CME

ACG Clinical Guideline: Management of Benign Anorectal Disorders

Arnold Wald, MD, MACG[1], Adil E. Bharucha, MBBS, MD[2], Bard C. Cosman, MD, MPH, FASCRS[3] and William E. Whitehead, PhD, MACG[4]

These guidelines summarize the definitions, diagnostic criteria, differential diagnoses, and treatments of a group of benign disorders of anorectal function and/or structure. Disorders of function include defecation disorders, fecal incontinence, and proctalgia syndromes, whereas disorders of structure include anal fissure and hemorrhoids. Each section reviews the definitions, epidemiology and/or pathophysiology, diagnostic assessment, and treatment recommendations of each entity. These recommendations reflect a comprehensive search of all relevant topics of pertinent English language articles in PubMed, Ovid Medline, and the National Library of Medicine from 1966 to 2013 using appropriate terms for each subject. Recommendations for anal fissure and hemorrhoids lean heavily on adaptation from the American Society of Colon and Rectal Surgeons Practice Parameters from the most recent published guidelines in 2010 and 2011 and supplemented with subsequent publications through 2013. We used systematic reviews and meta-analyses when available, and this was supplemented by review of published clinical trials.

Am J Gastroenterol 2014; 109:1141–1157; doi:10.1038/ajg.2014.190; published online 15 July 2014

Similar to recent guidelines (1,2), we used the GRADE (Grades of Recommendation Assessment, Development and Evaluation) system to assess the strengths of the recommendations and the overall quality of the evidence to support those recommendations. A strong recommendation was given if the committee felt that most individuals should receive the treatment and the recommendation would apply to most clinical situations, whereas a weak recommendation implies that clinicians should examine the available evidence themselves and future policy making will require debates and involvement of many stakeholders (3). Quality of evidence was considered high when available studies strongly suggest that further research is unlikely to alter our confidence about efficacy, moderate quality suggests that further research is likely to affect future recommendations, and low quality suggests that further research is very likely to affect future assessments and recommendations.

DEFECATORY DISORDERS

Methods used to review diagnostic tests

A systematic review of diagnostic tests for constipation was recently reported as part of a comprehensive guideline concerning the management of constipation (2). Our review focuses on studies that examined the concordance of the most commonly used diagnostic tests to each other or to an external standard where one is available. The diagnostic tests assessed are symptoms, digital rectal examination, anorectal manometry (ARM) with or without electromyography of the pelvic floor (EMG), the balloon expulsion test (BET), barium defecography, and magnetic resonance imaging (MRI) of the pelvic floor. **Figure 1** illustrates a suggested algorithm for managing defecatory disorders (DDs). The National Library of Medicine was searched for these terms that were cross-referenced to the terms that have been used to describe dyssynergic defecation: disordered defecation, pelvic floor dyssynergia, anismus, obstructed defecation, and functional outlet obstruction.

Definition and pathophysiology

A DD refers to difficulty in evacuating stool from the rectum in a patient with chronic or recurring symptoms of constipation (4,5). DD may be caused by functional or structural anorectal disturbances that may coexist. The functional disturbances include dyssynergia, defined as paradoxical contraction or failure to relax pelvic floor muscles during simulated defecation, typically defined as $<20\%$ decrease in anal canal pressures (5), and/or inadequate defecatory propulsion, defined as inadequate increase in rectal or intraabdominal pressure during simulated

[1]Division of Gastroenterology and Hepatology, University of Wisconsin School of Medicine and Public Health, Madison, Wisconsin, USA; [2]Division of Gastroenterology and Hepatology, Mayo Clinic, Rochester, MN, USA; [3]University of California San Diego School of Medicine, Halasz General Surgery Section, VA San Diego Healthcare System, San Diego, California, USA; [4]University of North Carolina Center for Functional GI and Motility Disorders, Chapel Hill, North Carolina, USA. **Correspondence:** Arnold Wald, MD, MACG, Division of Gastroenterology and Hepatology, University of Wisconsin School of Medicine and Public Health, 1685 Highland Avenue, Suite 4000, Madison, Wisconsin 53705, USA. E-mail: axw@medicine.wisc.edu
Received 3 September 2013; accepted 5 June 2014

