Office-Based Management of Fecal Incontinence

Vanessa C. Costilla, MD, Amy E. Foxx-Orenstein, DO, Anita P. Mayer, MD, and Michael D. Crowell, PhD

The authors are affiliated with the Mayo Clinic in Scottsdale, Arizona. Dr. Costilla is a Resident in the Department of Internal Medicine. Dr. Mayer is an Assistant Professor in the Division of Women's Health Internal Medicine. Dr. Crowell is a Professor and Dr. Foxx-Orenstein is an Associate Professor in the Division of Gastroenterology.

Address correspondence to:
Dr. Amy E. Foxx-Orenstein
Division of Gastroenterology
Mayo Clinic
13400 East Shea Blvd
Scottsdale, AZ 85259;
Tel: 480-301-6990;
E-mail: Foxx-Orenstein.Amy@mayo.edu

Abstract: Fecal incontinence (FI) is a devastating disorder that is more prevalent than previously realized. FI is the involuntary loss of stool. Many factors contribute to the pathophysiology of FI, including advanced age, bowel irregularity, parity, and obesity. A detailed history and focused rectal examination are important to making the diagnosis and determining contributing causes. Although multiple diagnostic studies are available to assess the cause of FI, specific guidelines that delineate when testing should be done do not exist. Clinicians must weigh the risk, benefit, and burden of testing against the need for empiric treatment. All types of FI are initially managed in the same way, which includes lifestyle modification to reduce bowel derangements, improved access to toileting, and initiation of a bulking regimen to improve stool consistency. If initial conservative management fails, pharmacologic agents, biofeedback, or surgery may be indicated.

Keywords
Fecal incontinence, anal incontinence, obesity incontinence, bariatric surgery incontinence, fecal seepage, flatal incontinence

Fecal incontinence (FI) is a socially and emotionally devastating disorder that significantly affects the lives of patients and their families. Anal incontinence is the more general term, which refers to the involuntary loss of gas or stool. FI specifically refers to the involuntary loss of stool.[1] FI is more common than previously thought, with a prevalence that varies by the population studied.[2] Among institutionalized persons, prevalence has been reported to be as high as 45%.[3] The prevalence of FI is similar between men and women at 7.7% and 8.9%, respectively, and increases with age, reaching 15.3% in persons age 70 years or older.[4]

Because of social stigma, many patients do not seek treatment, which suggests that prevalence in the general population is underestimated. In a self-reported survey, 36.5% of primary care patients reported episodes of FI, but only 2.7% of these patients had a documented diagnosis.[5] Healthcare costs are 55% higher in patients with FI than continent patients,[5] amounting to an estimated $11 billion annually.[6]

Primary care physicians, gastroenterologists, and gynecologists play a vital role in FI screening. Most patients can achieve significant

Table 1. Fecal Incontinence Subtypes

	Mechanism	Outcome
Passive incontinence	Internal sphincter weakness or tear	Loss of rectosigmoid perception and/or impaired rectoanal reflexes
Urge incontinence	Disruption of the external sphincter function	Diminished rectal capacity
Fecal seepage	Normal sphincter function	Incomplete evacuation of stool and/or impaired rectal sensation

improvement in symptoms through proper treatment. In addition, early diagnosis will prevent complications that can further reduce quality of life.

Pathophysiology

Bowel function and continence are controlled by multiple factors. Effective evacuation of fecal material involves a complex interaction of structural and sensory components within the anorectal unit and pelvic floor musculature.[7] Structural components involved in defecation begin with the rectum, a muscular tube approximately 12–15 cm long terminating at the anus.

The sphincter of the anus is composed of 2 muscular components: the internal anal sphincter (IAS) and the external anal sphincter (EAS). The IAS comprises the smooth muscle component of the anal sphincter, providing up to 80% of the resting anal canal pressure.[8] Smooth muscles of the IAS operate under involuntary control and are responsible for tonic activity that maintains the anal barrier at rest. Striated muscles of the EAS allow voluntary squeeze to further maintain continence. The puborectalis (PR) muscle, which forms a sling around the rectum, further augments these barriers. The PR is contracted at rest and maintains the anorectal angle at approximately 90°. During defecation, this angle becomes obtuse, allowing for passage of stool. With voluntary squeeze, this angle becomes acute to ensure continence.

Stool arriving in the rectum results in rectal distension and a reflexive decrease in anal resting pressure, which allows fecal material sampling by the sensitive anoderm.[1] An urge to defecate occurs based on the solid, liquid, or gas nature of the rectal contents. If the urge to defecate occurs at a socially inappropriate time, sympathetically mediated inhibition of rectal smooth muscle with voluntary squeeze of the EAS and PR occurs. Adequate rectal compliance is required for deferred defecation because the rectal contents are forced back into the rectal reservoir to await an acceptable time for defecation.[7,9,10]

Table 2. Risk Factors for Fecal Incontinence

Abnormal stool consistency – Liquid stool, fecal impaction
Female sex
Pregnancy
Birth trauma – Episiotomy, use of forceps, tear, prolonged labor
Parity
Perianal surgery or trauma – Hemorrhoidectomy, sphincterotomy, anal dilation
Neurologic causes – Dementia, cauda equina, stroke, multiple sclerosis, spinal cord lesions, neuropathy
Inflammation – Radiation, inflammatory bowel disease with anal fistula, wiping irritation
Hemorrhoids
Pelvic organ prolapse or rectal prolapse
Congenital anorectal abnormality
Obesity
Bariatric surgery
Limited mobility
Urinary incontinence
Cigarette smoking
Chronic obstructive pulmonary disease

FI results when continence mechanisms are compromised.[11] Disorders that reduce stool consistency, weaken striated pelvic floor muscles or the IAS, impair sensation, alter colonic transit time or stool volume, or compromise cognitive functioning can all contribute to loss of continence.[7] Subtypes of FI include passive incontinence, urge incontinence, and fecal seepage (Table 1).[7,12]

Risk Factors

Many factors contribute to impaired continence. These factors include liquid stool consistency, female sex, advanced age, multiparity, neurologic injury or disease, prior trauma, and poor general health (Table 2).[13,14] Less known risk factors include obesity, smoking, chronic obstructive pulmonary disease, pregnancy, hysterectomy, urinary incontinence, and bariatric surgery.[13,15-17] Diarrhea is by far the greatest risk factor for FI. One study found that women with diarrhea had an odds ratio of 53 (95% confidence interval, 6.1–471) of hav-

ing inadvertent loss of stool.[14] Rectal urgency with or without loose stools is a primary risk factor more than is obstetric injury.[18]

The incidence of FI increases significantly with age, attributed mostly to weak pelvic floor musculature and decreased anal resting tone. One study found anal resting tone decreased by approximately 0.5 mmHg per year and, in women, 3 mmHg per birth.[19]

Rectal sensation is not directly affected by age or parity.[19] Parity is associated with frequent sphincter defects due to repetitive trauma during delivery. Evidence shows a relationship between pelvic floor disorders and operative or traumatic vaginal delivery.[20,21] A method to determine which women are at greatest risk for FI does not exist, and current literature does not support a significant benefit of cesarean delivery over nontraumatic vaginal delivery for pelvic floor health or preserving anal continence.[22,23] Advising cesarean section to prevent pelvic floor disorders is not recommended[24] because pregnancy alone is considered a risk factor for incontinence.

