask respondents not to consider leakage during short-term diarrhoeal illnesses[18].

Excluding studies in selected patient populations (for example, those with diabetes mellitus and IBD)[19], the population-based median prevalence of FI is 7.7% and ranges between 2% and 20.7%[4]. It is lower (5.9%) when FI is defined by Rome II criteria[20] (FIG. 1). The prevalence of FI depends on the definition of FI and the method of data collection such as in person or by mail or the internet[20]. For example, a multinational survey estimated the FI prevalence defined as any FI, according to Rome III criteria, and according to Rome IV criteria to be 16.1%, 6.9% and 3.3%, respectively[21]. Both Rome III and Rome IV criteria require a FI duration of 6 months, but Rome III criteria require ≥1 FI episode, whereas Rome IV criteria require ≥2 FI episodes in the past 3 months. By comparison, differences in the prevalence of FI across countries were less pronounced; for example, FI prevalence defined as any FI was 13.3% in the UK, 17.5% in the USA and 17.6% in Canada[22].

Most studies have been conducted in cohorts with a median age of ~50 years, included more women than men, and were conducted in the American continents, followed by Europe, Oceania and Asia[4,20]. Few studies report data from the Middle East and Africa, where obstetric anal sphincter injury is not uncommon[23]. These population-based studies are likely to include people who have other diseases that predispose to FI in the general population.

Risk factors

Sex. In clinical practice, most patients with FI are women, arguably because obstetric injury to the pelvic floor and irritable bowel syndrome (IBS) are more common in women[24].

However, most epidemiological studies either did not observe or did not report sex differences in the prevalence of FI. In studies that did, the median prevalence, reported in systematic reviews, was greater, but not significantly, in women (8.9%) than in men (8.1%)[4,25] (FIG. 1). Higher rates in women are likely due to pelvic floor anatomy and damage associated with obstetric trauma.

Episiotomy
An incision made in the perineum — the tissue between the vaginal opening and the anus — during childbirth.

Age. Age was a strong risk factor for FI prevalence in most studies when investigated[4,25,26]. However, FI was variably defined across studies. Other studies did not evaluate the relationship between age and FI, partly because they were performed in a selected, typically elderly population, aged above 65 or 70 years. The median prevalence of FI was 5.7% for people aged 15–34 years and 15.9% for people >90 years of age[4]. FI prevalence increases substantially above the age of 65 years[20] (FIG. 1). Perhaps the association between age and FI is explained by age-associated weakening of pelvic floor muscles and other age-associated diseases that predispose to FI. Even in asymptomatic women and men, increased age is associated with reduced anal resting pressure and, to a lesser extent, reduced squeeze pressure, which suggest a weaker barrier; neurogenic injury in the EAS, which may partly explain reduced anal squeeze pressure; increased rectal pressure and recto-anal gradient (that is, rectal-to-anal pressure difference) during evacuation; and reduced rectal capacity (that is, a smaller reservoir) and sensation[27–29]. Taken together, these changes may predispose to FI in older persons.

Box 1 | **Evidence-based approach for primary and secondary prevention of FI**

This box summarizes our current understanding, partly evidence based, of measures directed towards primary and secondary prevention of faecal incontinence (FI).

Education
School and adult learning programmes to optimize pelvic floor function for future childbirth. Measures to prevent and manage obesity[a].

Prenatal and postnatal care
Postnatal programmes such as the Rééducation Périnéale in France, which incorporates up to 20 sessions of physical therapy for all women after childbirth. There is stronger evidence that such approaches are effective for urinary incontinence than for anal incontinence[306].

Vaginal delivery
Avoid operative vaginal delivery, which is associated with a considerably increased risk of FI[33].

Pregnant women with previous anal sphincter injury
Postpartum bowel symptoms after the next delivery were not significantly different between the caesarean section and vaginal delivery groups, administered per protocol[307]. However, 3 months after delivery, the anal squeeze pressure was lower after vaginal delivery but not after a caesarean section, and faecal continence at 6 months after a second delivery was comparable in women randomized to vaginal delivery or caesarean section[308].

Women with multiple risk factors for anal sphincter injury (for example, breech presentation, vaginal birth after caesarean delivery or high estimated fetal birth weight)[30,32,36]
Consider elective caesarean section rather than vaginal delivery.

Patients with constipation or diarrhoea
Effective management of bowel disturbances with dietary modifications, medications and/or pelvic floor biofeedback therapy for defecatory disorders[a].

Anal surgery
Anal surgery should, if possible, be sphincter-preserving (for example, during fistula or fissure management) and good surgical technique (for example, haemorrhoidectomy).

Radiotherapy for prostate cancer
Provide targeted, intensity-modulated and image-guided radiotherapy, with which gastrointestinal adverse effects are less frequent and typically resolve by 4 months. Injection of an FDA-approved, biocompatible polymer (hydrogel spacer) between the prostate and rectum before initiating radiation significantly reduces rectal adverse effects during prostate radiotherapy[309].

[a]Evidence that measures prevent FI is lacking.

Obstetric trauma. Obstetric trauma is the most common cause of anal sphincter injury in women. Over time, the incidence of operative vaginal delivery and perianal lacerations of the two highest severities during vaginal delivery have declined; the most severe lacerations occur in ≤5% of deliveries[30–33]. It is useful to separately consider the contribution of obstetric trauma to postpartum FI (occurring within 18 months after delivery) and late FI (occurring several decades after delivery)[18]. Some studies that investigated the obstetric risk factors for anal sphincter injury[32] and FI were agnostic to the latency between childbirth and FI[33–36], whereas other studies differentiated between early FI and late FI, occurring 4–12 years after childbirth[37]. Compared with spontaneous vaginal delivery, operative vaginal delivery was associated with a significantly higher hazard of anal incontinence and pelvic organ prolapse[33]. Women with clinically recognized anal sphincter tears are more than twice as likely to report postpartum FI than women without sphincter tears[38]. The risk factors for anal sphincter injury during vaginal delivery include a second stage of labour of >2 h, Asian race, nulliparity, high fetal birth weight, vaginal birth after caesarean delivery, abnormal presentation (for example, breech or posterior), episiotomy (especially midline), and instrumented delivery with forceps or vacuum assistance[30,32,36]. In addition to the anal sphincter injury, maternal age >35 years and obesity seem to further increase FI risk[36]. Compared with caesarean section, spontaneous vaginal delivery was associated with an increased risk of FI[36]. However, a caesarean section does not completely protect against FI[35]. Although obstetric anal sphincter injury is associated with an increased risk of FI, episiotomy is not protective[36]. Measures for primary and secondary prevention of FI are listed in BOX 1.

The prevalence of FI increases with age[14,18,22]. By univariate analyses, selected obstetric variables are risk factors for late-onset FI that occurs several years after childbirth[37,39]. However, after adjusting for diarrhoea and rectal urgency, which are the strongest risk factors for FI, as well as for other risk factors, obstetric risk factors did not independently predict FI in older women[39,40].

Bowel disturbances. Diarrhoea, most often owing to cholecystectomy and/or IBS, is the strongest risk factor for FI in the community[14,22,39–41]. This perhaps suggests that loose stools are more important risk factors for FI in older women than obstetric injury[40,42]. The role of constipation as a risk factor for FI is unclear, as constipation has been found to not affect[22] or be associated with a reduced[39] or increased[14] risk of FI. Patients who are referred for anorectal tests often have coexistent constipation and FI[43]. These patients may have worse QoL than those with FI who do not have constipation[44]. Faecal impaction may predispose to overflow FI, especially in elderly patients[45].

Lifestyle variables. Obesity[37] and smoking[40,46] are associated with FI. The mechanisms by which obesity may predispose to FI are partly understood. Obesity is a risk factor for diarrhoea and accelerated colonic transit and is associated with increased intra-abdominal pressure,

Rectocele
Bulging of the rectum into the vagina owing to weakening of the fibrous tissue that separates the rectum from the vagina.

Sarcopenia
Progressive loss of skeletal muscle mass.

Slow-twitch muscle fibres
A subtype of muscle fibres that contain more blood-carrying myoglobin and, therefore, have their own source of energy necessary to sustain force for an extended period of time; however, in general, they generate less force than fast-twitch fibres.

which may damage the pelvic floor, as well as with greater rectal pressure[47]. Compared with patients with FI without obesity, patients with FI with obesity were more likely to have had a cholecystectomy, diarrhoea, a larger maximum rectal capacity, and a greater rectal and upper anal resting pressure[48]. In contrast to urinary incontinence, FI does not improve but may worsen after bariatric surgery, which is often associated with diarrhoea secondary to gastric bypass[49,50].

In a population-based, case–control study of FI in women, current smoking (OR 4.7, 95% CI 1.4–15) but not ex-smoking was an independent risk factor for FI even after adjusting for bowel disturbances (diarrhoea and IBS), cholecystectomy, rectocele, stress urinary incontinence and elevated BMI[40]; the odds ratio for current smoking was similar to that for all other risk factors apart from diarrhoea (OR 53, 95% CI 6.1–471) and BMI (OR per unit 1.1, 95% CI 1.004–1.1). A follow-up study in the same cohort found that heavy smoking (≥20 pack-years) was associated ($P=0.052$) with external but not internal sphincter atrophy via MRI. Smoking impairs muscle protein synthesis and increases the expression of genes associated with impaired muscle maintenance, thereby increasing the risk of sarcopenia[51]. Finally, caffeinated, and to a lesser extent decaffeinated coffee, increased colonic motor activity in healthy people[52,53] but there is no evidence linking caffeine consumption to FI.

Physical and mental disability. FI is associated with physical and/or mental disabilities. In some of these cases, FI may have persisted throughout childhood into adult life. A comparison of 54,816 people aged 60–89 years with dementia with an age-sex stratified sample of 205,795 people without dementia in the years 2001–2010 in the UK Primary Care Database found that the rate of a first diagnosis of FI was threefold higher in people with dementia than in those without dementia[54]. In a cross-sectional study of the entire nursing home population in one Norwegian municipality, the prevalence of FI a few times per month was 42.3%[55]. Diarrhoea, urinary incontinence and dementia were risk factors for FI. Individuals who were nursing home residents for 4–5 years had a >2.5 times increased likelihood of FI than those resident for <1 year[55]. Residents with deficiency in feeding, dressing, toilet use and mobility had higher odds of having FI than those without deficiencies in activities of daily living. Needing help for transfer between bed and chair was a risk factor for FI. Of 15,432 patients in a home hospice, 65% had FI[56].