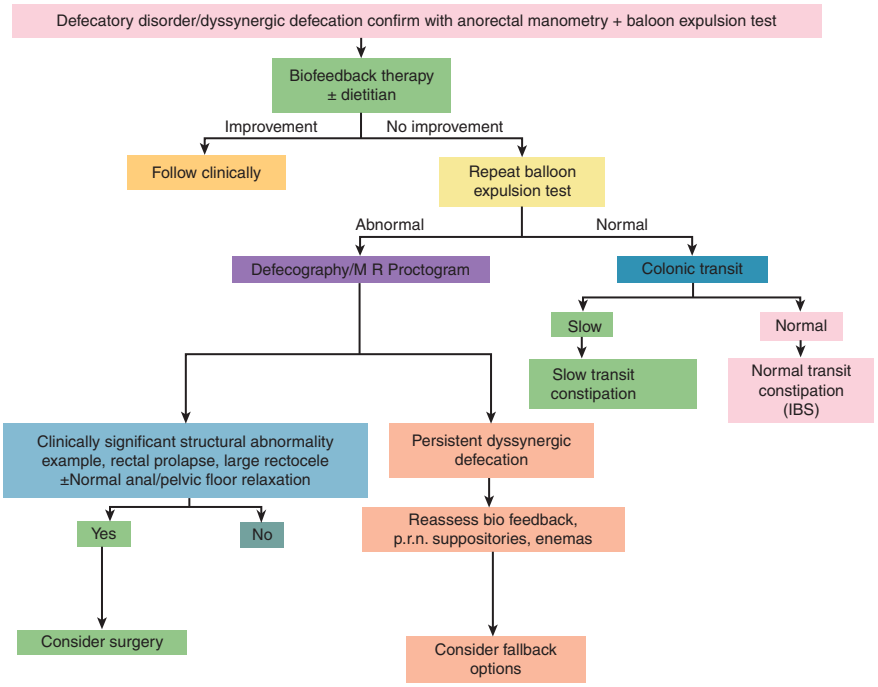

Figure 1. Suggested algorithm for the evaluation and management of defecatory disorders (DDs). IBS, irritable bowel syndrome; MR, magnetic resonance. Reproduced with permission from Bharucha and Rao (161).

defecation (5). Recent studies using high-resolution manometry suggest that there are three groups that involve one or a combination of these manometric patterns (6). Conventional ARM studies support the view that these two physiological mechanisms are predictive of the inability to evacuate stool from the rectum, are reproducible (7), and are able to discriminate healthy controls from patients with difficult defecation (8). Moreover, biofeedback training protocols designed to correct dyssynergia and inadequate rectal propulsion have been shown in three randomized controlled trials (RCTs) (9–11) to significantly improve the ability to evacuate the rectum. It is uncertain whether all manometric subgroups respond equally well to biofeedback training.

When ARM shows normal relaxation of pelvic floor muscles during simulated defecation, this argues against DD. However, in interpreting a test showing paradoxical contraction or failure to relax the pelvic floor muscles, it is important to recognize that this finding is present in a significant proportion of normal subjects. A recent study that used a high-resolution ARM catheter found that failure to decrease anal canal pressures by 20% during simulated defecation (i.e., dyssynergia) was present in 37% of healthy control women and in 54% of women with chronic constipation who were able to evacuate a 50-ml balloon (12). In another report from the same center, the rectoanal pressure gradient was negative or abnormal in 84% of healthy women (13). An older study using conventional ARM reported that only 16% of healthy subjects displayed dyssynergia (14). Moreover, dyssynergia by conventional ARM did not predict abnormal balloon expulsion in healthy subjects (15). These discordant findings show that high-resolution ARM alone cannot be used to make a diagnosis of this undoubtedly heterogeneous disorder. However, this must be balanced against the fact that multiple studies (see treatment section) show that patients selected on the basis of manometric evidence of dyssynergia and inability to evacuate a balloon have a high likelihood of improving their symptoms of disordered defecation when they are taught with biofeedback to relax their pelvic floor muscles during simulated defecation.

DD is not a neurological disorder, and no other structural basis has been identified. Rather, DD is believed to be frequently due to maladaptive learning based on two observations: most patients with DD learn to relax the pelvic floor and/or increase rectal pressure appropriately when provided with biofeedback training (see below); this suggests that DD is not due to an anatomical defect, and it is often associated with a history of painful defecation in children (16) or with a history of sexual abuse or other pelvic floor trauma in adults (17). These behavioral contributions to the etiology of disordered defecation may explain some of the inconsistencies noted above.