Obesity is a risk factor for FI.[25] FI has been reported to be nearly 50% more prevalent in obese women compared with women of normal weight.[26,27] Although bariatric surgery is the most effective treatment for morbid obesity, post–bariatric surgery patients are also at significant risk for FI due to changes in stool consistency. Forty-eight percent of women and 42% of men post–bariatric surgery reported liquid stool incontinence, and 21% of women and 30% of men reported solid stool incontinence.[16]

In younger women, FI is strongly associated with functional bowel disorders, including irritable bowel syndrome (IBS), constipation, and diarrhea. In one study, 22% of patients with IBS reported at least occasional seepage and FI, attributable to deranged bowel habits.[28]

For both genders and across all age ranges, the causes of FI are manifold and may overlap. Many patients have sphincter injuries that remain asymptomatic for years until they experience age- or hormone-related changes, such as muscle or tissue atrophy, which then limit the ability to compensate for their remote injury.

Clinical Evaluation

A detailed history and focused rectal and neurologic examinations are important to making the diagnosis and determining potential contributing causes. The history should include an evaluation of medications and dietary habits that may alter bowel frequency and stool consistency. Obtaining a bowel diary can be helpful. It should include information on the number of episodes, product (gas, mucus, liquid, or solid), volume, stool consistency, ability to sense stool, urgency, straining, and feeling of constipation. Table 3 provides a checklist for questions that patients being evaluated for FI should be asked.[29] Neurologic evaluation should assess cognition, strength, and gait.

The perineum should be inspected for moisture, skin irritation, feces, scars, anal asymmetry, fissures, and gaping of the sphincter. The presence of an anal wink and perineal sensation should be ascertained. Anal

Table 3. Fecal Incontinence History Checklist

Medical history
• Diabetes mellitus • Cognitive impairment • Neurologic disorder (eg, stroke, spinal cord disease, Parkinson disease) • Inflammation (eg, inflammatory bowel disease–associated, infectious, ischemic, microscopic, radiologic) • Constipation • Irritable bowel syndrome • Anal cancer • Connective tissue disease (eg, scleroderma, lupus)
Surgical history
• Anorectal • Pelvic • Bariatric
Obstetric history
• Pregnancy (parity, prolonged delivery) • Vaginal deliveries with trauma (episiotomy, tear, forceps)
Functional status
• Limited mobility (use of wheelchair, walker) • Living situation
Medication list (not all inclusive)
• Diarrhea provoking: laxatives, orlistat, metformin, donepezil, rivastigmine, antibiotics, magnesium, selective serotonin reuptake inhibitors • Constipation provoking: loperamide, diphenoxylate/atropine, opioids, tricyclic antidepressants, verapamil, memantine, calcium
Diet (not all inclusive)
• Diarrhea provoking: prunes, plums, beans, alcohol, artificial sweeteners, lactose-containing foods, caffeine
Bowel pattern and stool characteristics
• Frequency of bowel movements • Stool consistency • Variability in stool consistency • Urgency, ability to arrive to the toilet in time • Ability to control flatus • Awareness of leakage • Volume of fecal loss • Exacerbating or relieving factors

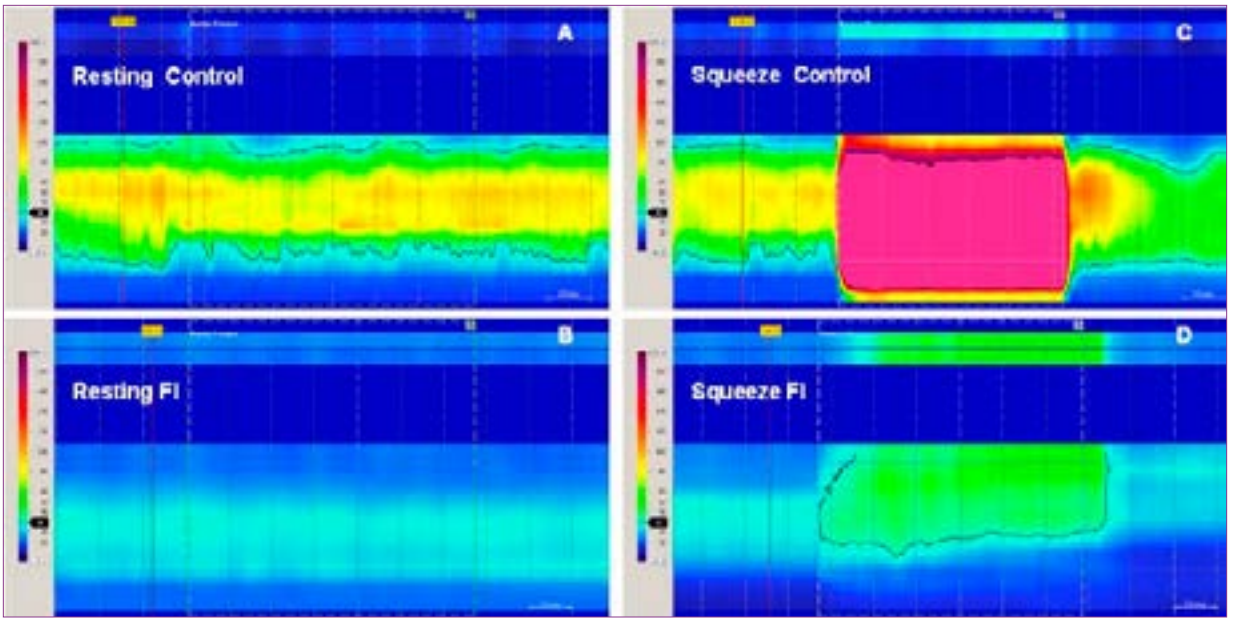

Figure 1. High-resolution anorectal manometry showing a high-resolution topographic contour plot of resting anorectal motor function in a healthy control with normal resting internal anal sphincter tone **(A)** and in a patient with fecal incontinence (FI) and weak internal anal sphincter tone **(B)**. Resting pressures in the patient with FI never exceed 30 mmHg. The high-resolution topographic contour plot shows maximal squeeze pressures in a healthy control with normal external anal sphincter squeeze pressure and endurance **(C)** and in a patient with FI demonstrating weak external anal sphincter squeeze pressure and rapid fatigue **(D)**. The resting and squeeze event windows are shown within the dashed white lines. Pressures in mmHg are calibrated to the color contour chart on the left. A solid black contour line delineates all pressures at 30 mmHg or above.

spasm can sometimes be identified visually. When bearing down briefly as if to evacuate, the degree of perineal descent and any bulging or prolapse from the rectum or vagina should be noted. Check for the presence of large or prolapsed hemorrhoids, as they can cause soiling.

A digital rectal examination is key for identifying anatomic abnormalities. Sharp, knifelike pain indicates active mucosal injury such as an acute or chronic fissure, ulcer related to injury, infection, or possible inflammatory bowel disease (IBD). Check for subtle anal spasms; lax or intense anal tone at rest and with bearing down provides potential clues to pelvic floor disorders.