Menopause and menopausal hormone therapy. The EAS, smooth muscle of the internal anal sphincter (IAS) and connective tissue of the anal canal express steroid hormone receptors[57]. In one study, menopause was a significant risk factor for FI by univariate but not multivariate analysis[40]. Data linking menopausal hormone therapy (MHT) with FI are inconclusive[57]. Among women after menopause in the US Nurses Health Study, current or past MHT use was associated with a modestly increased risk of FI (defined as ≥1 liquid or solid FI episode per month)[57]. This risk increased with longer MHT duration and in those who received MHT that contained a combination of oestrogen and progestin versus oestrogen monotherapy, and decreased with time since discontinuation.

Surgery. Most common anorectal surgical procedures have small but recognized risks of causing FI. This risk is highest after the deliberate division of the IAS for treatment of fissure by lateral sphincterotomy (5–10%)[58–60] or division of one or both sphincters for anal fistula[61,62]; however, conventional haemorrhoidectomy also carries some risk (~1% when transient symptoms are excluded[63]) as does low anterior resection surgery and proctectomy. Surgery for rectal prolapse may improve or worsen continence function with high rates of new-onset urgency considered a particular concern after stapled transanal rectal resection[64]. After pelvic radiotherapy with or without para-aortic irradiation for genitourinary or gynaecological malignancies, up to 40% and 5% of patients have grade 2 or grade 3, respectively, gastrointestinal adverse effects, including FI, which typically resolve over time[65].

Mechanisms/pathophysiology

Several factors contribute to the maintenance of faecal continence: colorectal motility, stool volume and consistency, the ability of the rectum to serve as a reservoir and to evacuate stools, rectal sensation, and the pelvic floor and anal canal barrier (FIGS. 2 and 3). FI occurs when patients develop bowel disturbances, typically diarrhoea, that overcome the anorectal continence barrier and/or develop anorectal sensorimotor dysfunctions (that is, anal weakness, a stiff and/or small rectal reservoir, and increased or reduced rectal sensation). These risk factors contribute to and increase the likelihood of developing FI.

Anatomy and physiology

Pelvic floor. The levator ani and puborectalis muscle (PRM) are tonically contracted at rest, and contract further to maintain continence during activities that increase intra-abdominal pressure such as coughing[66,67]. The PRM, which is superior to the EAS, forms a U-shaped sling around the anorectal junction to maintain a relatively acute anorectal angle (FIG. 2). Increased intra-abdominal pressure serves as a 'flap valve' to seal the anterior rectal wall over the anal canal[67]. When the PRM contracts, the anorectal angle becomes more acute; the anorectum is compressed in both sexes and also closes the vagina and urethra in women[68]. The deeper muscles (levator ani) and PRM are principally innervated directly from sacral nerve roots S3 and S4 (REFS.[68,69]) and branches of the pudendal nerve[68]. The pudendal nerve also innervates the levator ani in some cadaver specimens[70].

Anal canal. The EAS is predominantly composed of slow-twitch muscle fibres that maintain a sustained tonic contraction, which also contributes to anal resting tone. A hermetic seal is provided by interdigitation of the three vascular anal cushions[71]. The EAS and PRM also have fast-twitch fibres that enable the anus to contract rapidly to supplement continence when threatened (for example, when stool enters the upper part of the

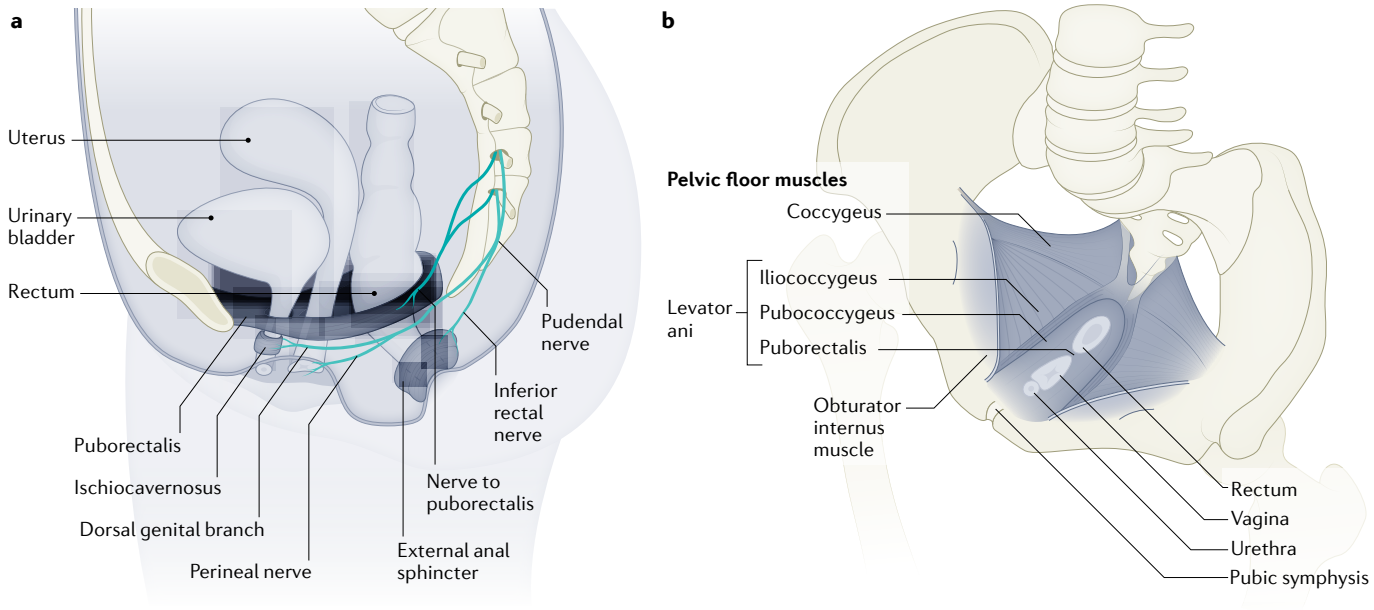

Fig. 2 | **Neuromuscular innervation of the pelvic floor muscles and anal sphincter. a** | This sagittal slice shows that the pelvic floor supports the bladder, uterus and anorectum. The external anal sphincter and ischiocavernosus muscles are supplied by the pudendal nerve. The puborectalis is innervated by a separate nerve that arises from the motor roots of S2 and S3 above the pelvic floor. It is important to recognize that neurogenic changes may be caused not only by a pudendal neuropathy or more proximal neurogenic injury, but also by trauma to the local nerves during vaginal delivery. The internal anal sphincter (not shown) is innervated by parasympathetic and sympathetic nerves as well as the myenteric plexus. **b** | The pelvic diaphragm is subdivided into the coccygeus and the three components of levator ani: pubococcygeus, iliococcygeus and puborectalis. These muscles are attached peripherally to the pubic body, the ischial spine, and to the arcus tendineus, a condensation of the obturator fascia in between these areas. Deficiency of the pelvic floor can lead to prolapse or 'sagging' of organs through the urogenital hiatus. Part **b** reprinted from REF.[305], Springer Nature Limited.

anal canal)[72] or when intra-abdominal pressure increases rapidly[73]. Activation of muscle spindles in the EAS may contribute to this reflex response[74]. The conjoint longitudinal muscle, which binds and braces the sphincters, also anchors the anal canal to the perianal skin[75]. The EAS is innervated by the pudendal nerves[76,77] and perhaps also by the fourth sacral nerve[78]. The pudendal nerve is particularly susceptible to traction injury, for example, through prolonged or instrumental vaginal deliveries[79] or chronic straining[80].

The IAS[81,82] is primarily responsible for generating anal resting tone[83]. The interstitial cells of Cajal generate slow myoelectric waves at a frequency of 15–30 cycles per minute (cpm)[83], with corresponding pressure fluctuations at a dominant frequency of 16 cpm in humans[84]. These slow waves rather than myogenic mechanisms generate the anal resting tone[83].

The IAS receives tonic excitatory input from postganglionic sympathetic nerves (that is, hypogastric and inferior mesenteric plexuses)[85] and inhibitory input from preganglionic parasympathetic nerves that travel with the sacral nerves[86]. Additionally, nerves from the myenteric plexus of the distal rectum travel in the intersphincteric space to innervate the IAS[87,88].

The anal canal mucosa contains numerous nerve endings and is exquisitely sensitive[74]. When the IAS relaxes, ~7 times every hour, anal sensory receptors sample rectal contents and discriminate between flatus and solid or liquid stool[89–91]. The recto-anal inhibitory reflex evoked by rectal distention during anorectal manometry is the equivalent of the sampling reflex, mediated by the myenteric plexus and modulated by the spinal cord[92]. However, the precise contribution of anal sensory function to preserving continence remains unclear as topical local anaesthetic application abolished anal sensation but did not impair continence to rectally infused saline[93].

Rectum and sigmoid colon. The rectum has two or three transverse folds that may retard transit of stool[94]. In the sigmoid colon, retrograde propulsive contractions may retard passage of stool into the rectum[95,96]. The presence of a distinct rectosigmoid sphincter is controversial[97,98]. Usually, empty[99] rectal distention evokes a rectal contractile response[100–102]. If defecation is inopportune, this contractile response subsides; the rectum serves as a reservoir that accommodates to store stool until defecation is socially convenient[103].

Normal rectal sensation, which is mediated by enteric visceral afferents[104] and somatic afferents from the pudendal nerve to the lower rectum[105], enables graded perception of filling, leading to the conscious urge to defecate. If defecation is inconvenient, the EAS and PRM are voluntarily contracted, the rectum relaxes or accommodates to hold stool, and the urgency subsides[92].

Aetiology and pathophysiology

Several conditions predispose to FI by affecting stool consistency and/or causing one or more anorectal disturbances that frequently coexist[5,106] (FIG. 4 and BOX 2). Among women, the age of onset of FI is approximately evenly distributed in three age groups: ≤40 years, 41–60 years and >61 years[18]; the most common putative

Cells of Cajal
Pacemaker cells in the intestinal smooth muscle.