Recommendations for diagnostic assessment

1. DDs are defined as difficulty in evacuating stool from the rectum in a patient with chronic or recurring symptoms of constipation (strong recommendation, moderate quality of evidence).

2. Gastroenterologists and other providers should not make the diagnosis of DD on the basis of a single abnormal test because none is sufficiently specific. However, confidence in the diagnosis is increased if there is a combination of a clinical history of chronic constipation and two abnormal tests, i.e., impaired ability to evacuate a 50-ml water-filled balloon or abnormal defecography and evidence from pelvic floor EMG or ARM that the patient is unable to relax pelvic floor muscles or increase rectal pressure during simulated defecation (strong recommendation, moderate quality of evidence).
3. Digital rectal examination is a useful first test to screen for DD, as it has good negative predictive value (weak recommendation, low quality of evidence).
4. Barium or MR defecography can identify structural causes of outlet obstruction if one is expected. They may also confirm or exclude the diagnosis of DD when the clinical features suggest DD but the results of ARM and BET are equivocal (moderate recommendation, moderate quality of evidence).

It would seem intuitive that symptoms such as a sensation of incomplete evacuation, sensation of blocked evacuation, and the need to exert pressure in the vagina or around the anus to facilitate defecation should be specific to DD. However, published studies show that the positive and negative predictive value of symptoms alone for the diagnosis of DD is inadequate (18–20).

A *digital rectal examination* begins with perianal inspection. A gloved finger is inserted in the anal canal to assess anal tone at rest and when the patient is asked to squeeze or contract the sphincter. The patient is then asked to strain as if to defecate; the normal response is a decrease in anal canal pressure around the examiner's finger, whereas a contraction around the finger suggests DD. The examining finger is then inserted more deeply to palpate the puborectalis muscle; the patient is again asked to strain as if to defecate and the normal response is for the muscle to relax, thus widening the anorectal angle. One published abstract (21) assessed the accuracy of digital examination for the diagnosis of DD. The positive predictive value for digital rectal examination was 61% and the negative predictive value was 91%. A second study (22) compared a composite physical examination that included digital rectal examination to ARM and reported a PPV of 97% and a negative predictive value of 37%, but this study cannot be used to assess the predictive value of digital rectal examination alone. Thus, there are insufficient data to be confident of the utility of digital rectal examination for the diagnosis of DD.

ARM involves the following: (i) measuring anal canal pressures to assess contraction vs. relaxation of the pelvic floor muscles and (ii) measuring rectal pressure to determine whether there is adequate rectal propulsive force during simulated defecation. These two mechanisms may be assessed separately or may be integrated into a defecation index (anorectal gradient) in one of two ways: by subtracting anal canal pressure from rectal pressure during simulated defecation (a positive difference is normal) (13) or by calculating the ratio of rectal pressure to anal canal pressure during simulated defecation (a value of > 1.0 is normal) (14). The utility of the rectoanal gradient is unclear because (i) there is considerable overlap between asymptomatic subjects and patients with DDs (12,13) and (ii) the correlation between the rectoanal gradient and BET is relatively weak (12,13). However, when pelvic floor relaxation is measured by ARM independently of the anorectal gradient, the concordance between dyssynergia and a failed BET ranges from 72 to 95% (20,23,24); see **Table 1**. Further studies are necessary to ascertain the utility of the rectoanal gradient for identifying DD.

Some clinical conditions result in discrepancies between dyssynergia and the symptoms of DD: a subset of patients presenting with fecal incontinence (FI) have paradoxical contractions of their pelvic floor muscles during evacuation but normal BET; these individuals may have learned to cope with the threat of FI by contracting pelvic floor muscles when there is any sensation of increased pressure in the rectum. Conversely, patients with structural causes for obstructed defecation such as rectal prolapse may be unable to evacuate a balloon even though their pelvic floor muscles relax appropriately during simulated defecation (25).

EMG activity is recorded either from stainless steel plates mounted on an acrylic anal plug or from electrodes taped to the skin on opposite sides of the anus (26), and it is frequently used in biofeedback training for DD (9,11). It can also be used to identify patients with dyssynergia (27) and appears to show excellent agreement with manometry (9). One study showed that averaged EMG activity was a better predictor of failure to evacuate a balloon than was ARM (28).