Diagnostic Studies

Although multiple diagnostic studies assess the cause of FI, specific guidelines delineating when testing should be done do not exist. Clinicians must weigh the risk, benefit, and burden of testing against the need for empiric treatment. Consideration is given to a patient's ability to participate in testing, comorbidities, and the potential diagnostic yield of the study.[30] Diagnostic testing can aid in the following clinical scenarios: 1) suspected anal sphincter injury, 2) overflow incontinence versus sphincter injury, 3) suspicion of pelvic floor dysfunction, 4) noncorrelation of the history and examination, and 5) when testing could eliminate other causes of FI.

Endoanal Ultrasound

Endoanal ultrasound remains the standard for identifying anal sphincter injuries, including tears, scars, atrophy, and anal fistulae.[31] It provides excellent resolution of the IAS but is less accurate with the EAS. EAS imaging is operator-dependent. Distinguishing the EAS from perirectal fat is challenging because both structures are echogenic.

Anorectal Manometry

Anorectal manometry systems quantify IAS and EAS function, rectal sensation, and compliance.[1] The water-perfused probe is least expensive and traditionally used. Solid-state probes with closely spaced pressure sensors are becoming more common. High-resolution 3-D manometry with up to 256 sensors are now available to evaluate pressure profiles and topographic changes, which might increase diagnostic yields.[1]

Anorectal manometry should be performed in laboratories with experienced interpretive personnel. Anal

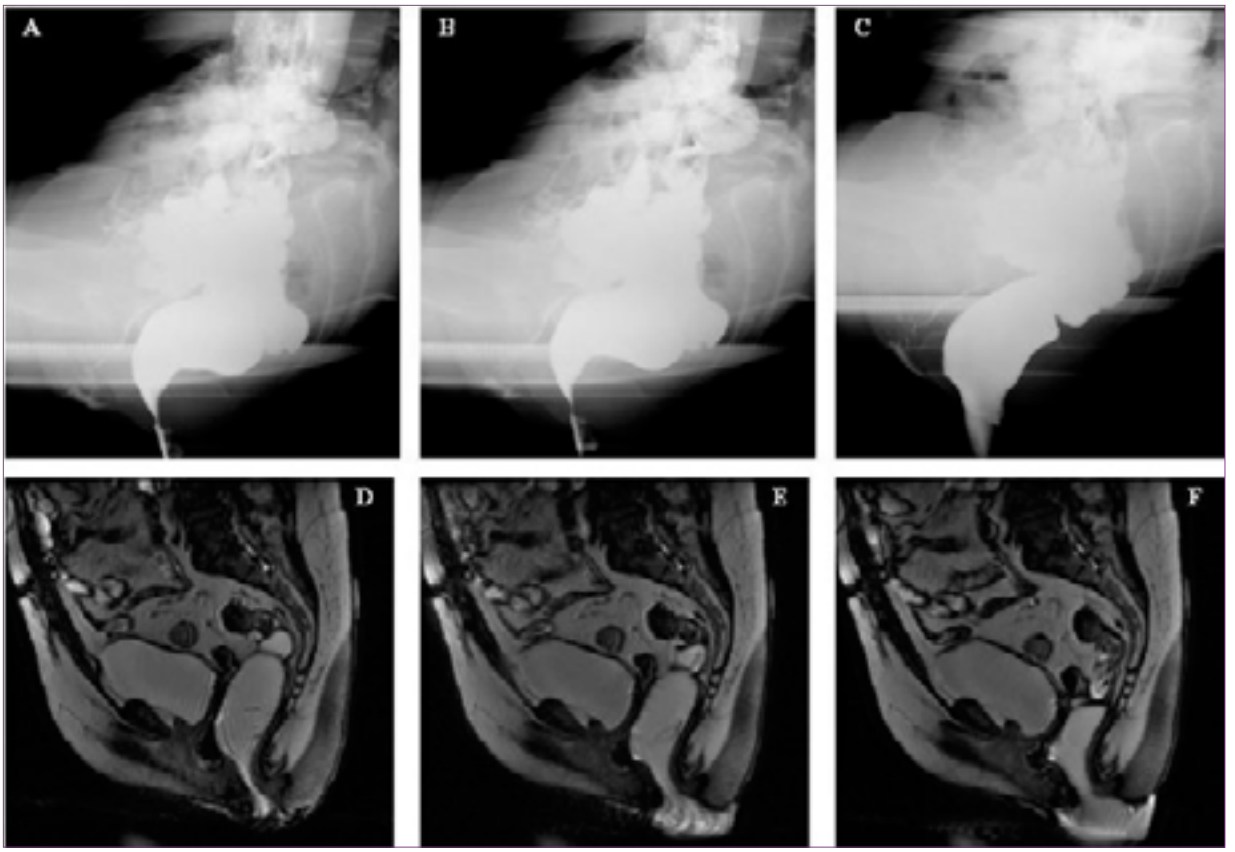

Figure 2. Defecography anorectal images at rest **(A)**, squeeze **(B)**, and evacuation **(C)** and corresponding dynamic pelvic magnetic resonance imaging **(D–F,** respectively**)**.

resting and squeeze pressures and rectoanal inhibitory reflex should be measured. Anal resting and squeeze pressures are often low in FI, suggesting weak IASs and EASs, respectively. Other factors, such as watery stool or reduced sensation, may contribute to FI in patients with normal anal pressures. Figure 1 illustrates resting and squeeze pressures using high-resolution manometry.

The rectal balloon distention test measures rectal sensation and compliance by assessing sensory-motor responses to incremental volumes of air or water.[32] Sensation may be normal, reduced, or increased in patients with FI. Reduced sensation may allow stool leakage before the EAS can contract. Patients with increased rectal sensation may have rectal hypersensitivity or reduced rectal capacity.[9] Patients with reduced compliance or capacity experience rectal urgency and frequent defecation.

The balloon expulsion test can help identify, but does not exclude, a functional defecation disorder.[31] A balloon is inflated with water to a fixed volume, usually 50 mL, inside the rectum. The patient is then seated on a commode and asked to expel the balloon. The test is normal if the patient expels the balloon within 60 seconds. This test is often used in screening for chronic constipation.

Standard Defecography and Pelvic Magnetic Resonance Imaging

Standard defecography provides dynamic evaluation of the pelvic floor and can indicate the presence of rectal prolapse, enterocele, rectocele, and cystocele.[33] Oral liquid barium delineates the small intestine. Thick barium paste is inserted into the rectosigmoid, and then dynamic anatomy and pelvic floor motion images are recorded with the patient at rest, coughing, squeezing, and straining to expel the barium.[31] Defecography is not standardized across institutions and is not widely available.

Dynamic pelvic magnetic resonance imaging (MRI) is the only imaging modality that can evaluate global pelvic floor anatomy as well as the anal sphincter without radiation exposure.[34] This modality has played a key role in identifying mechanisms of difficult or complex bowel function. It is expensive and not widely available. Figure 2 compares standard defecography with dynamic pelvic MRI.