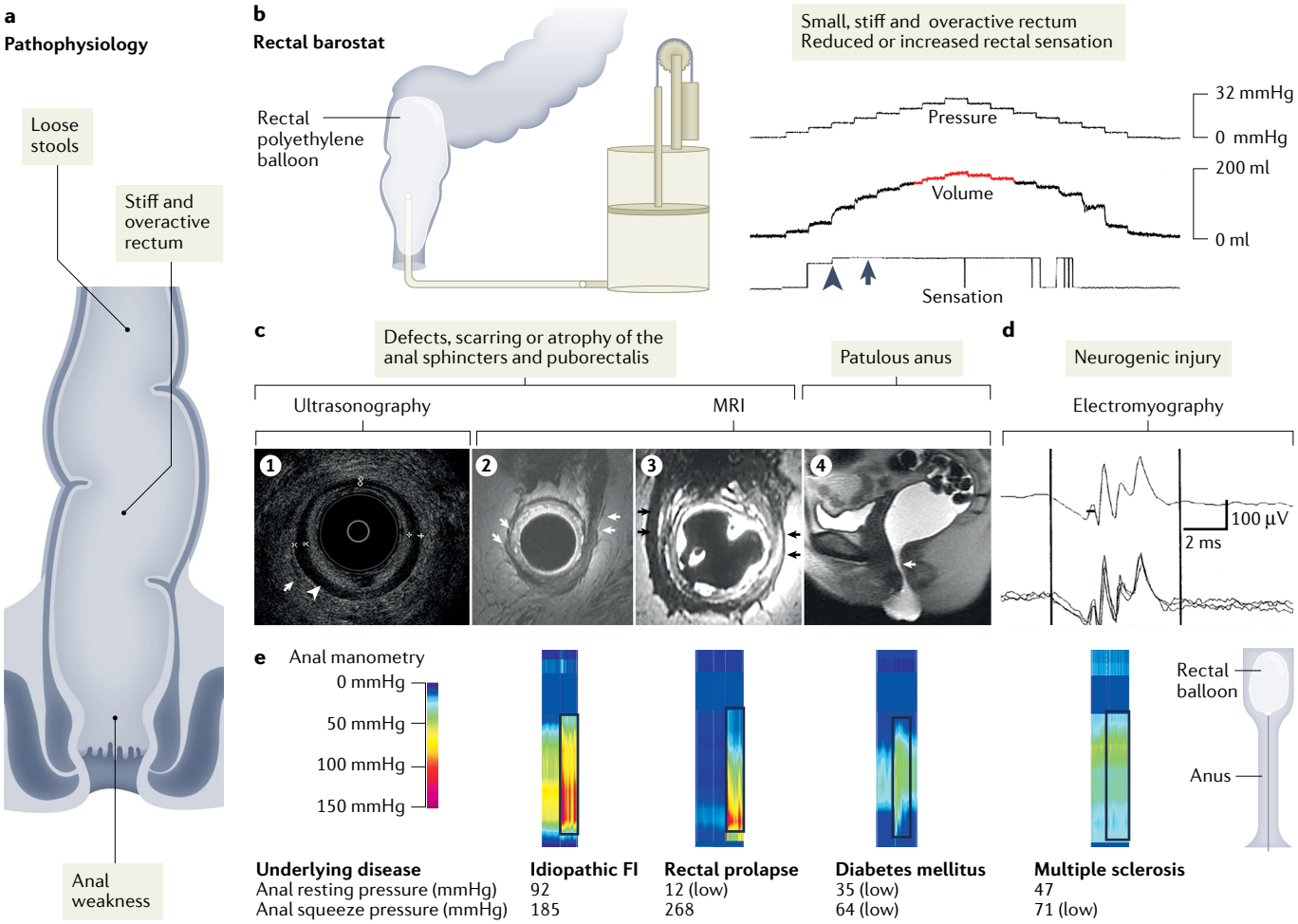

Fig. 3 | **Pathophysiology of FI. a** | Many patients have loose stools, which overwhelm the rectal reservoir and predispose to incontinence, especially in patients with anal weakness. **b** | Rectal distensibility (that is, compliance and capacity) and sensation can be evaluated with a barostat that measures pressure and volume during distention of a highly compliant polyethylene balloon in the rectum. In this example, the maximum rectal balloon volume at a distending pressure of 32 mmHg was <200 ml, which suggests reduced rectal capacity. The arrowhead and arrow denote the desire to defecate and rectal urgency, respectively. **c** | Shows normal-appearing internal (arrowhead) and external (arrow) anal sphincters (1), atrophy of the right but not left side of the puborectalis (arrows, 2), and atrophy of the left but not the right side of external sphincter (arrows, 3). (4) Shows a patulous anal canal in which ultrasonography gel leaks through the anus before evacuation (arrow). **d** | Shows a recording obtained with needle electromyography of the external anal sphincter with prolonged (19.7 ms) polyphasic motor unit potentials (10 phases) suggestive of neurogenic injury. **e** | Shows anal pressure topography, measured with high-resolution manometry, at rest and during squeeze in four patients with faecal incontinence. The pressures, ranging from 0 to 150 mmHg, are shown in colours that are depicted in the colour scale. Pressures were measured at rest and during squeeze (squeeze pressure shown in black rectangle insert). In the patient with idiopathic faecal incontinence (FI), the anal resting and squeeze pressures were normal. Compared with the patient with normal values, resting and/or squeeze pressures were reduced in disease states. Rectal prolapse and diabetes mellitus are associated with reduced anal resting pressure and diabetes mellitus and multiple sclerosis were associated with reduced squeeze pressure. Part **b** reprinted from REF.[167], CC BY 4.0 (https://creativecommons.org/licenses/by/4.0/).

risk factors for FI are complicated vaginal delivery and loose stools. When FI begins several decades after vaginal delivery, the precise contribution of obstetric anal sphincter injury to FI is unclear, prompting the designation 'idiopathic' FI. However, other disturbances (for example, bowel dysfunctions) may contribute to idiopathic FI. In men, the most common causes of FI are an underlying rectal evacuation disorder, anal weakness owing to iatrogenic sphincter injury, and loose stools, which may coexist among individual patients. There are three salient differences between men and women with FI. Anal weakness is more common in women, whereas reduced rectal sensation and an underlying evacuation disorder are more common in men[107].

Anal sphincter weakness or injury. Anal weakness is the most widely recognized anorectal disturbance in FI. Among a cohort of women with an average age of 61 years, ~80% and ~40% have reduced anal squeeze and resting pressure, respectively, which correspondingly reflect EAS and IAS weakness[5]. Sphincter injury may weaken both sphincters. Neurogenic injury along the brain–EAS neuroaxis may also weaken the EAS[1,108]. In studies published more than 20 years ago, some patients had an exaggerated recto-anal inhibitory reflex[106,109]; the aetiology of this is unknown.

In women with obstetric injury, IAS injury, in addition to EAS injury, increases the risk of FI[34]. Obstetric trauma can also lead to damage or avulsion of the levator ani

(including the PRM)[110,111] (FIG. 2). As the risk of FI is higher in patients who undergo caesarian section than in nulliparous women[112], factors associated with pregnancy itself may have a role in damage to the pelvic floor components, for example, pudendal nerve injury and mechanical or hormonal changes that occur during pregnancy or during pushing and labour without vaginal delivery[113]. In women with postpartum anal incontinence, continence improved to a greater extent in those who received intensive training in pelvic floor muscle exercises with or without biofeedback therapy than in those with written education alone[114]. Obstetric injury is the most common cause of anal sphincter injury. Other causes include surgical trauma (such as rectovaginal or anovaginal fistula surgery, anal fissure surgery, anal dilatation) or accidents (such as impalement).

Up to 20% of patients with systemic sclerosis have FI, which may result from one or more disturbances associated with this disease: atrophy and fibrosis of the IAS, rectal prolapse (arguably secondary to collagen accumulation in the rectum and excessive straining), and diarrhoea, which may be due to small intestinal bacterial overgrowth[115].

Disturbances of rectal compliance and sensation. Reduced rectal compliance, increased rectal sensation and loose stools may predispose to FI after pelvic radiation[116], surgery for colorectal cancer (for example, low anterior resection syndrome)[117] and IBD[14]. Some patients with 'idiopathic' FI (for which a direct causation cannot be established) have reduced rectal compliance and/or capacity, which may be reversible and is associated with increased rectal sensation as well as with rectal urgency[5,118–120] because the same amount of stool is filled into a smaller reservoir. Rectal compliance is reduced in patients with colitis and in those with high spinal cord lesions[121,122].

Rectal sensation may be normal, reduced or increased in FI[123]. When rectal sensation is reduced, the EAS may not contract promptly when the rectum is distended by stool, predisposing to FI[124]. Conversely, increased rectal sensation, perhaps secondary to an exaggerated contractile response to distention and/or reduced rectal capacity, may explain the symptom of rectal urgency[5,118]. Chronic constipation is associated with FI in some but not all studies[14,39]. Perhaps laxative-induced loose stools and/or high anal pressures with impaired rectal evacuation and faecal seepage explain FI in patients with constipation[44,125]. Conversely, in patients with faecal impaction, who are typically older, an obtuse anorectal angle, low anal pressures and impaired anorectal sensation, which prevents conscious contraction of the EAS when the IAS is relaxed, lead to faecal soiling[126].

Pelvic floor disorders. Among patients with congenital anorectal malformations, the long-term risk of FI is 17–77%[127]. Structural disorders of the pelvic floor, such as rectal prolapse and descending perineum syndrome,

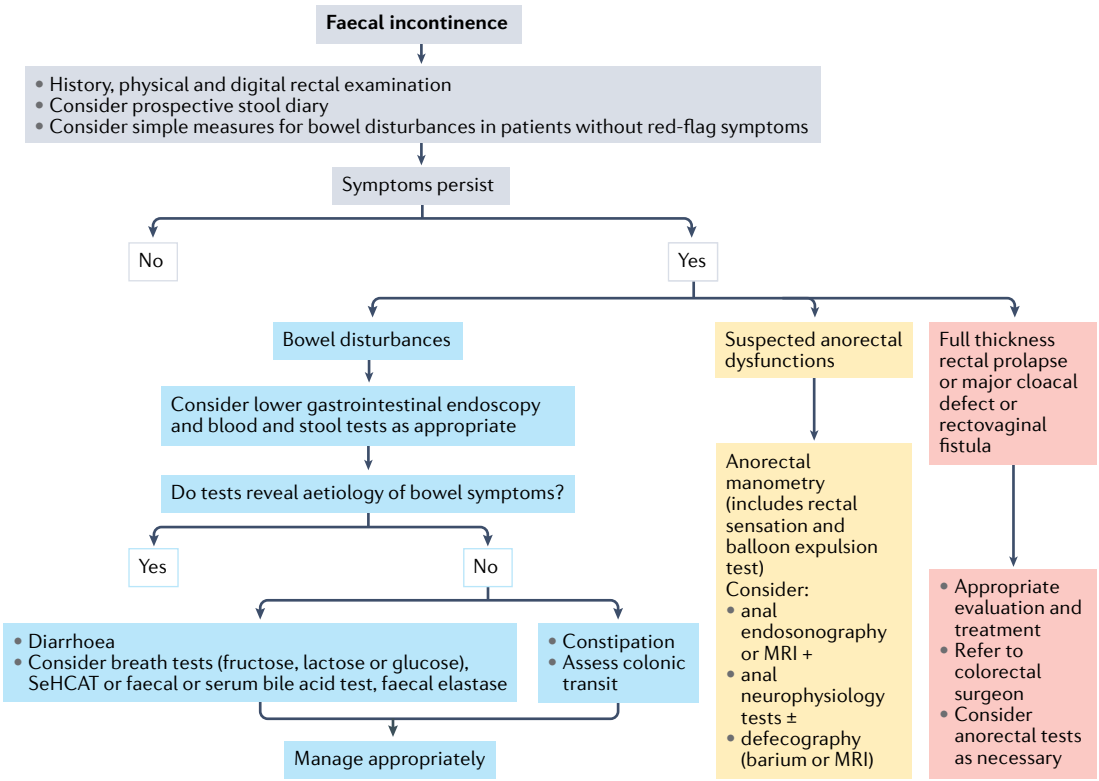

Fig. 4 | **Diagnostic approach for FI.** After a meticulous clinical assessment, simple measures (for example, reduction of excess consumption of caffeine, fructose or loperamide for diarrhoea) should be considered in patients without red flags (for example, bloody stool or weight loss). In patients with persistent symptoms, testing for bowel disturbances and anorectal dysfunctions should be performed as guided by the clinical features. Where appropriate, patients should be referred to a colorectal surgeon. FI, faecal incontinence; SeHCAT, selenium homocholic acid taurine test.