BET is a test of simulated evacuation in which a balloon-tipped catheter is lubricated and inserted into the rectum. It is then filled with water or air (typically with 50 ml), but sometimes with a volume required to produce a sustained sensation of urgency to defecate. The time required for the patient to evacuate the balloon in privacy is measured. A variation of the BET is to inject FECOM—a rubber compound that has the consistency of soft stool—into the rectum as a substitute for the water- or air-filled balloon (29). However, the methods of conducting the test and the upper limit of normal evacuation time vary slightly across studies. BET has a high specificity for dyssynergia, defined by paradoxical contraction and/or failure to relax the pelvic floor (**Table 1**). A balloon expulsion time of > 2 min is definitely abnormal (28).

Barium defecography is performed by injecting barium contrast mixed with Metamucil or another thickening agent into the rectum and taking lateral images of the anorectum during pelvic floor contraction, and before, during, and after attempted defecation (30). The angle between the axis of the rectum and of the anal canal provides an indirect measure of whether the puborectalis muscle relaxes (normal response) or contracts (indicative of DD) during simulated defecation. Additional information is obtained on structural causes of outlet dysfunction including rectal prolapse, rectocele, and enterocele. Defecography, once regarded as the gold standard for diagnosis of DD, has been largely replaced by the BET and ARM because (i) it is simpler to perform BET and ARM

Table 1. Sensitivity and specificity of balloon evacuation test for dyssynergia as defined by ARM

Study	DD definition	Sensitivity (%)	Specificity (%)
Raza and Bielefeldt (23)	ARM	68	91
Minguez *et al.* (20)	ARM + defecography	88	89
Chiarioni *et al.* (36)	ARM	94	75
Chiarioni *et al.* (25)	ARM	60	100

ARM, anorectal manometry; DD, defecatory disorder.

than defecography; (ii) defecography is often not interpreted by established criteria that limits the diagnostic utility of this test; and (iii) defecography involves radiation, whereas BET and ARM do not.

However, in contrast to BET and ARM, defecography characterizes structural causes of outlet dysfunction. Consequently, defecography is often used when ARM and BET are equivocal, do not concur with the clinical impression, or for patients who are unable to evacuate a balloon but who relax the pelvic floor normally during simulated defecation. These recommendations are supported by several studies that demonstrate that defecography can identify impaired evacuation in patients with symptoms of DD but with normal BET and anal EMG testing (28,31).

MRI is an alternative to barium defecography (32). The test is performed by imaging the pelvic floor, whereas patients perform the maneuvers as described for defecography. Advantages of MRI over defecography include the following: (i) better resolution of soft tissue surrounding the rectum and anal canal, including the bladder, uterus, and small intestine during dynamic imaging; (ii) improved ability to visualize anal sphincter and levator ani muscles with endoanal MRI; and (iii) lack of radiation. MRI is particularly useful in patients with normal balloon expulsion to identify structural lesions and to guide surgical therapy, e.g., for rectoceles in patients with vaginal splinting and cystoceles or uterine prolapse in patients undergoing surgery (33). The utility of MRI was demonstrated in a study of 52 patients with DD and 41 control subjects in which MRI disclosed features of DD in 94% of all patients (31). MRI identified DD in some patients with normal BET, but as ARM was not performed it is unknown whether manometry would have correlated positively with MRI. Barium defecography is performed in the seated position, whereas MR defecography is performed in the supine position. Nonetheless, the correlation between dynamic MRI and colpocystoproctography for quantifying prolapse in all three pelvic floor compartments (anterior, middle, and posterior) is excellent (34).

Whole-gut transit, which can be evaluated using radiopaque markers or scintigraphy, is no longer advocated as a diagnostic criterion for DD. One study showed that patients with DD were significantly more likely than patients with slow transit constipation to have delays in transit in the left side of the colon vs. the right side, but discrimination was modest (35). Moreover, many patients with DD have slow colonic transit that may normalize after successful treatment of DD (see below). ARM and BET provide better discrimination of DD from normal. Colonic transit is best used in constipated patients who exhibit normal ARM and BET or in patients who fail to improve despite correction of DD using biofeedback.