MRI of the anal sphincter provides superior spatial resolution of the IAS and EAS.[30,34] The EAS can be distinguished from surrounding perirectal fat, allowing better diagnosis of EAS atrophy.[9] However, MRI is costlier than defecography and not widely available.

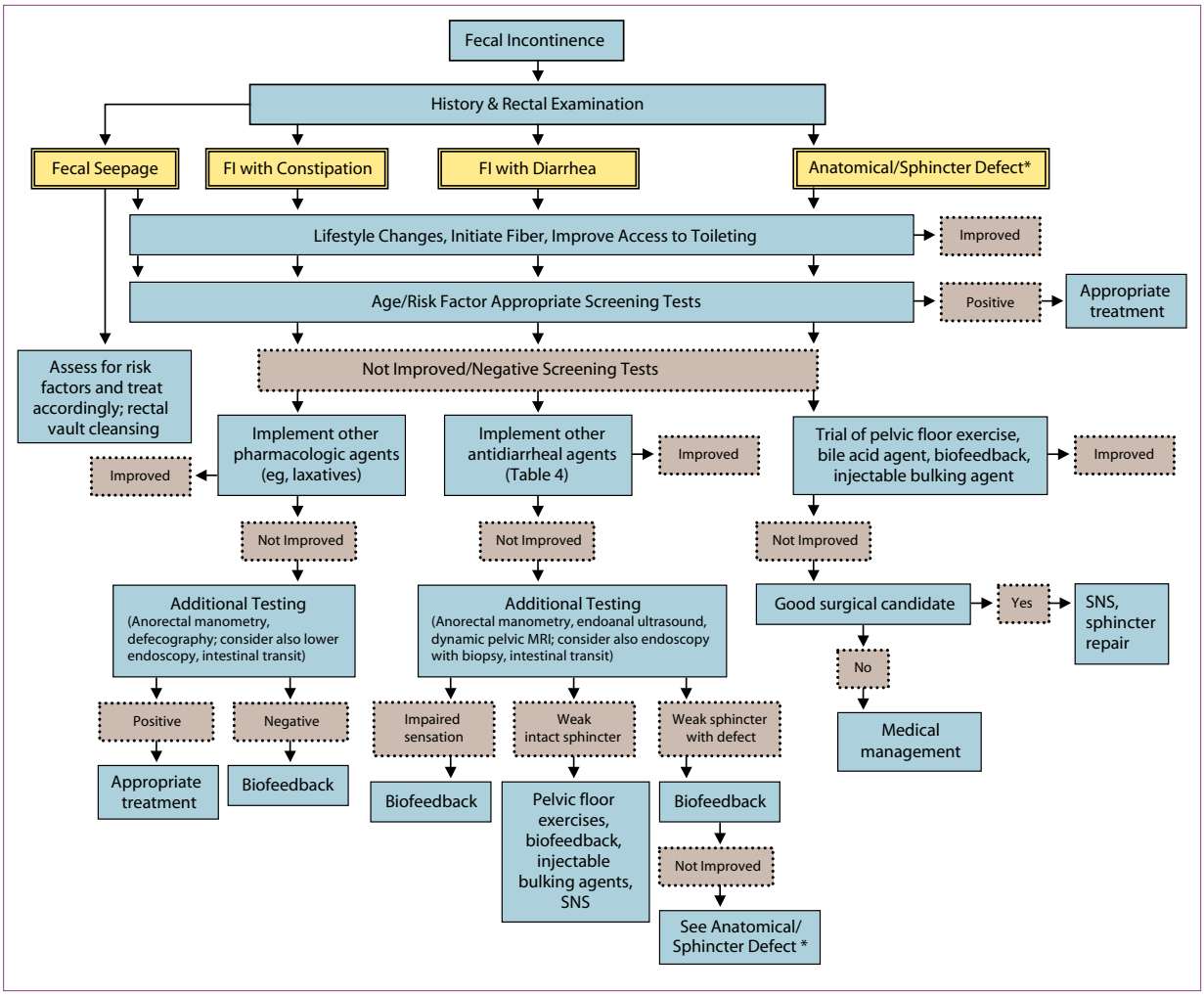

Figure 3. An algorithm for the management of fecal incontinence (FI).
MRI=magnetic resonance imaging; SNS=sacral nerve stimulation.

Neurodiagnostic Studies

Anal electromyography may identify sphincter denervation, myopathic damage, neurogenic damage, or a mixed injury.[31] Needle or surface electrodes may be used. However, mapping of sphincter defects is no longer recommended.[35]

Pudendal nerve terminal motor latency (PNTML) measures the muscular integrity between the terminal pudendal nerve and the anal sphincter,[1] helping to determine whether sphincter weakness is due to pudendal nerve injury, sphincter injury, or both. PNTML was previously thought to identify patients likely to benefit from surgical repair of sphincter defects. Recent studies do not support this assertion, however,[36] and the American Gastroenterological Association recommends against PNTML testing for evaluation of FI.[32]

Stool tests and intestinal transit studies may be used to explain the cause of a patient's underlying diarrhea or constipation. Endoscopy may be necessary to diagnose diseases that exacerbate FI, such as IBD, celiac disease, or microscopic colitis. Ultimately, ascertaining the principal cause of the patient's FI is essential because it will direct treatment strategies and affect clinical outcomes.

Management

All types of FI are initially managed in the same way. Management consists of lifestyle modification to improve bowel derangements, access to toileting, and initiation of a fiber regimen (Figure 3).

Lifestyle Modification

Lifestyle modification may improve FI. Patient education is important to help ensure adherence. Among other issues, medication use and diet should be examined. Polypharmacy is common, especially in older adults. Diarrhea is a common adverse effect of medications, with some causing loose stools or increased gastrointestinal motility. As such,

medications that exacerbate FI, including over-the-counter herbs and vitamins, should be reviewed and adjusted.

A patient's diet can be evaluated to elicit factors that exacerbate FI, including diets that are high in sugar and caffeine. Alternatively, a diet high in fiber may improve stool consistency and decrease episodes of FI and seepage. Dietary changes to prevent loose stool should be pursued.

Some studies show that daily exercise improves FI, while other studies suggest that exercise has no effect on the frequency of FI episodes.[37] Nevertheless, physical therapy and exercise that improve patient mobility will improve toileting opportunities and toilet accessibility. Additionally, FI frequency decreases in obese patients who lose weight.[25]

The use of absorbent products is a common self-care practice for patients with FI. Few products are specifically designed to absorb fecal material. Patients report using a variety of products such as pads, panty-liners, and pull-up briefs, which are all originally designed for urine or menstrual leakage.[38,39] Persons more likely to use these products include women, those reporting a higher FI severity score, and the elderly. Containment of anal leakage with the use of pads is problematic because of the odor produced and resultant skin conditions due to contact of the skin with fecal matter.[40,41]

Use of anal plugs is another option for patients with FI. Anal plugs come in different designs and sizes and are meant to block involuntary loss of stool.[42,43] A Cochrane systematic review concluded that patient tolerance of the anal plug limits its usefulness; however, if tolerated, anal plugs are effective in preventing incontinence.[44]

Limited mobility contributes to FI, especially in elderly and physically impaired patients. Limited mobility may be alleviated by scheduled toileting and changes within the home to allow better toilet access. Examples include moving a patient's sleeping area closer to a bathroom or providing a portable bedside commode. Furthermore, ensuring access to assist devices, including walkers and canes, may help patients get to a toilet in time.