Box 2 | **Pathophysiology of FI**

Several medical and surgical conditions are associated with the symptom of faecal incontinence (FI). The cause of FI is often multifactorial; however, the major causative disorders can be categorized as follows.

Anal sphincter injury or weakness

- Obstetric injury[a,b]
- Surgery: haemorrhoidectomy[a], lateral sphincterotomy[a], fistulectomy[a,b], congenital anorectal malformations[a,b] and surgery for these conditions
- Anorectal or perineal trauma or congenital abnormalities[a,b]
- Myopathy, for example, scleroderma[a]

Structural disorders of the pelvic floor and surgery involving rectal excision

- Rectal prolapse
- Descending perineum syndrome
- Rectocele surgery involving rectal excision

Neurological disorders

- Central nervous system: stroke, dementia, multiple sclerosis, cerebral palsy, spina bifida, spinal cord injury, spinal cord tumour, Parkinson disease
- Peripheral nervous system, for example, diabetic neuropathy or pudendal nerve damage

Inflammatory conditions

- Inflammatory bowel disease: Crohn's disease, ulcerative colitis
- Radiotherapy

Diarrhoea or constipation

- Diarrhoea: cholecystectomy, irritable bowel syndrome, infective gastroenteritis, medications
- Constipation: defecatory disorder, impaction with overflow

[a]Affects internal anal sphincter. [b]Affects external anal sphincter.

may cause FI[128]. As rectal prolapse progresses, supporting structures of the anorectum may be stretched along with the pudendal nerves and pudendal neuropathy can develop[129]. With increased grades of rectal prolapse, the rectal walls protrude through the anal canal, leading to increasing weakness of the anal sphincteric mechanism. In descending perineum syndrome, prolonged excessive straining stretches the supporting structures of the pelvic floor and pudendal nerves and the risk of FI is up to 39%[130]. Solitary rectal ulcer syndrome involves a similar mechanism with associated rectal ulceration and a 17% risk of FI[131].

Surgical procedures that involve partial excision of the rectum reduce rectal capacity and increase the risk of FI. After low anterior resection for rectal cancer, patients may have evacuation urgency, frequency and difficulty; up to 20% have FI[132]. Pathogenic mechanisms include reduced rectal capacity, direct muscle injury or damage to the nerve supply of the pelvic floor or IAS[133], and impairment of the recto-anal inhibitory reflex[134]. The stapled transanal rectal resection procedure also involves rectal excision, is associated with rectal urgency and may predispose to FI[64,135].

Neurological disorders. Patients with FI due to a neurological disease invariably have other deficits (such as limb weakness or reduced sensation) before they develop FI[14] (BOX 2). In these patients, FI may be secondary to lower anal resting and/or squeeze pressures (for example, owing to diabetic neuropathy, multiple sclerosis or spinal cord injury)[136,137], reduced rectal sensation (for example, owing to visceral neuropathy[123] or Parkinson disease[138]), impaired cognition (for example, owing to dementia or intellectual disability), and/or mobility. Constipation (including dyssynergic defecation[139]) and faecal impaction may also be precipitating factors for FI in these patients[136,138].

Inflammatory conditions. The pooled prevalence of FI in patients with IBD is 24%[140]. Risk factors for FI include altered rectal compliance, anal sphincter and pelvic floor dysfunction, and diarrhoea owing to colonic inflammation or resection[141]. In particular, perianal Crohn's disease is associated with anal sphincter weakness[142]. There is an association with disease activity whereby FI is more common when there is active disease compared with remission in patients with ulcerative colitis or Crohn's disease[143]. Pelvic radiotherapy may lead to FI owing to reduced rectal compliance, rectal hypersensitivity, reduced resting anal pressure or loose stools[116].

Diarrhoea or constipation. The high prevalence of FI in non-anorectal gastrointestinal conditions (such as IBS, coeliac disease or the post cholecystectomy state) highlights the importance of loose stool or diarrhoea in the pathophysiology of FI[39,40]. Less frequently, constipation may be associated with FI, for example, when there is faecal impaction and overflow[126], a defecatory disorder with rectal prolapse[144], or descending perineum syndrome[128].

Diagnosis, screening and prevention

History

Embarrassed by FI, many patients are reluctant to volunteer the symptom to family members, friends and providers[1,3]. Hence, establishing trust and rapport with patients is critical to uncovering details of FI. Important details include the duration, frequency, type (that is, flatus, liquid stool or solid stool), amount and awareness of FI, severity of urgency, and use of perineal protective devices for FI. Patients should be asked about their bowel pattern, including frequency, stool consistency (Bristol Stool Form Scale)[145], completeness of bowel evacuation, digital assistance to defecate, and ability to discriminate between formed stool, unformed stool and gas[146]. Finally, risk factors for FI and its effects on QoL should be ascertained (BOX 2). FI may be characterized by three categories, which may overlap. First, passive incontinence involves involuntary leakage without awareness, suggesting impaired sensation and/or weakness of the IAS[147]. Second, urge incontinence is characterized by marked rectal urgency with an inability to hold stool despite active attempts, which is associated with weakness of the EAS, reduced rectal capacity and/or rectal hypersensitivity[5,147–149]. Third, faecal seepage involves staining or a small amount of leakage owing to incomplete evacuation or impaired rectal sensation, IAS dysfunction or neuropathy[150,151]; the leakage often occurs after defecation.

Given the unpredictable nature of symptoms, a prospective 7–14-day stool diary is useful for assessing the

characteristics of FI and its relationship with bowel habits[146,152]. A study from 2020 suggests that a digital stool diary is as accurate as a paper diary and preferred by some patients[153].

Severity classification and grading systems

Most instruments for rating the severity of FI evaluate the type and frequency of bowel leakage[1]. The Cleveland Clinic Faecal Incontinence Score (also termed Wexner score)[154], the St Mark's Incontinence Score (also termed Vaizey score)[155] and the Fecal Incontinence Severity Index[156] are the most widely used scales in clinical trials. All these instruments enquire about the type and frequency of FI. The Vaizey, International Consultation on Incontinence Questionnaire (ICIQ) Anal Incontinence Symptoms and Quality of Life Module (ICIQ-B), ICIQ Inflammatory Bowel Disease (ICIQ-IBD), and the Accidental Bowel Leakage Evaluation instruments enquire about defecation urgency[1,157,158]. The Faecal Incontinence Severity Score (FISS) and the Accidental Bowel Leakage Evaluation instrument[152,157,158] also evaluate the volume of FI. In most clinical trials, the primary outcome is defined by a ≥50% reduction in the frequency of FI or the number of days with FI[152,159]. However, some women consider the threshold for improvement to be higher (specifically, 77% reduction in the frequency of FI)[160].

Physical examination

All patients with FI should have a detailed physical, neurological and digital rectal examination (DRE)[161]. Perineal inspection may reveal faecal matter, prolapsed haemorrhoids, dermatitis, scars, skin excoriations or gaping anus. Attempted defecation may disclose excessive perineal descent or rectal prolapse. The normal anocutaneous reflex is characterized by brisk contraction of the EAS upon stroking the perianal skin with a cotton bud in each perianal quadrant. An impaired or absent reflex suggests neuromuscular injury. In addition to anal sphincter and puborectalis tone at rest and during squeeze, the DRE should evaluate for dyssynergia by asking the patient to bear down at which point normally the anal sphincter and puborectalis should relax and the perineum should descend by 2–4 cm. However, some asymptomatic healthy persons may not relax the anal sphincter and/or puborectalis, perhaps partly because it is challenging to simulate defecation in an office. The correlation between anal resting and squeeze function evaluated with DRE and manometry was stronger in some studies[162] than in others[163]. The DRE is also useful to identify dyssynergia (with a sensitivity and specificity of 75% and 87%, respectively[164]), faecal impaction and patulous anus[165]. The assessment of anal and pelvic floor function by DRE is probably influenced by several factors, including examiner finger size, technique and experience as well as the ability of a patient to perform actions during the DRE.

Diagnostic tests

FIGURE 4 provides a suggested algorithm that is guided by clinical features and society guidelines[166,167]. When necessary, lower gastrointestinal endoscopy to exclude colonic inflammation or malignancy and/or gastrointestinal transit studies to identify slow or fast transit should be performed. Dietary elimination and/or a carbohydrate breath test should be considered when appropriate[168]. The use of breath hydrogen tests following glucose ingestion to identify small intestinal bacterial overgrowth is controversial[169]. Additional tests for diarrhoea include the selenium homocholic acid taurine test, faecal and serum bile acid testing, which detects bile acid malabsorption, and faecal elastase testing, which detects pancreatic insufficiency. Thereafter, anorectal manometry plus additional tests, as indicated, should be performed (FIG. 3). The findings should be compared with age-matched and sex-matched normal values and interpreted in the context of symptoms.