Combined tests. None of the currently available tests has sufficient positive predictive value when used alone; all yield false positive or false negative results. It is therefore recommended that the diagnosis of DD be based on a combination of tests of anorectal function. We recommend basing the diagnosis on a combination of three criteria: a clinical history of chronic or recurrent symptoms of constipation, an ARM showing dyssynergia, and a BET showing the inability to evacuate a 50-ml balloon (34). One study showed that ARM and BET are significant independent predictors of the clinical response to biofeedback (34). The requirement that patients have a history of chronic constipation addresses the observation that dyssynergia and delayed balloon evacuation may each occur in patients without symptoms of constipation; these patients would not be referred for treatment of constipation.

Differential diagnosis

Several entities should be distinguished from DD because different treatments are appropriate for these entities.

Structural causes of outlet dysfunction such as rectal prolapse and rectocele should be distinguished from DD because, when they are not accompanied by dyssynergia or inadequate rectal propulsion, they are unlikely to respond to biofeedback training. Some patients with DD exhibit excessive perineal descent, with or without prolapse of other pelvic organs. In such patients, it can be challenging to determine the contributions of structural and functional disturbances to DD. Additional testing for obstructed defecation may involve physical examination for rectal prolapse, rectocele, and perineocele, or imaging studies such as barium or MR defecography.

Slow transit constipation is characterized by delayed transit through the colon and is believed to be due to abnormal colon motility. It is diagnosed by a prolonged colonic or whole-gut transit test. There are many patients who show both slow transit and evidence of DD. However, in up to 2/3 of these patients, slow transit is secondary to outlet dysfunction rather than an independent, comorbid condition (36). The evidence to support this interpretation is as follows: when patients with documented delays in colonic transit were stratified into those with or without evidence of DD, and all were treated with biofeedback to correct DD, 2/3 of the patients with DD normalized their transit, whereas patients with no DD showed no improvement in transit time following biofeedback training.

Recommendations for the treatment of disordered defecation

5. Biofeedback is the preferred treatment for DD in adults (strong recommendation, moderate quality of evidence).

The treatment protocols used in most RCTs include the following steps (24):

(1) Patient education—explain to patients that they unconsciously squeeze their anus when they are trying to defecate and this holds the stool in the rectum.
(2) Simulated defecation training—for patients who do not increase intraabdominal pressure during simulated defecation, the use of feedback on rectal balloon pressure teaches them to tighten their abdominal wall muscles and lower their diaphragm to push stool out.
(3) Training to relax pelvic floor muscles while simulating defecation—for patients who paradoxically contract their pelvic floor muscles during simulated defecation, provide visual feedback on anal canal pressure or averaged EMG activity from the anal canal to teach this skill.
(4) Practicing simulated defecation—patients practice defecation of a lubricated, inflated balloon while the therapist gently pulls on the catheter to assist them. Remind the patient to relax the pelvic floor muscles, increase abdominal pressure using abdominal wall muscles, and concentrate on the sensations produced by balloon passage.

Some centers also include sensory training in an effort to lower the threshold for the sensation of urgency to defecate (10). This is done by identifying the urge threshold and then presenting a series of rectal balloon distensions, some of which are slightly higher than this threshold and some of which are slightly lower. Patients usually learn to improve their sensitivity for the sensation of urgency.

Table 2 summarizes nine published RCTs of biofeedback in adults. The three largest studies used the treatment protocol described above, and the average number of treatment sessions was 5 in all three studies. These studies showed biofeedback to be significantly more effective than a laxative (9), diazepam and placebo tablets (11), and sham biofeedback or medical management (10). Three other studies showed biofeedback to be superior to control conditions consisting of patient education (37), medical management consisting of dietary advice from a nutritionist plus exercise and laxatives (38), and simple practice of balloon defecation (39). Although one study (40) did not show biofeedback to be superior to practice of balloon defecation, it is doubtful that all patients had DD, as 18 of 30 in the biofeedback group had normal BET before training. Other studies suggest that biofeedback is inferior to botulinum injection into the pelvic floor (41,42) or surgical division of the puborectalis (42). However, the outcomes of biofeedback training in these two studies were notably inferior to the others.

Chronic constipation with associated fecal impaction and overflow FI is frequently seen in children (43), and the mechanism for this appears to be dyssynergic defecation because these children squeeze their pelvic floor muscles when instructed to try to defecate. However, the majority of studies in children suggest that biofeedback is no better than laxatives (44–51). We speculate that the poorer outcomes may occur because biofeedback requires a high level of motivation and sustained attention that may be beyond the ability of many children (16). However, the explanation is unknown.