Pharmacologic Therapy

Initial efforts should focus on modifying stool formation because formed stool is much easier to control than loose stool. Initiation of bulking agents such as methylcellulose, psyllium, or partially hydrolyzed guar gum is often effective. Pharmacotherapy for diarrhea with agents such as loperamide, diphenoxylate/atropine, alosetron (Lotronex, Prometheus), clonidine, cholestyramine, colestipol, probiotics, tincture of opium, and amitriptyline is usually reserved for patients with more refractory symptoms that do not respond to conservative therapies.[45] Guidelines for the use of common antidiarrheal medications in FI are provided in Table 4.

Patients with diarrhea-predominant IBS require special consideration because fiber therapy may exacerbate abdominal pain and bloating, contributing to poor compliance. If these symptoms do not abate after 7 days, initiation of other pharmacotherapy, including loperamide, a tricyclic antidepressant, probiotics, or alosetron, may provide more effective relief for this subset of patients.[46]

Chronic constipation may lead to rectal distention resulting in a chronically enlarged rectum and altered rectal sensation. Increased rectal capacity and decreased rectal sensitivity can increase a patient's risk of overflow incontinence. In taking a history, it is important to clarify the volume and consistency of fecal loss because overflow incontinence will most likely be associated with small volume, liquid, or soft stool loss without a preceding normal bowel movement. Overflow incontinence is particularly prevalent in elderly patients.

Once constipation has been identified as the underlying cause for the patient's FI, an empiric trial of fiber therapy should be initiated. Although stool softeners such as docusate are often used in clinical practice, fiber should be the first line of therapy. A large study comparing the efficacy of psyllium with docusate found that psyllium was significantly superior to docusate in providing relief of constipation.[47]

Fiber should be initiated in low doses and titrated gradually to 25–30 g daily (including dietary fiber). Caution is advised when initiating fiber therapy in patients with IBS, as it may worsen symptoms of abdominal bloating and discomfort.

Additional pharmacologic agents should be reserved for patients not responding to or intolerant of conservative interventions. When recommending laxatives, the practitioner should take care not to stimulate excessive gas production that can contribute to flatal incontinence. Nonabsorbable sugars (eg, lactulose and sorbitol) draw water osmotically into the intestinal lumen, stimulate colonic motility, and can cause abdominal discomfort and flatulence. A review by Gallegos-Orozco and colleagues of agents useful in the treatment of constipation was published in 2012 in the *American Journal of Gastroenterology*.[48]

Fecal seepage is distinctly different from FI in that it usually involves the loss of small liquid or soft stool after a normal bowel movement.[12,49] However, patients may report an abnormal bowel habit or symptoms more consistent with anal sphincter dysfunction, which may not be detected as a physiologic abnormality on objective anorectal testing. Seepage may be caused by hemorrhoids, poor hygiene, anal fistula, rectal prolapse, and hypo- or hypersensitivity of the rectum.

Interestingly, fecal seepage is more prevalent in men and patients with preserved anal sphincter and rectal function.[49] In patients with fecal seepage, assessment and treatment of a specific cause may resolve symptoms. If symptoms persist, clearance of the rectal vault should be performed at regularly scheduled times each day, regardless

Table 4. Common Antidiarrheal Medications in Fecal Incontinence

Medication	Adult dosing	Adverse effects	Special considerations
Fiber supplementation	Powder preferred. Begin 1–2 teaspoons or tablets of preferred formulation twice daily. Titrate to 25–30 g/day (includes diet + supplement).	Flatulence, bloating, abdominal pain, anorexia	• May interfere with absorption of other medications. • May reduce insulin requirements.
Loperamide	Begin at 2 mg PO twice daily. May titrate to 4 mg twice daily as needed. If larger doses are needed, slowly titrate.	Paralytic ileus, rash, fatigue, cramping, constipation, nausea, vomiting	• May increase resting anal sphincter tone. • Cautious use with active inflammatory disease of the colon or infectious diarrhea.
Diphenoxylate/atropine	Begin 2 tablets of diphenoxylate 2.5 mg/atropine 0.025 mg daily. Titrate up slowly to a maximum of 2 tablets 4 times per day.	Toxic megacolon, CNS effects	• Atropine may cause anticholinergics effects. • Cautious use with active inflammatory disease of the colon or infectious diarrhea.
Cholestyramine	Begin 4 g PO daily. Maximum dose is 24 g/day.	Flatulence, nausea, dyspepsia, abdominal pain, anorexia, sour taste, headache, rash, hematuria, fatigue, bleeding of gums, weight loss	• May interfere with absorption of other medications. • Contraindicated in patients with biliary obstruction.
Colestipol	Begin 2 g PO daily. Titrate to a maximum of 16 g/day.	Gastrointestinal bleeding, abdominal pain, bloating, flatulence, dyspepsia, liver dysfunction, musculoskeletal pain, rash, headache, anorexia, dry skin	• May interfere with vitamin and medication absorption.
Clonidine	Begin 0.1 mg PO twice daily. May increase to 0.3 mg twice daily.	Rebound hypertension, dry mouth, sedation, CNS effects, constipation, headache, rash, nausea, anorexia	• Wean off medication slowly if ineffective.
Tincture of opium	Begin 1–2 drops PO twice daily. Slowly titrate up to a maximum dose of 12 drops twice daily.	Sedation, nausea, dry mouth, anorexia, urinary retention, weakness, flushing, pruritus, headache, rash, CNS depression, hypotension, bradycardia, respiratory depression, dependency, euphoria	
Alosetron	Begin 0.5 mg PO daily. May be increased to 0.5 mg PO twice daily if no response in 4 weeks. Maximum dose is 1 mg twice daily. Approved for female patients with IBS with refractory diarrhea and pain.	Constipation, severe ischemic colitis	• Discontinue if no improvement at 1 mg twice daily for 4 weeks.

CNS=central nervous system; IBS=irritable bowel syndrome; PO=per os.

of the urge to defecate. Clearance of the rectal vault can be attempted with enemas or suppositories. Tap water enemas are preferred for chronic usage because repeated application of sodium phosphate or glycerin (common components of over-the-counter enema and suppository preparations) may precipitate mucosal damage and result in rectal bleeding. Ideally, the designated time for routine clearance of the rectal vault should be within 30 minutes after a meal to maximize postprandial colonic reflexes. A variety of agents have been used to bulk the anal sphincter to provide a barrier, including silicone, carbon-coated beads, and dextranomer in hyaluronic acid. A Cochrane systematic review, published in 2010, was unable to draw definitive conclusions on the efficacy of injectable bulking agents due to the limited number of trials available.[50] However, a systematic review published in 2011 found that use of silicone

or ceramic microspheres of calcium hydroxylapatite was associated with greater success than other agents.[51]

Dextranomer in stabilized hyaluronic acid is the most recent agent to be studied and approved by the US Food and Drug Administration. It has been shown to reduce the number of FI episodes by more than 50% in over half of all patients studied.[52,53] Adverse events from injectable bulking agents include anorectal pain, minor bleeding, or, rarely, rectal abscesses.