Anorectal manometry. During manometry, anal pressures at rest, during squeeze and in simulated evacuation as well as rectal sensation are assessed and the rectal balloon expulsion test is performed[170,171]. The equipment and methods for anorectal manometry vary considerably[171–174]. High-resolution or high-definition catheters display pressures with increased spatial resolution. Air-filled miniaturized balloons are another option[175,176]. The International Anorectal Physiology Working Group recommended a protocol for anorectal function testing[170], which may be simplified[177,178]. The incremental clinical utility of high-resolution solid-state manometry over non-high-resolution manometry is unclear[179]. High-resolution and high-definition manometry measure pressures in 2D and 3D, respectively. The 3D measurements disclose circumferential symmetry, which may discriminate between contraction of the EAS and the PRM and hint at the presence of EAS defects[174,180]; however, more evidence is necessary. Moreover, the 3D probe is larger (12.75-mm diameter) than the 2D probe (4.2-mm diameter), may be uncomfortable for some patients and is more prone to artefact during the examination[181].

Anal squeeze and, to a lesser extent, anal resting pressures are reduced in women with FI[5,107,159]. Anal resting and squeeze pressures and the anal squeeze duration are greater in men than in women with FI[107]. A low anal resting pressure suggests weakness of the IAS and a low squeeze pressure suggests weakness of the EAS and/or PRM. A reduced anal squeeze duration suggests increased fatigability of the EAS[148,182]. In patients with cauda equina lesions, reflex anal contraction during coughing and voluntary squeeze responses are absent; in lesions above the conus medullaris, only the voluntary response is absent[183,184]. However, voluntary anal contraction is also reduced in parous women and in women with FI[185]. Hence, weakness of the EAS is not specific for spinal cord injury.

Rectal compliance and sensation. During rectal air balloon distention[186], higher volume thresholds for two or more end points (first perception, desire and urgent desire to defecate, and maximum tolerable volume) indicate rectal hyposensitivity[170,187,188]. Conversely, lower thresholds indicate rectal hypersensitivity[5,118,188].

Enterocele
Enterocele or small bowel prolapse occurs when the small intestine descends into the lower pelvic cavity and pushes at the top part of the vagina, creating a bulge.

Psyllium
A form of fibre made from the husks of the *Plantago ovata* plant seeds; also known as ispaghula.

Rectal compliance, which is the change in volume for a given change in rectal pressure, is optimally measured with a barostat[5,102,170]. When measurements are obtained by manual distention, a highly compliant balloon[189] is preferred to a latex balloon[170,186,190].

Endoanal ultrasonography and MRI. Endoanal ultrasonography or MRI may identify anal sphincter injury (scar or defect) or thinning (which suggests atrophy) in patients with anal weakness. Endoanal ultrasonography and MRI findings generally concur[5] but ultrasonography and MRI are better for visualizing the IAS and EAS, respectively[5]. MRI is superior for discriminating between an EAS tear or a scar and for identifying EAS atrophy[5], which is very uncommon in asymptomatic women[191].

IAS defects reflect more severe anorectal injury than isolated EAS injuries[34,192]. It can be challenging to interpret the clinical significance of anal sphincter injury, partly because even asymptomatic women have postpartum sphincter defects, which are less common with 3D ultrasonography or MRI (10% of women) than with 2D ultrasonography (up to one-third of women)[42,191,193,194].

Defecography. Performed in the seated (barium fluoroscopy) or the supine (MRI) position, defecography assesses the anorectal angle, location of the pelvic floor, anal canal length, and structural abnormalities (such as rectocele) at rest, during squeeze and during evacuation[5,190,195,196]. Because anorectal structural abnormalities are observed even in asymptomatic people, they may not be clinically relevant[190,195]. Image analysis programmes facilitate more standardized and reproducible measurements[197]. MR defecography is less sensitive but as specific as barium defecography in identifying rectal intussusception. For rectocele and enterocele, these tests are equivalent[198].

Rectal balloon expulsion test. Normally, humans can expel a 50-ml water-filled balloon from the rectum in <1 min (REFS.[186,199]). Some patients with faecal seepage[151] and/or overflow FI secondary to faecal impaction[150] have impaired rectal evacuation, which suggests a coexisting evacuation disorder, arguably more common in nursing home residents[200].

Neurophysiological testing. When spinal cord lesions are suspected, MRI of the lumbosacral spine, including the conus medullaris and cauda equina, is performed. The pudendal nerve terminal motor latency test suffers from several pitfalls and should not be used[190]. Performed infrequently, and only in specialist centres, anal sphincter needle electromyography may disclose neurogenic or muscle injury in the EAS and other lumbosacral muscles[28]; comparison with age-matched normal values is important because even nulliparous older women have features of neurogenic injury. Measurement of the anorectal motor evoked potentials in response to transcutaneous magnetic stimulation of the lumbar and sacral spinal cord reveals delayed conduction in patients with spinal cord injury and in 65% of patients with FI with a twofold higher prevalence of anal neuropathy than rectal neuropathy[201,202]. This test is safe and easy to perform but not widely available.

Management

Non-surgical management

Conservative therapy should be attempted as first-line management of FI before surgical interventions. However, some patients (for example, those with recent obstetric anal sphincter injury or large full-thickness rectal prolapse) are likely to require surgery sooner rather than later. Conservative therapies, which are inexpensive and generally safe, include dietary modifications, lifestyle changes, behavioural therapies and pharmacological agents to manage diarrhoea and/or constipation. These options can be implemented even without a detailed diagnostic evaluation. Before pursuing surgical options, it is important to provide an adequate trial of conservative therapy for a sufficient duration[7,203].

Diet and lifestyle modification. Initially, modifiable risk factors, such as low dietary fibre intake[204,205], bowel dysfunction[22,40,206], smoking and increased BMI, should be targeted[40,207]. Fibre has long been recommended as a first step strategy for FI[6,203]. In one randomized trial, psyllium 15 g per day decreased episodes of loose/liquid FI compared with placebo or other dietary fibres[208]. In a crossover study, both low-dose psyllium and loperamide, administered individually, reduced the frequency of FI; the differences were not significant[209]. These data suggest that dietary fibre intake may benefit some patients with FI. However, it is unknown whether these effects are different from those with placebo. Studies examining fibre intake in patients who have coexisting FI and constipation are lacking. Recommending increased fibre intake for patients with diarrhoea is seemingly counterintuitive as increased fibre intake is commonly advised for the treatment of constipation. Pending further study, clinicians should consider suggesting increased dietary fibre intake or supplementation for patients with FI, especially for those with constipation[208].

Additional dietary changes for the management of FI include limiting the intake of caffeine, alcohol, fatty foods, fructose and lactose. While compliance was not evaluated, dietary guidance to limit the intake of these foods improves FI both with[210] and without[211] concomitant fibre supplementation. For individuals experiencing loose or watery stools, a diet low in fermentable oligosaccharides, di-saccharides, mono-saccharides and polyols can be offered[212]. Other practical approaches include a trial-and-error strategy in which patients self-identify individual dietary triggers, modify meal times, ingest smaller meal portions and/or alter food preparation methods (such as avoidance of excessive spice, grease and eating outside the home)[213].

Weight loss may be attempted for individuals with overweight or obesity. In observational studies[214,215], bariatric surgery improved or worsened FI. In a randomized trial[207], weight loss of ≥5 kg was associated with decreased FI symptom severity. Cessation of smoking and regular, low-impact physical activity may be beneficial but have not been studied. Scheduled toileting may also be helpful, particularly in individuals with limited mobility[216].

Behavioural treatments. Patients with FI may exhibit recto-anal sensorimotor dysfunction characterized by reduced anal pressures, rectal hypersensitivity, and reduced rectal capacity and compliance[102]. Pelvic floor rehabilitation aims to improve anal sphincter contractions, sensory dysfunction and coordination. Pelvic floor exercises designed to improve voluntary contraction of the anal sphincter and pelvic floor are taught verbally, sometimes supplemented with written materials and DRE. During pelvic floor biofeedback therapy, patients receive visual or auditory feedback of bodily activity (for example, of anorectal pressures during squeeze)[6]. Several trials of pelvic floor biofeedback therapy have been conducted. In a seminal randomized trial in 171 patients, neither pelvic floor exercises nor biofeedback therapy were superior to standard care supplemented with advice and education; the response rate was ~50% in all groups[9]. Prompted by these findings, another trial evaluated biofeedback therapy in patients who did not respond to standard care and education. In patients who did not respond to education and medical management, 76% of patients randomized to biofeedback-assisted pelvic floor rehabilitation reported adequate relief compared with 41% who were randomized to pelvic floor exercises[11]. By contrast, in a randomized factorial trial in 300 women[217], biofeedback therapy was not different to education for FI symptoms; a planned secondary analysis demonstrated a 50% reduction in FI episodes to be associated with loperamide and biofeedback therapy compared with placebo and education groups after accounting for baseline characteristics and treatment adherence[218]. Whether these benefits of receiving loperamide and biofeedback therapy are explained by loperamide and/or biofeedback therapy is unknown. In another randomized controlled trial[219], biofeedback therapy improved FI symptoms more than attention control. In 400 patients who underwent biofeedback therapy in a tertiary care setting, symptoms improved by ≥50% in two-thirds of patients at the end of treatment (6 weeks) and in 71% of patients 6 months later[220]. No factors predicted the response to biofeedback therapy apart from urge FI, which predicted an early but not sustained (at 6 months) response[220].

These trials provided office-based biofeedback therapy, which is inconvenient for patients. In a randomized trial of 30 patients (26 women), 65% and 60% of patients treated with biofeedback therapy at home and the office, respectively, reported a ≥50% reduction in the frequency of FI; home-based biofeedback therapy (20 patients) was not inferior to office-based biofeedback therapy (10 patients) provided with manometry[221]. The home-based device, which is approved by the FDA, provides anal electrical stimulation and anal resistance exercises. While the data from these studies are mixed, taken together, they justify biofeedback therapy when the response to dietary modifications and bowel management is insufficient[6]. Biofeedback therapy can improve voluntary anal contraction[11,221], reverse rectal sensory disturbances[222], and even improve recto-anal coordination in patients with coexisting rectal evacuation disorders[146,223].

Pharmacological agents. Several pharmacological agents are available for the treatment of FI. Loperamide in doses up to 4 mg three times daily has demonstrated efficacy in placebo-controlled studies of patients with chronic diarrhoea through improved stool consistency, anal sphincter pressures and gastrointestinal transit[224,225]. In a planned secondary analysis of a randomized trial[223], loperamide did not worsen constipation in patients with normal stool consistency. Other anti-diarrhoeal medications, such as diphenoxylate plus atropine, or cholestyramine[226,227], may be offered, although data are limited. In an uncontrolled study, amitriptyline improved faecal continence, presumably by decreasing rectal motor activity and/or intestinal transit[228]. For patients with FI and constipation, osmotic and stimulant laxatives may promote rectal emptying and prevent overflow FI[45]. Other laxatives, including stimulants (for example, bisacodyl, senna or prucalopride) and prosecretory (for example, lubiprostone, linaclotide or plecanatide) agents, may be attempted[229]; however, evidence in patients with FI and constipation are lacking. The effects of the α2 adrenergic agonist clonidine were not different to placebo in all-comers with FI; however, in patients with diarrhoea and FI, a non-significant trend towards fewer days with FI was observed[230]. In a small, uncontrolled study with a short follow-up duration, rectal injection of botulinum toxin A improved FI symptoms 3 months later[231].