PROCTALGIA SYNDROMES

Definitions and pathophysiology

Chronic proctalgia is also known by other names including levator ani syndrome, levator spasm, puborectalis syndrome, pyriformis syndrome, and pelvic tension myalgia. It is defined by recurring episodes of rectal pain or aching, with each episode lasting 20 min or greater (52). All commonly used treatments for chronic proctalgia are directed at relaxing the striated muscles of the pelvic floor based on the assumption that chronic proctalgia is due to sustained contraction (spasm) of pelvic floor muscles. The observation that tenderness or pain during digital rectal examination when pressure is applied to the levator ani muscles is a strong predictor of response to biofeedback treatment (53) supports this hypothesis.

Recent evidence suggests that the pathophysiology of chronic proctalgia and dyssynergic defecation may overlap (51). An abnormal BET and manometric findings of dyssynergia were correlated with the presence of levator tenderness, and physiological improvements with biofeedback were associated with improvement in chronic proctalgia. Further research is needed to understand why these findings are associated with constipation in some patients and chronic proctalgia in others. Stress and anxiety are often thought to contribute to chronic proctalgia, but there is little evidence for this. Nevertheless, psychological counseling is often incorporated into the treatment (51).

Recommendations for diagnostic assessment

1. Gastroenterologists and other providers should make a diagnosis of chronic proctalgia based on a history of recurring episodes of rectal pain, each lasting at least 20 minutes, a digital rectal examination showing tenderness to palpation of the levator ani muscles, and exclusion of other causes for rectal pain by history and diagnostic testing (strong recommendation, moderate quality of evidence).
2. Gastroenterologists and other providers should obtain an imaging study or endoscopy to rule out structural causes of rectal pain (strong recommendation, low quality of evidence).
3. Gastroenterologists and other providers should obtain a BET and ARM to identify patients with chronic proctalgia and levator muscle tenderness who are likely to respond to biofeedback (strong recommendation, high quality of evidence).

Diagnosis is based on symptoms of chronic rectal pain with episodes lasting at least 20 min, physical examination findings of tenderness with palpation of the puborectalis, and a workup to exclude alternative explanations for the pain. No other

Table 2. Biofeedback treatment of DD in adults

Study	Inclusion criteria	Sample size	Comparator	Outcome
Koutsomanis *et al.* (40)	Adults with functional constipation	60	Balloon defecation training	No difference
Chiarioni *et al.* (9)	Adults with DD	109	PEG laxative	Biofeedback superior
Heymen *et al.* (11)	Adults with DD	84	Diazepam (10 mg), placebo pills	Biofeedback superior to diazepam and placebo
Rao *et al.* (10)	Adults with DD	77	Sham feedback, medical management	Biofeedback superior to both
Simon and Bueno (37)	Elderly with functional constipation	30	Education	Biofeedback superior
Faried *et al.* (41)	Adults with DD	48	Botulinum A	No difference
Faried *et al.* (42)	Adults with DD	60	Botulinum A or surgery (division of puborectalis)	Surgery superior
Rao *et al.* (38)	Adults with DD	26	Usual medical care	Biofeedback superior
Pourmomeny *et al.* (39)	Adults with DD	65	Balloon defecation training	Biofeedback superior

DD, defecatory disorder; PEG, polyethylene glycol.

diagnostic criteria have been validated (52). However, in one large treatment study (53), failure to evacuate a 50-ml water-filled balloon and the inability to relax pelvic floor muscles during simulated defecation correlated with the presence of tenderness to palpation and were predictive of the success of biofeedback treatment. These observations suggest that BET and ARM should be added to the diagnostic workup to improve selection of patients for biofeedback treatment. Independent confirmation of these findings is awaited.

There is frequent overlap of symptoms between chronic proctalgia and other conditions such as chronic prostatitis and chronic pelvic pain syndrome (53,54). In clinical practice, we recommend excluding structural causes of chronic pelvic pain with imaging studies and/or colonoscopy before proceeding with a trial of conservative treatment (see below).

Recommendations for treatment

4. Biofeedback to teach relaxation of pelvic floor muscles during simulated defecation is the preferred treatment. (strong recommendation, moderate quality of evidence).
5. Electrical stimulation is superior to digital massage but inferior to biofeedback (moderate recommendation, low quality of evidence).