Nonpharmacologic Options

Nonpharmacologic options include biofeedback, sacral nerve stimulation (SNS), and surgery.

Biofeedback is a form of operant conditioning in which information about a physiologic process, which might otherwise be unconscious, is presented to a subject with the aim of having the subject modify that process consciously. For patients with FI, the process often involves physiologic monitoring of the striated pelvic floor muscles to facilitate directed strengthening exercises. Another method combines strengthening exercises with sensory discrimination training, consisting of brisk EAS contraction in response to rectal balloon distention.

Biofeedback has been advocated as first-line therapy for patients whose symptoms are mild to moderate. Although there is insufficient evidence with which to select patients suitable for anorectal biofeedback training, most experts agree that the appropriate patient for referral should have physiologic evidence of anal dysfunction, be able to cooperate, be well motivated, and possess some degree of perception of rectal distention and the ability to contract the EAS. Severe FI, pudendal neuropathy, and underlying neurologic problems have been associated with a suboptimal prognosis.[54-56] A systematic review of randomized controlled trials found limited evidence supporting the clinical efficacy of biofeedback. However, the authors found few easily comparable well-designed studies available for review.[55] A more recent Cochrane review of 21 trials supported the use of biofeedback in patients who had tried and failed other methods of treatment.[57]

SNS is a promising yet challenging treatment option for FI.[58] Originally investigated as a method for paraplegic rehabilitation and return to ambulation, SNS was instead found to improve voiding.[59] Subsequently, SNS was noted to have a positive effect on FI. Despite lack of understanding the underlying physiologic mechanism with which SNS acts to improve symptoms, its use in FI was first reported in 1995.[60] Early reports showed success rates between 67% and 100%.[61] These initial results led to broad popularity and rapid deployment in Europe.

A considerable advantage of SNS for FI is the ability to test for treatment efficacy with percutaneous nerve evaluation (PNE) prior to permanent implantation of the SNS system. PNE involves the percutaneous placement of an electrode through a sacral foramina to allow temporary stimulation of a sacral nerve, usually S3, with the goal of identifying patients who will benefit from permanent SNS placement.[58]

Long-term outcomes regarding the success of SNS beyond the original short-term results are beginning to emerge, with success rates of 54–92%. Parameters of success are variably reported to include reduced FI episodes, full continence, a perceived improvement in ability to defer defecation, improvement in Cleveland Clinic FI scores, resting and squeeze anal pressures, and other criteria.[61-64]

Long-term adverse event reports are limited in patients receiving SNS to treat FI. However, postoperative complications have been reported in up to 30% of elderly patients undergoing SNS for urinary symptoms.[65] Complications include pain at the site of implant, pouch infection, sensation of electric shock, and, rarely, lead displacement or battery failure requiring reoperation. Failure rates of up to 25% and revision rates up to 50% have been reported in urologic disorders.[66] One recent study of patients receiving SNS for FI found that 24% had surgical revisions.[67]

Surgery is indicated for patients whose FI is a consequence of a severe anatomic defect. Sphincteroplasty is the most common surgery, using either the overlapping sphincter repair or "end-to-end" repair technique. Overlapping sphincter repair has gained favor and is generally the first line of surgical intervention. Wound disruption leading to delayed healing occurs frequently. Although as many as 60% of patients report benefit from the operation,[68] other studies suggest that long-term efficacy of overlapping sphincteroplasty is poor, with only 14% of patients remaining fully continent at 5 years.[69] Patients who were older at the time of sphincteroplasty had worse long-term outcomes.[70] In one systematic review, sphincteroplasty reduced FI, but no patients were completely continent after 10 years. Sustained improvements in health-related quality of life were reported; however, additional surgery for incontinence was performed in up to 18% of patients.[71]

Graciloplasty and gluteus maximus transposition are options that provide patients with a new biologic sphincter. These procedures are preferred in patients with severe sphincter damage in which sphincteroplasty would not suffice. In graciloplasty, the gracilis muscle is mobilized, and the distal tendon is divided. The muscle is wrapped around the anal canal.[72] In dynamic graciloplasty, electrodes are attached to the muscle and connected to a neurostimulator that is implanted in the lower abdominal wall.[73] Dynamic graciloplasty is done to improve the functionality of the transposed gracilis muscle. The overall success rate ranges from 52–62%.[73-75] However, the complication rate of dynamic graciloplasty is high, with infection occurring in 25–28% of cases.[76] Other complications

include fecal evacuation problems, leg pain, bowel injury, perineal pain, and neoanal strictures.

Artificial anal sphincters may be an option in some patients. The artificial sphincter is placed around the native sphincter via perianal tunnels. The artificial sphincter remains inflated until the patient wishes to defecate, at which time the device is deactivated.[68] The overall success was about 47–53% in the patients who were able to retain their device.[77,78] Most patients required operative revisions, and 33% required device explantation.[76] Other complications included infection, device erosion or malfunction, chronic pain, and obstructed defecation. Artificial anal sphincters should only be considered in patients with severe incontinence whose other options are limited.

A 2010 Cochrane review analyzed 11 trials comparing different surgical interventions. Given the limited number of trials and small sample sizes, it was not possible for the authors to recommend one type of surgical technique over another.[79] Trials with larger sample sizes are needed for more definite recommendations. Colostomy or permanent stoma for FI is considered a reasonable option for patients who have failed or had poor response to multiple alternative treatments.

Conclusion

FI is a disabling disorder resulting in reduced physical, emotional, and social quality of life. A detailed evacuation history and anorectal examination are key to devising a focused diagnostic and effective, tiered treatment strategy for these patients. The majority of patients with FI can achieve marked improvements in quality of life with the simple measures that have been outlined in this review. Surgical interventions should be reserved for the rare patient, such as the patient with a clear anatomic defect, who cannot be managed with more conservative approaches.

The authors have no conflicts of interest to disclose.