Non-pharmacological therapies. If initial therapies prove unsuccessful, transanal irrigation or barrier devices may be considered. Individuals with neurogenic bowel disorders or chronic constipation and faecal retention may benefit from transanal irrigation to facilitate rectal cleansing and prevent unwanted stool leakage[232–234]. Patients or caregivers can irrigate the rectum with tap water either with a syringe or another device[235]. Compared with conservative therapy, transanal irrigation improved FI symptoms and symptom-associated QoL in patients with spinal cord injury[236]. In observational studies, the long-term response to rectal cleansing in FI was 30–80%[237]. Mechanical insert or barrier devices may be offered for immediate relief in patients with passive FI. Older anal plugs, including a polyurethane anal plug, were not widely adopted owing to a lack of high-quality evidence of their effectiveness[238] and conflicting reports of patient discomfort[233,239,240]. New devices, such as the Renew anal insert and Eclipse vaginal bowel-control system, have shown promise and improved tolerability in open-label trials. In one open-label trial, 62% of patients treated with Renew inserts completed therapy and demonstrated ≥50% reduction in FI frequency; 78% of patients reported high satisfaction[241]. In an open-label trial of the Eclipse vaginal insert, the insert was successfully fitted in 55% of patients, of whom 78.7% reported a ≥50% improvement in FI episodes. Although further research demonstrating long-term efficacy and safety are needed, barrier devices may represent suitable therapeutic options for patients who require on-demand therapy.

Passive FI
Leakage of bowel content without noticing and/or without the ability to withhold. FI, faecal incontinence.

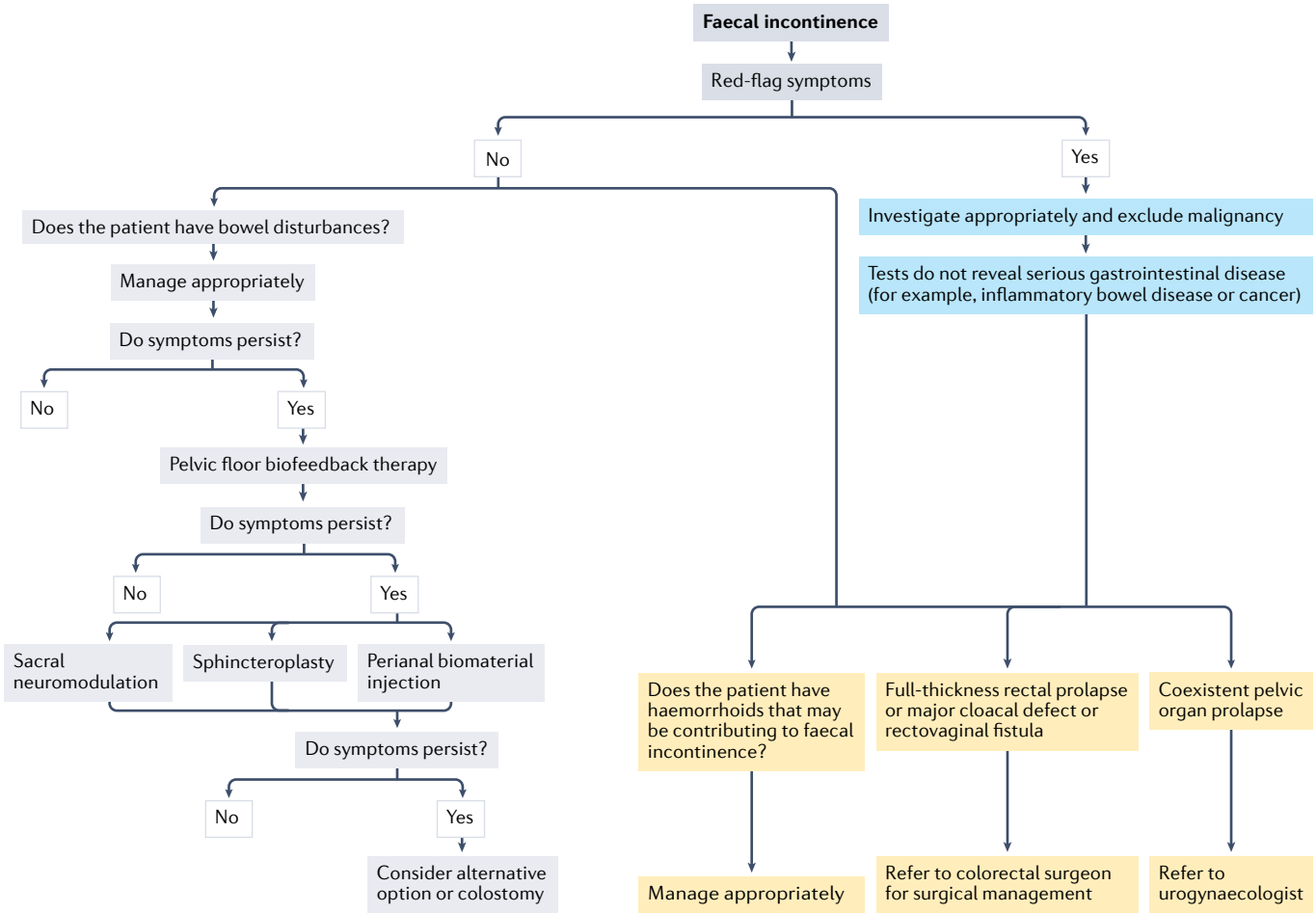

Fig. 5 | **Treatment of FI.** Patients with red-flag symptoms (for example, bloody stool or weight loss) should be managed appropriately. The next steps, arranged sequentially, are to identify and manage bowel disturbances with medications, pelvic floor biofeedback therapy that is tailored to the underlying disturbance (that is, anal hypocontractility, impaired rectal evacuation, or increased or decreased rectal sensation), sacral nerve stimulation or perianal biomaterials and, ultimately, colostomy. Transanal irrigation is a useful option for patients who are not eligible or fail to respond to other measures. Patients with major anorectal structural disturbances, such as full-thickness rectal prolapse or pelvic organ prolapse, should be referred to a colorectal surgeon or urogynaecologist, respectively. FI, faecal incontinence.

Neuromuscular stimulation and non-invasive neuromodulation. Beginning in the 1960s, several studies have electrically stimulated the anal sphincter and pelvic floor with intra-anal and intra-vaginal electrical probes, needles, and implanted stimulators. These options, some of which are available over the counter, are used as adjuncts to pelvic floor physiotherapy. In a large, randomized trial, bowel continence improved comparably with active and sham electrical stimulation[242]. SNM was introduced to manage FI in the 1990s. Percutaneous tibial nerve stimulation is a less expensive and invasive alternative neuromodulation option to SNM but, in contrast to earlier uncontrolled case series, a large randomized controlled trial observed no significant clinical benefit of percutaneous tibial nerve stimulation compared with sham electrical stimulation[243]. Another option under investigation is translumbosacral neuromodulation with repetitive magnetic stimulation; a small randomized study suggested that such stimulation at 1 Hz was more effective than at 5 Hz or 15 Hz in patients with FI[244].

Surgery

Considerations before surgery. Patients with FI generally consult surgeons when other therapies have failed or if they have a disease that overtly requires surgery. The proportion of patients with FI that meet these criteria is uncertain but they are a small minority when specific groups that do not commonly benefit from surgery are excluded, for example, those with faecal impaction, hospitalized acutely unwell, with cognitive and behavioural issues, with neurological and spinal disease, and the very elderly and/or frail. As a guide, around 1 in 10 patients attending specialist tests of anorectal function will have a clear surgical target[245] and these are typically women, aged 50–70 years, and parous. Examination and investigation in this group reveal a functionally weakened EAS with or without a defect or scarring on endoanal ultrasonography.

FIGURE 5 provides an algorithm that embodies some recommendations of the International Consultation on Incontinence sponsored by the International Continence

Cloaca
A common cavity at the end of the digestive tract for the release of digestive, excretory and genital products in vertebrates except for most mammals, including humans. In humans, a cloaca is a congenital abnormality, caused by disease or iatrogenic.

Gutter deformity
Deformity in the anal canal that prevents the canal from being closed at rest.

Society[246]. The primary objectives are to identify red-flag symptoms and overt diagnoses such as rectal impaction with overflow, large haemorrhoids or prolapsing polyps that impair anal closure. Structural problems that lead to FI, such as cloaca with or without anovaginal fistula, rectal prolapse and rectovaginal fistula, should be identified and managed. Previous non-surgical treatments, which may not have been adequately trialled, overall functional status and plans for further family should be evaluated. Most surgical approaches are contraindicated until family plans have been fulfilled.

The pathophysiology of FI should be defined by specialist radio-physiological investigations that assess IAS and EAS structure and function, rectal sensation, and rectal emptying, which may be reduced owing to a structural disturbance (such as rectocele, intussusception or functional defecation disorder)[3]. Rectal evacuation disorders and FI frequently overlap[43]. Concomitant bladder or vaginal diseases (such as overactive bladder or prolapse) that affect the management of FI should also be addressed. Before surgery is considered, pelvic floor muscle function should be optimized by expert physiotherapy, including biofeedback therapy, and targeted weight loss[247,248].

In addition to the severity of symptoms, their effects on QoL and objective disturbances, surgical management is also guided by age, surgical fitness, obesity and smoking status. In Europe, such decisions are now often made at multidisciplinary meetings, which are mandatory in some countries[249], to avoid injudicious surgery[250]. These meetings should include urogynaecology input so that multi-compartment prolapse syndromes are correctly addressed at one operation.