Electrical stimulation of pelvic floor muscles, biofeedback to teach relaxation of those muscles, massage of the levator ani muscles, sitz baths, and botulinum toxin injections have all been advocated for the treatment of chronic proctalgia. However, only two RCTs have been reported. Chiarioni *et al.* (53) randomized 157 patients with chronic proctalgia to receive nine treatment sessions of pelvic floor biofeedback, electrical stimulation, or massage. Before randomization, all patients were stratified based on whether or not they reported tenderness during palpation of the levator ani muscles. Among patients who reported tenderness on palpation, the intent-to-treat analysis showed that 87% reported adequate relief of rectal pain following biofeedback, compared with 45% for electrical stimulation and 22% for digital massage; relief was well maintained for 12 months of follow-up. For patients with tenderness on digital examination, the number of pain days per month decreased from 14.7 at baseline to 3.3 after biofeedback, 8.9 after electrical stimulation, and 13.3 after massage, and results were also well maintained for 12 months. However, patients with no tenderness during digital rectal examination did not show improvements with any of these treatments. The investigators also showed that failure to relax pelvic floor muscles when simulating defecation and abnormal BET at baseline were predictors of which patients responded positively to biofeedback treatment, suggesting that the pathophysiology of chronic proctalgia has similarities to dyssynergic defecation. Confirmatory studies from other centers are needed.

A small randomized controlled crossover study compared the effects of 100 units of botulinum A toxin vs. placebo injections injected into the levator ani (54). Only 7 of 12 patients completed the study, and there was no significant benefit of botulinum toxin.

Proctalgia fugax, which is characterized by intense sensations of rectal or anal canal pain lasting only a few seconds to minutes (see below), should be distinguished from chronic proctalgia, in which painful episodes are more prolonged (51). In addition, many other diseases and disorders may be responsible for anorectal pain, including anal fissure, proctitis, solitary rectal ulcer, and coccygodynia. Gynecological conditions and urologic disorders (e.g., chronic prostatitis) may also be confused with chronic proctalgia.

The pathophysiology of proctalgia fugax is unknown, although thickening of the internal anal sphincter and elevated resting pressure in the anal canal have been reported; these findings suggest spasm of the internal anal sphincter. No trigger events have been consistently identified. A rare congenital form of the disorder has been described (55).

Recommendations for diagnostic assessment

6. Gastroenterologists and other providers should make a diagnosis of proctalgia fugax on the basis of a history of intermittent bouts of severe pain in the anal canal or lower rectum lasting less than 20 minutes (strong recommendation, low quality of evidence).
7. Gastroenterologists and other providers should exclude structural causes of anorectal pain (e.g., anal fissure, hemorrhoids, cryptitis, malignancy) by imaging, endoscopy, or other appropriate tests (strong recommendation, low quality of evidence).

Recommendations for treatment

8. Gastroenterologists and other providers should assure patients that the disorder is benign. The evidence for specific treatments is no better than anecdotal (moderate recommendation, low quality of evidence).

On the basis of their systematic review, Jeyarajah *et al.* (55) recommend the following progression of steps for the patient with frequent, debilitating episodes of proctalgia fugax: (i) reassurance and warm baths, (ii) topical glyceryl trinitrate 0.2% p.r.n., (iii) salbutamol inhalation 200 g p.r.n., (iv) warm water enemas, (v) clonidine 150 µg b.i.d., (vi) local anesthetic block, and (vii) botulinum toxin injection into the anal sphincters. For patients with demonstrated thickening of the internal anal sphincter and high anal resting pressures, these authors suggest that limited internal anal sphincterotomy may be considered. We do not endorse these recommendations because the evidence for these treatments is no better than anecdotal, and some treatments on this list may be associated with adverse effects. The typical patient with proctalgia fugax has infrequent and short episodes of pain for which neither treatment nor prevention is practical.

FECAL INCONTINENCE

Definition and epidemiology

FI is the involuntary loss of solid or liquid feces. The more general term, anal incontinence, also includes involuntary loss of flatus. Although incontinence of flatus can be embarrassing, a threshold to discriminate inadvertent expulsion of gas from incontinence is not available. The prevalence of FI in the community increases with age and, depending on survey methods and definition of FI, it varies from 2.2 to 25% (56). FI can affect daily life (57) and may predispose to institutionalization (58): up to 50% of nursing home residents in one survey had FI (59). Despite these potentially devastating consequences, only a small proportion of incontinent patients discuss the symptom with a physician (60–62). Hence, physicians should ask patients with predisposing risk factors for FI, particularly diarrhea and constipation, whether they have FI.