References

1. Rao S. Advances in diagnostic assessment of fecal incontinence and dyssynergic defecation. *Clin Gastroenterol Hepatol.* 2010;8:910-919.e2.
2. Halland M, Talley NJ. Fecal incontinence: mechanisms and management. *Curr Opin Gastroenterol.* 2012;28:57-62.
3. Varma MG, Brown JS, Creasman JM, et al. Fecal incontinence in females older than aged 40 years: who is at risk? *Dis Colon Rectum.* 2006;49:841-851.
4. Whitehead WE, Borrud L, Goode PS, et al. Fecal incontinence in US adults: epidemiology and risk factors. *Gastroenterology.* 2009;137:512-517.e2.
5. Dunivan GC, Heymen S, Palsson OS, et al. Fecal incontinence in primary care: prevalence, diagnosis, and health care utilization. *Am J Obstet Gynecol.* 2010;202:493.e1-493.e6.
6. Nelson R, Norton N, Cautley E, Furner S. Community-based prevalence of anal incontinence. *JAMA.* 1995;274:559-561.
7. Rao SS. Pathophysiology of adult fecal incontinence. *Gastroenterology.* 2004;126(suppl 1):S14-S22.
8. Hawes SK, Ahmad A. Fecal incontinence: a woman's view. *Am J Gastroenterol.* 2006;101(12 suppl):S610-S617.
9. Bharucha AE, Fletcher JG, Harper CM, et al. Relationship between symptoms and disordered continence mechanisms in women with idiopathic faecal incontinence. *Gut.* 2005;54:546-555.
10. Andrews C, Bharucha AE, Seide B, Zinsmeister AR. Rectal sensorimotor dysfunction in women with fecal incontinence. *Am J Physiol Gastrointest Liver Physiol.* 2007;292:G282-G289.
11. Landefeld CS, Bowers BJ, Feld AD, et al. National Institutes of Health state-of-the-science conference statement: prevention of fecal and urinary incontinence in adults. *Ann Intern Med.* 2008;148:449-458.
12. Rao SS. Diagnosis and management of fecal incontinence. American College of Gastroenterology Practice Parameters Committee. *Am J Gastroenterol.* 2004;99:1585-1604.
13. Lam TJ, Kuik DJ, Felt-Bersma RJ. Anorectal function evaluation and predictive factors for faecal incontinence in 600 patients. *Colorectal Dis*. 2012;14:214-223.
14. Bharucha AE, Zinsmeister AR, Schleck CD, Melton LJ 3rd. Bowel disturbances are the most important risk factors for late onset fecal incontinence: a population-based case-control study in women. *Gastroenterology.* 2010;139:1559-1566.
15. Forsgren C, Zetterström J, Lopez A, Nordenstam J, Anzen B, Altman D. Effects of hysterectomy on bowel function: a three-year, prospective cohort study. *Dis Colon Rectum.* 2007;50:1139-1145.
16. Roberson EN, Gould JC, Wald A. Urinary and fecal incontinence after bariatric surgery. *Dig Dis Sci.* 2010;55:2606-2613.
17. Parés D, Vial M, Bohle B, et al. Prevalence of faecal incontinence and analysis of its impact on quality of life and mental health. *Colorectal Dis.* 2011;13:899-905.
18. Bharucha AE, Zinsmeister AR, Locke GR, et al. Risk factors for fecal incontinence: a population-based study in women. *Am J Gastroenterol.* 2006;101:1305-1312.
19. Boyle DJ, Knowles CH, Murphy J, et al. The effects of age and childbirth on anal sphincter function and morphology in 999 symptomatic female patients with colorectal dysfunction. *Dis Colon Rectum.* 2012;55:286-293.
20. Bharucha AE, Fletcher JG, Melton LJ 3rd, Zinsmeister AR. Obstetric trauma, pelvic floor injury and fecal incontinence: a population-based case-control study. *Am J Gastroenterol.* 2012;107:902-911.
21. Handa VL, Blomquist JL, Knoepp LR, Hoskey KA, McDermott KC, Munoz A. Pelvic floor disorders 5-10 years after vaginal or cesarean childbirth. *Obstet Gynecol.* 2011;118:777-784.
22. Nelson RL, Westercamp M, Furner SE. A systematic review of the efficacy of cesarean section in the preservation of anal continence. *Dis Colon Rectum.* 2006;49:1587-1595.
23. Nelson RL, Furner SE, Westercamp M, Farquhar C. Cesarean delivery for the prevention of anal incontinence. *Cochrane Database Syst Rev.* 2010:CD006756.
24. Nygaard I. Vaginal birth: a relic of the past in bulldogs and women? *Obstet Gynecol.* 2011;118:774-776.
25. Parés D, Vallverdú H, Monroy G, et al. Bowel habits and fecal incontinence in patients with obesity undergoing evaluation for weight loss: the importance of stool consistency. *Dis Colon Rectum.* 2012;55:599-604.
26. Melville JL, Fan MY, Newton K, Fenner D. Fecal incontinence in US women: a population-based study. *Am J Obstet Gynecol.* 2005;193:2071-2076.
27. Altman D, Falconer C, Rossner S, Melin I. The risk of anal incontinence in obese women. *Int Urogynecol J Pelvic Floor Dysfunct.* 2007;18:1283-1289.
28. Drossman DA, Sandler RS, Broom CM, McKee DC. Urgency and fecal soiling in people with bowel dysfunction. *Dig Dis Sci*. 1986;31:1221-1225.
29. Ahmad M, McCallum IJ, Mercer-Jones M. Management of faecal incontinence in adults. *BMJ.* 2010;340:c2964.
30. Stoker J. Magnetic resonance imaging in fecal incontinence. *Semin Ultrasound CT MR.* 2008;29:409-413.
31. Bharucha AE. Update of tests of colon and rectal structure and function. *J Clin Gastroenterol.* 2006;40:96-103.
32. Cazemier M, Terra MP, Stoker J, et al. Atrophy and defects detection of the external anal sphincter: comparison between three-dimensional anal endosonography and endoanal magnetic resonance imaging. *Dis Colon Rectum.* 2006;49:20-27.
33. Rogers RG, Abed H, Fenner DE. Current diagnosis and treatment algorithms for anal incontinence. *BJU Int.* 2006;98(suppl 1):97-106; discussion 107-109.
34. Fletcher JG, Busse RF, Riederer SJ, et al. Magnetic resonance imaging of anatomic and dynamic defects of the pelvic floor in defecatory disorders. *Am J Gastroenterol.* 2003;98:399-411.
35. Azpiroz F, Enck P, Whitehead WE. Anorectal functional testing: review of collective experience. *Am J Gastroenterol.* 2002;97:232-240.
36. Chen AS, Luchtefeld MA, Senagore AJ, Mackeigan JM, Hoyt C. Pudendal nerve latency. Does it predict outcome of anal sphincter repair? *Dis Colon Rectum.* 1998;41:1005-1009.
37. Diamant NE, Kamm MA, Wald A, Whitehead WE. AGA technical review on anorectal testing techniques. *Gastroenterology.* 1999;16:735-760.