Surgical options. TABLE 1 lists abandoned and currently used surgical approaches for FI. The discontinuation of several procedures underscores the recognition of several important points. First, restoring anatomy frequently does not restore function. Indeed, human social defecation depends on the concerted functions of smooth and striated muscle and the peripheral and central nervous systems. Manual division, tightening and/or reinforcing tissues may not improve function; the division of nerves or scarring during these procedures may aggravate dysfunction. Hence, surgery must be tailored to address an abnormality that is likely to be contributing to FI. This is clearly the case for patients with overt rectal prolapse bypassing the sphincter complex and for those with a cloacal defect or obvious gutter deformity leading to leakage (both readily evident on inspection of the anus); however, these patients represent only a small proportion of those seen by a surgeon with FI. Second, as the perineum is subject to bacterial contamination, postsurgical infection and/or erosion are not uncommon, especially after complex reconstructions such as gracilis or gluteus transposition[251–253] and all perianal implants[254–256]. Third, procedures that tighten, such as sphincteroplasty[257] and post-anal repair[246], or augment tissue[257,258] may impair rectal evacuation, especially in patients with pre-existing defecatory disorders. Finally, the outcomes of all procedures seem to deteriorate with time. Most regress to a mean of ~50% ongoing benefit at long-term follow-up. No procedures have been compared in high-quality trials; indeed, the only high-quality evidence in the field concerns primary sphincter repair at the time of obstetric injury to prevent rather than treat FI[259].

Several surgical options, often guided by local policy, are available: SNM, sphincteroplasty, some implantable biomaterials and, as a final resort, colonic stoma.

Sphincteroplasty. Prompted by a desire to avoid harm, sphincteroplasty should be reserved for women who are young (for example, <45 years), fit and, therefore, likely to heal, with a palpable and sonographic defect and a history of obstetric injury. The patient must accept a high (>50%) risk of minor wound infection, moderate (estimated 20%) risk of wound breakdown and a small risk (estimated 5%) of complete breakdown with iatrogenic fistulation or cloaca, which may necessitate a diverting stoma[246]. They must also appreciate that sustained benefit will usually be time-limited (only ~25% of patients benefit at 10 years). However, this single intervention is relatively inexpensive and offers a short-term cure to some[246,257].

Sacral neuromodulation. For nearly all other similar patients, the default option for surgical FI management is probably SNM. Success rates of ~50% at 5–10 years are the norm in large observational cohorts and national registries, although these values depend on the method of reporting[246,260–262]. The risk of significant harm is very low but patients must recognize that they commit to a two-stage procedure, a lifelong contract with their device and physician for reprogramming, battery charging or battery changing (depending on the system), and a not uncommon need for troubleshooting interventions[263]. For example, in a Danish series of 101 patients followed up for 5 years[264], 521 reportable events, including loss of efficacy and pain, were recorded in 94 patients, leading to surgical device removal in 20 patients.

SNM is clearly favoured in patients without a sphincter defect but, in those with defects, factors favouring SNM include concomitant overactive bladder diagnosis[265] and abnormal rectal sensory function[266]. The initial procedures should be performed with a standardized technique using radiological guidance to ensure optimal electrode placement[267] and in a department with the infrastructure to support long-term follow-up monitoring. Pudendal nerve stimulation has also been trialled in specific patient groups, notably those with neurogenic incontinence, for example, owing to cauda equine syndrome, and those failing SNM. Results in small cohorts have been highly favourable[268] but reliable target access and lead fixation have hampered adoption for both urinary and faecal indications.

Other surgical options. For patients with an IAS defect and reduced anal resting tone (sphincter hypotonia), who often have predominantly passive incontinence, injectable biomaterials[269] or SNM (but not sphincter repair) may be considered. Of available biomaterials, dextranomer in stabilized hyaluronic acid, which is the only approved submucosal agent, was superior to

Table 1 | **Summary evidence for surgical treatments of FI**

Surgical approach (year first described)	Number of studies (number of participants)	Main findings	Comments
Sphincter repairs			
Anal sphincteroplasty (1923)	No RCTs (excepting comparison of repair with or without adjuncts such as levatoplasty[a]); >20 observational studies with ≥50 patients since 1990[b]; total >1,500 patients[246]	Observational data show excellent or good continence in 6–86% of patients at last follow-up (median 50% at 5 years and 25% at 10 years)	Conclusions confounded by patient selection, length of follow-up and attrition bias (patients who have stoma for complications or failure often excluded in follow-up data)
Post-anal repair (1975)	2 RCTs (n = 56) comparing post-anal repair with other forms of pelvic floor repair; in addition, 20 observational studies of variable quality with a total of >500 patients[246]	Two RCTs show poor results (42% and 27% continence); long-term observational data show ~33% success after 5 years[246]	Largely abandoned since 2010 owing to poor results
Sphincter reconstruction			
Non-stimulated (1952) and electrically stimulated (1988) muscle transposition (gracilis neosphincter or gluteoplasty)	No RCTs; 11 poor-quality observational studies (n = 618) with variations in technique	Improved continence in 50–70% of patients; mortality 1% and median 1 morbidity per patient[251] including major wound complication in 25% of patients[252]	Largely abandoned owing to complexity of surgery and risks of harm (complications of stoma, donor site chronic pain), perineal healing, and new-onset evacuation difficulty requiring further interventions such as anterograde enema procedures
Sphincter augmentation			
Artificial bowel sphincter (1987)	One poor-quality RCT (n = 14); 21 observational studies of variable quality (n = 599)	RCT underpowered for comment[297]; longer-term observational data[254,255] show up to 50% explant rate owing to implant infection/erosion and deteriorating continence in remaining patients	Device withdrawn in 2000 owing to risks of harm, especially implant infection, and need for explantation or revision
Magnetic anal sphincter (2012)	No RCTs; 2 observational studies when repeat publications excluded (n = 63)	Observational data show global satisfaction in ~50% at long-term follow-up with low (<5%) explantation rates[256]	Device withdrawn in 2018 for commercial reasons
Injectable biomaterials			
Submucosal bulking agents (1993)	5 RCTs (n = 382)[269]; 12 observational studies (n = 218)	Pivotal RCT of dextranomer in stabilized hyaluronic acid versus placebo: 52% versus 31% of patients >50% reduction in FI episodes; observational data cover 8 different agents with heterogeneous outcomes (overall 50–100% success rates quoted)	Heterogeneity of agents precludes generalization; attractive owing to outpatient application but harms reported, including proctalgia, pruritis, bleeding and abscess formation
Intersphincteric implants: GateKeeper (2011) and SphinKeeper (2016)	No RCTs; 10 small observational studies (GateKeeper, n = 137; SphinKeeper, n = 92)	Observational data show ~50% success rate at ≥12 months follow-up[273] based on reduction in symptom scores and FI episodes; no major harms	Although concerns were expressed about implant migration[272], the procedure seems to be safe
Cell therapies with AMDCs (2013) or expanded MSCs (2021)	1 RCT of AMDCs[275]; 2 observational studies of AMDCs (n = 49)[274]; 1 observational study of MSCs[298]	RCT of AMDCs showed no difference between control and active treatment; AMDC observational data show 80–90% success at ≥12 months follow-up; MSC observational study demonstrated no efficacy	Regenerative approaches (AMDCs and MSCs) seem to be safe but efficacy is variable across studies; high cost may prove prohibitive
Neuromodulation			
SNM (1994)	7 RCTs (n = 277) with varying designs and comparators; 30 observational studies with >50 patients and >12 month follow-up (n = 3,622)	RCTs support the superiority of SNM over no stimulation, bulking agents and optimal medical therapy with considerable quality caveats; observational data show 46–58% ITT success rate at >36 months follow-up	Outcome appraisal highly dependent on method of analysis (ITT versus per protocol) owing to data censoring; low risk of harm but high rates of re-intervention
Pudendal neuromodulation (2010)	No RCTs; 4 observational studies (n = 32)	Short-term/medium-term observational data show 60–100% success rates	Selected patients with failed SNM and neurogenic FI (mainly cauda equina syndrome)
Other			
Temperature-controlled radiofrequency energy delivery using SECCA device (1999)	1 small RCT and 11 observational studies (n = 226)	RCT showed no effect over sham[299]; observational data show response rates of 6–84% with a deteriorating response at longer follow-up	No reports of patients treated since 2015, suggesting a decline in popularity
Puborectal sling (1974) and transobturator posterior anal sling (2014)	No RCTs; 2 observational studies (n = 181)	52% and 69% achieved 50% reduction in FI episodes with common but minor device-related events	FDA-controlled study encouraging[300] but subsequent mesh implant concerns may limit popularity

Table 1 (cont.) | **Summary evidence for surgical treatments of FI**

Surgical approach (year first described)	Number of studies (number of participants)	Main findings	Comments
Other (cont.)			
Pelvic organ prolapse procedures	Few RCTs and numerous observational studies for multiple approaches	Performed RCTs[301] and observational studies[144] show profound FI symptom reductions	Selected population only with well-defined prolapse syndromes
Stoma	No RCTs; paucity of observational data specific to FI	Systematic review shows colostomy more cost-effective than artificial bowel sphincter or graciloplasty[302]	Strong recommendation (on low-quality evidence) supporting colostomy for end-stage FI in American Society of Colon and Rectal Surgeons Clinical Practice Guideline[276]

AMDCs, autologous muscle-derived cells; FI, faecal incontinence; ITT, intention to treat; MSCs, mesenchymal stem cell; RCT, randomized controlled trial; SNM, sacral neuromodulation. [a]Studied adjuncts include defunctioning stoma, levatoplasty and post-operative biofeedback therapy. [b]Delayed repair data including overlapping and end-to-end repairs with or without levatoplasty and/or cloacal repair.

placebo[270] but not to pelvic floor biofeedback therapy[271]. However, because of variably reported adverse events (including proctalgia, bleeding and infection[270]), intra-sphincteric materials are being studied[246,272,273]. In preliminary studies, these seem safe despite issues of accurate placement and migration[272] but high-quality controlled data are required. Regenerative medicine approaches using autologous muscle-derived cells or mesenchymal stem cells have been tried with variable outcomes[274,275]; highly encouraging observational data in selected patient populations[274] require confirmation in definitive clinical trials. Finally, the role of a stoma, usually a colostomy, should not be forgotten, especially for patients who are housebound by their symptoms and who have global pelvic floor and sphincteric dysfunction refractory to other approaches[276].

Quality of life

Effects of FI on QoL

In many people, FI considerably impairs their QoL. Some aspects, such as the effect on daily activities, are captured by questionnaires. In community women, around one in five report a moderate or severe impact on ≥1 of the 15 QoL domains that may be grouped into activities associated with predictable toilet access (for example, when working at home), unpredictable toilet access (for example, when shopping) and eating (for example, going out to eat)[277]. Patients are also bothered by the unpredictability of FI, the fear of experiencing a FI episode, the need to know where toilets are located, the fear of bad odour and the need to incorporate coping strategies[278]. Indeed, many patients drastically alter their lives[279]. Despite these consequences, patients are often reluctant, perhaps embarrassed, to share the symptom, let alone its effect on QoL, with other people[1,280]. The severity of FI is correlated with its effects on QoL[277]. Treatments that reduce FI also tend to improve the QoL[262].

Nancy J. Norton, a founder of the non-profit International Foundation for Functional Gastrointestinal Disorders, observed that patients with FI "differ widely in backgrounds and other demographic factors, but there is a common denominator: the disorder affects nearly every aspect of their daily lives. These individuals are attempting to manage bodily processes that cannot be controlled. The cost is loss of confidence, self-respect, modesty, and composure"[10]. Our impression is that the effects of FI on QoL are not adequately characterized in clinical practice.

Other consequences of FI include depression[281,282], financial burden[283], work absenteeism[284] and, arguably, institutionalization[1]. In 2010, the average annual total (direct and indirect) cost for FI was US$ 4,110 per person[283]. The contemporary impact of FI on work productivity is unknown. In 1990, a US Householder survey reported that patients with FI annually missed a mean of 15.1 days of work or school because of their FI[284].

FI management approaches have differing effects on FI-related QoL. In three randomized trials, some conservative therapies improved QoL[9,11,219]; however, differences between trial groups (for example, advice, advice plus sphincter exercises, hospital-based computer-assisted sphincter pressure biofeedback therapy, hospital and home biofeedback therapy) for effects on symptoms and QoL were not significant[9]. Compared with pelvic floor exercises, biofeedback therapy more effectively improved symptoms but not QoL[11]. Arguably, the study duration and/or the magnitude of therapeutic effects were not sufficient to improve QoL. In a prospective uncontrolled study, long-term QoL improved after sacral nerve stimulation for FI[262]. In a randomized controlled trial, FI-related QoL improved to a greater extent after sacral nerve stimulation than percutaneous nerve stimulation[285].

QoL instruments in FI

Similar to other conditions, the overall QoL of patients with FI comprises overall well-being, health-related QoL, such as a condition that predisposes to FI, and QoL related to FI. Selected generic QoL instruments (such as EuroQol (EQ-5D)) distinguish women with and without FI and are useful for comparing the effect of FI and other diseases on QoL[286]. TABLE 2 shows selected FI QoL instruments; other instruments are discussed elsewhere[287]. The FI-specific QoL instruments (such as the FISS) evaluate the effect of FI on QoL in a more granular manner than generic QoL scales. Other scales used to assess FI QoL enquire about the effects of bowel symptoms, and not FI, on QoL[288,289]. The Rockwood and the FISS-QoL instruments also include separate questions that cover the severity of FI[18,277,290,291].

Table 2 | **Comparison of instruments for rating the effect of FI on QoL**

Instrument	Faecal Incontinence Quality of Life Scale[290]	MHQ[292] and MMHQ[293a,b]	CRAIQ[294] and PFIQ-7 (REF.[295])[b]	Faecal Incontinence Severity Scale–QoL instrument[277]	International Consultation on Incontinence Questionnaire–Bowel Symptoms[288b]
Initial report, year	2000	2001 (MHQ) and 2005 (MMHQ)	2001 (CRAIQ) and 2005 (PFIQ-7)	2006	2011
Daily living	X			X	X
Activities with more predictable toilet access					
Employment		X	X[c]	X	
Work at home (household chores)		X	X[c,d]	X	
Family life		X	X[c]	X	
Eat before leaving home	X			X	
Stay overnight away	X		X[c]		
Visit friends at home or social life	X	X	X[c,d]	X	
Sex life	X	X	X[c]	X	X
Hobbies or leisure			X[c]		
Activities with less predictable toilet access					
Movie or church	X		X[c,d]	X	
Shopping			X[c]	X	
Walking		X	X[c,d]		
Exercise		X	X[c,d]	X	
Ability to leave home	X		X[c]	X	X
Volunteer activities			X[c]		
Travel by car or bus (shorter distances)			X[c]	X	
Travel by plane or train (longer distances)	X	X	X[c,d]	X	
Sleeping		X	X[c]		X
Rushing to toilet	X	X			X
Unpredictability or uncertainty about toilets	X		X[c]		X
Eating					
Going out to eat	X			X	
Amount of food	X	X			
Management					
Wearing pads		X		X	X
Taking medications		X		X	X
Effect on mental health					
Depression	X	X	X[c,d]	X	
Nervousness	X	X	X[c,d]		
Frustration, anger or fear			X[c,d]		
Embarrassment	X	X	X[c]		X
Validation					
Content validity	A	B[b]	B[b]	A	B[b]
Test–retest reliability	A	A	A	B	A
Internal consistency	A	A	A	B	A
Construct validity	A	A	A	A	A
Criterion validity	A	A	B	A	A
Sensitivity to change	A[303]	A[304]	A	B	A

'A' refers to attributes that have been partly or adequately validated. 'B' refers to attributes that have not been validated. For cells with an 'A' rating that do not include a citation, the citation is provided in the column heading. CRAIQ, Colorectal-Anal Impact Questionnaire; FI, faecal incontinence; MHQ, Manchester Health Questionnaire; MMHQ, Modified Manchester Health Questionnaire; PFIQ-7, Pelvic Floor Impact Questionnaire-short form 7; QoL, quality of life. [a]The domains marked X in this column refer to the MMHQ. [b]The questions in this instrument enquire about the effects of bowel symptoms, not specifically of faecal incontinence, on QoL. [c]Items that are in CRAIQ. [d]Items that are in PFIQ-7.

Box 3 | **Selected key priorities for future research in FI**

Epidemiology

- What is the impact of temporal trends in obstetric practices (for example, utilization of operative vaginal deliveries) on the incidence of faecal incontinence (FI)?
- What is the natural history of women with anal sphincter injury and FI in the community?
- Developing a more refined understanding of the relationship between symptoms of FI and its effect on quality of life

Mechanisms of normal and disordered anorectal functions

- Mechanisms responsible for generating tone in the internal and external anal sphincters
- Mechanisms of normal and disordered rectal accommodation
- Relationship between stool consistency and recto-anal functions
- Contribution of reflexes (for example, postural pelvic floor reflex, cough reflex and sampling reflex) to normal and disordered faecal continence
- Understanding the brain-to-anal sphincter neuronal axis in health and FI with new tools
- Understanding anorectal functions in non-human primate models

Management of FI

- Effects of anti-diarrhoeal agents on FI
- Effects of constipation management on FI
- Comparative trials of various treatments for FI
- Developing and assessing regenerative medicine therapies for FI
- Understanding mechanisms of action of therapies used to treat FI
- Assessing the long-term effects of therapies used to treat FI

Somatization
A personality trait to respond with somatic symptoms to psychological stress.

The Rockwood QoL scale is probably the most widely used FI QoL instrument in research studies[290]. The Manchester Health Questionnaire is administered on paper[292]. Designed specifically for a telephone interview, the Modified Manchester Health Questionnaire combines severity and FI-related QoL questions from the Fecal Incontinence Severity Index and the Manchester Health Questionnaire, respectively[293]. The Pelvic Floor Impact Questionnaire (PFIQ) includes 93 items, of which 31 items each are dedicated to urinary incontinence, pelvic organ prolapse and colorectal symptoms (using the Colorectal-Anal Impact Questionnaire)[294]. This is useful, as some patients with FI are also bothered by urinary incontinence and/or pelvic organ prolapse. The Pelvic Floor Impact Questionnaire-short form 7 (PFIQ-7) is a condensed version of the PFIQ[295]. The FISS severity scale has been extensively validated but the FISS-QoL instrument needs additional validation[277].

Physicians tend to focus on the severity of FI rather than on its effects on QoL. The severity of FI is correlated with its overall impact on QoL[277,296]. In addition to FI severity, somatization also independently predicts the QoL in patients with FI; greater somatization scores were associated with lower QoL[296]. The QoL was also worse in patients with FI and concurrent constipation than in those without concurrent constipation[44].

Outlook

Over the past decade, our understanding of the epidemiology and pathogenesis of FI has evolved substantially. Several instruments to rate the severity of FI and QoL have been developed and validated. Newer therapeutic options, including SNM, anal barrier devices and anal injection of biomaterials, are available and several controlled trials of newer and older therapies, including dietary fibre supplementation and biofeedback therapy, have been conducted. Ongoing trials are comparing the efficacy, safety and expense of these treatments and those of regenerative approaches to restore anal sphincter and neuromuscular function[12]. These advances have been primarily spurred by initiatives from key funders and through awareness of FI by international patient organizations. Nonetheless, FI remains a common condition that is often unrecognized by clinicians and can have devastating effects on the QoL of patients. Over time, it is conceivable that the incidence of FI may decline together with the reduced utilization of operative vaginal delivery, which predisposes to anal sphincter injury. However, that decline may be offset by increasing consultation for FI; hence, health-care utilization for FI may not change substantially. We endorse the broad research priorities identified by speakers at a State of the Science Workshop sponsored by the National Institutes of Health in 2013 (REF.[6]) and highlight key selected priorities for future research in BOX 3.

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Competing interests
A.E.B. is a consultant for Allergan, Medical Insights Group and GI Supply; receives royalties from Medspira; has patents with Medtronic, Medspira, and Minnesota Medical Technologies; and is listed as an inventor on the following patents relating to anorectal devices: WO2011014530A2 (applicant: Mayo Clinic; status: issued); US201562239397 (applicant: Minnesota Medical Technologies; status: issued); WO2014/068560 (applicant: Mayo Clinic and Medtronic; status: issued). C.H.K. is a consultant for Medtronic, Coloplast, Cook Myosite, Saluda Medical, Motilent, Takeda, Enteromed, Exero Medical, Alimentary Health, Amber Therapeutics, and Enterika; is on the speaker board of Medtronic; has shared or receives royalties from Amber Therapeutics and Enterika; and is listed as an inventor on the following patents for a surgical instrument and a pudendal nerve stimulation device: US11058405B2 (applicant: Queen Mary University of London; status: issued), US application serial number 63/160,322 (applicant: Amber Therapeutics Ltd, London, UK; status: filed). A.M. is a consultant for Allergan. P.E. is a consultant for Alimentary Health, Aptiny, Arena, Cemet, Indigo, SymbioPharm, and 4DPharma and is on the speaker Board of Alimentary Health, Biocodex, Biogen, Indigo, MDC, Medice, Merz, and Sanofi. S.R. is a consultant for InTone MV, Allergan, Ironwood, Neurogut, and Laborie. S.M.S. is a consultant for Laborie. A.S., I.M. and N.O. declare no competing interests.

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