38. Bliss DZ, Lewis J, Hasselman K, Savik K, Lowry A, Whitebird R. Use and evaluation of disposable absorbent products for managing fecal incontinence by community-living people. *J Wound Ostomy Continence Nurs.* 2011;38:289-297.
39. Bliss DZ, Savik K. Use of an absorbent dressing specifically for fecal incontinence. *J Wound Ostomy Continence Nurs.* 2008;35:221-228.
40. Brazzelli M, Shirran E, Vale L. Absorbent products for containing urinary and/or fecal incontinence in adults. *J Wound Ostomy Continence Nurs.* 2002;29:45-54.
41. Rohwer K, Bliss DZ, Savik K. Incontinence-associated dermatitis in community-dwelling individuals with fecal incontinence. *J Wound Ostomy Continence Nurs.* 2013;40:181-184.
42. Chew MH, Quah HM, Ooi BS, et al. A prospective study assessing anal plug for containment of faecal soilage and incontinence. *Colorectal Dis.* 2008;10:677-680.
43. Norton C, Kamm MA. Anal plug for faecal incontinence. *Colorectal Dis.* 2001;3:323-327.
44. Deutekom M, Dobben A. Plugs for containing fecal incontinence. *Cochrane Database Syst Rev.* 2005:CD005086. Update in: *Cochrane Database Syst Rev.* 2012:CD005086.
45. Scarlett Y. Medical management of fecal incontinence. *Gastroenterology.* 2004;126(suppl 1):S55-S63.
46. Wald A. Irritable bowel syndrome—diarrhoea. *Best Pract Res Clin Gastroenterol.* 2012;26:573-580.
47. McRorie JW, Daggy BP, Morel JG, Diersing PS, Miner PB, Robinson M. Psyllium is superior to docusate sodium for treatment of chronic constipation. *Aliment Pharmacol Ther.* 1998;12:491-497.
48. Gallegos-Orozco JF, Foxx-Orenstein AE, Sterler SM, Stoa JM. Chronic constipation in the elderly. *Am J Gastroenterol.* 2012;107:18-25; quiz 26.
49. Rao SS, Ozturk R, Stessman M. Investigation of the pathophysiology of fecal seepage. *Am J Gastroenterol.* 2004;99:2204-2209.
50. Maeda Y, Laurberg S, Norton C. Perianal injectable bulking agents as treatment for faecal incontinence in adults. *Cochrane Database Syst Rev.* 2010:CD007959.
51. Hussain ZI, Lim M, Stojkovic SG. Systematic review of perianal implants in the treatment of faecal incontinence. *Br J Surg.* 2011;98:1526-1536.
52. Danielson J, Karlbom U, Sonesson AC, Wester T, Graf W. Submucosal injection of stabilized nonanimal hyaluronic acid with dextranomer: a new treatment option for fecal incontinence. *Dis Colon Rectum*. 2009;52:1101-1106.
53. Graf W, Mellgren A, Matzel KE, Hull T, Johansson C, Bernstein M; NASHA Dx Study Group. Efficacy of dextranomer in stabilised hyaluronic acid for treatment of faecal incontinence: a randomised, sham-controlled trial. *Lancet.* 2011; 377:997-1003.
54. Prather CM. Physiologic variables that predict the outcome of treatment for fecal incontinence. *Gastroenterology*. 2004;126(1 suppl 1):S135-S140.
55. Fernández-Fraga X, Azpiroz F, Aparici A, Casaus M, Malagelada JR. Predictors of response to biofeedback treatment in anal incontinence. *Dis Colon Rectum*. 2003;46:1218-1225.
56. Norton C, Hosker G, Brazzelli M. Biofeedback and/or sphincter exercises for the treatment of faecal incontinence in adults. *Cochrane Database Syst Rev.* 2000:CD002111.
57. Norton C, Cody JD. Biofeedback and/or sphincter exercises for the treatment of faecal incontinence in adults. *Cochrane Database Syst Rev*. 2012:CD002111.
58. Maeda Y, O'Connell PR, Matzel KE, Laurberg S. Sacral nerve stimulation for fecal incontinence: at a crossroad and future challenges. *Dis Colon Rectum.* 2012;55:621-624.
59. Brindley GS. History of the sacral anterior root stimulator, 1969-1982. *Neurourol Urodyn.* 1993;12:481-483.
60. Matzel KE, Stadelmaier U, Hohenfellner M, Gall FP. Electrical stimulation of sacral spinal nerves for treatment of faecal incontinence. *Lancet.* 1995;346:1124-1127.
61. Boyle DJ, Murphy J, Gooneratne ML, et al. Efficacy of sacral nerve stimulation for the treatment of fecal incontinence. *Dis Colon Rectum.* 2011;54:1271-1278.
62. Altomare DF, Ratto C, Ganio E, Lolli P, Masin A, Villani RD. Long-term outcome of sacral nerve stimulation for fecal incontinence. *Dis Colon Rectum.* 2009;52:11-17.
63. Matzel KE, Lux P, Heuer S, Besendörfer M, Zhang W. Sacral nerve stimulation for faecal incontinence: long-term outcome. *Colorectal Dis.* 2009;11:636-641.
64. George AT, Kalmar K, Panarese A, Dudding TC, Nicholls RJ, Vaizey CJ. Long-term outcomes of sacral nerve stimulation for fecal incontinence. *Dis Colon Rectum.* 2012;55:302-306.
65. Griebling TL. Sacral nerve stimulation in the elderly. *Int Urogynecol J.* 2010;21(suppl 2):S485-S489.
66. Elneil S. Urinary retention in women and sacral neuromodulation. *Int Urogynecol J.* 2010;21(suppl 2):S475-S483.
67. Faucheron JL, Voirin D, Badic B. Sacral nerve stimulation for fecal incontinence: causes of surgical revision from a series of 87 consecutive patients operated on in a single institution. *Dis Colon Rectum.* 2010;53:1501-1507.
68. Madoff RD. Surgical treatment options for fecal incontinence. *Gastroenterology.* 2004;126(suppl 1):S48-S54.
69. Halverson AL, Hull TL. Long-term outcome of overlapping anal sphincter repair. *Dis Colon Rectum.* 2002;45:345-348.
70. Zutshi M, Tracey TH, Bast J, Halverson A, Na J. Ten-year outcome after anal sphincter repair for fecal incontinence. *Dis Colon Rectum.* 2009;52:1089-1094.
71. Glasgow SC, Lowry AC. Long-term outcomes of anal sphincter repair for fecal incontinence: a systematic review. *Dis Colon Rectum.* 2012;55:482-490.
72. Müller C, Belyaev O, Deska T, Chromik A, Weyhe D, Uhl W. Fecal incontinence: an up-to-date critical overview of surgical treatment options. *Langenbecks Arch Surg.* 2005;390:544-552.
73. Madoff RD, Rosen HR, Baeten CG, et al. Safety and efficacy of dynamic muscle plasty for anal incontinence: lessons from a prospective, multicenter trial. *Gastroenterology.* 1999;116:549-556.
74. Baeten CG, Bailey HR, Bakka A, et al. Safety and efficacy of dynamic graciloplasty for fecal incontinence: report of a prospective, multicenter trial. Dynamic Graciloplasty Therapy Study Group. *Dis Colon Rectum.* 2000;43:743-751.
75. Geerdes BP, Heineman E, Konsten J, Soeters PB, Baeten CG. Dynamic graciloplasty. Complications and management. *Dis Colon Rectum.* 1996;39:912-917.
76. Tan JJ, Chan M, Tjandra JJ. Evolving therapy for fecal incontinence. *Dis Colon Rectum.* 2007;50:1950-1967.
77. Christiansen J, Rasmussen OO, Lindorff-Larsen K. Long-term results of artificial anal sphincter implantation for severe anal incontinence. *Ann Surg.* 1999;230:45-48.
78. Wong WD, Congliosi SM, Spencer MP, et al. The safety and efficacy of the artificial bowel sphincter for fecal incontinence: results from a multicenter cohort study. *Dis Colon Rectum.* 2002;45:1139-1153.
79. Brown SR, Wadhawan H, Nelson RL. Surgery for faecal incontinence in adults. *Cochrane Database Syst Rev.* 2010:CD001757.