Etiology

Conditions that cause bowel disturbances and/or anorectal weakness can predispose to FI (**Table 3**) (63). In community-based epidemiological studies, advancing age, increased body mass index, diarrhea, rectal urgency, cholecystectomy, anal fistula, non-childbirth anal injury, urinary incontinence, chronic illnesses (e.g., diabetes mellitus or stroke), and psychoactive medications are associated with FI (64–70). Among women with no underlying systemic disease, diarrhea and rectal urgency are the strongest independent risk factors for FI. Although obstetric anal sphincter injury can cause immediate FI, it more typically begins 2–3 decades after vaginal delivery among unselected women (62). These observations suggest that similar to urinary incontinence, obstetric pelvic floor injury is an important risk factor for early-onset FI (e.g., postpartum FI) but much less so for late-onset FI.

Recommendations for diagnostic assessment

1. Gastroenterologists and other providers should ask patients about the presence of FI directly rather than relying on spontaneous reporting (strong recommendation, high quality of evidence).
2. Gastroenterologists and other providers should identify conditions that may predispose to FI, as shown in **Table 1** (strong recommendation, high quality of evidence).
3. Gastroenterologists and other providers should determine symptom severity by quantifying stool type using the Bristol stool scale, as well as characterizing the frequency, amount of leakage, and the presence of urgency (strong recommendation, moderate quality of evidence).
4. Gastroenterologists and other providers should obtain bowel diaries because they are superior to self-reports for characterizing bowel habits and FI (strong recommendation, moderate quality of evidence).

Pictorial representations of stool form (e.g., Bristol Stool Form Scale) and bowel diaries are efficient and reliable methods to characterize bowel habits and are better predictors of colonic transit than self-reported stool frequency (71,72). The frequency, amount (i.e., small stain, moderate amount (i.e., more than a stain but less than a full bowel movement), or large amount (i.e., full bowel movement)), type of leakage, and the presence of urgency should be ascertained to provide an index of symptom severity that is simple to calculate and strongly correlates with the effect of FI on the quality of life (57,62). Semiformed or liquid stools stress pelvic floor continence mechanisms more than formed stools; incontinence for solid stool suggests more severe sphincter weakness than does incontinence for liquid stool. An awareness of the desire to defecate before an incontinent episode may also provide clues to pathophysiology. Patients with *urge incontinence* experience the desire to defecate, but cannot reach the toilet on time. Patients with *passive incontinence* are not aware of the need to defecate before the incontinent episode. Patients with urge incontinence have reduced squeeze pressures (73) and/or squeeze duration (74) and/or reduced rectal capacity with rectal hypersensitivity (62), whereas patients with passive incontinence have lower resting pressures (73). Nocturnal incontinence occurs uncommonly and is most frequently encountered in diabetes mellitus and scleroderma.

Table 3. Common causes of fecal incontinence

Anal sphincter weakness
Traumatic: obstetric, surgical (e.g., fistulotomy, internal sphincterotomy)
Nontraumatic: scleroderma, internal sphincter degeneration of unknown etiology
Neuropathy: peripheral (e.g., pudendal) or generalized (e.g., diabetes mellitus)
Disturbances of pelvic floor: rectal prolapse, descending perineum syndrome
Inflammatory conditions: radiation proctitis, Crohn's disease, ulcerative colitis
Central nervous system disorders: dementia, stroke, brain tumors, multiple sclerosis, spinal cord lesions
Diarrhea: irritable bowel syndrome, post-cholecystectomy diarrhea
Other: fecal retention with overflow, behavioral disorders

Reproduced and modified with permission from Bharucha (160).

Recommendation for physical examination

5. Gastroenterologists and other providers should perform a physical examination to eliminate diseases to which FI is secondary (strong recommendation, moderate quality of evidence).
6. Gastroenterologists and other providers should perform a digital anorectal examination to identify rectal masses, gauge anal sphincter tone at rest, during voluntary contraction of the anal sphincter and pelvic floor muscles, and during simulated defecation (75) (strong recommendation, moderate quality of evidence).
7. Gastroenterologists and other providers should perform a digital rectal examination before making a referral for anorectal manometry (strong recommendation, moderate quality of evidence).

Perianal pinprick sensation and the anal wink reflex evaluate the integrity of the sacral lower motor neuron reflex arc. For experienced observers, agreement between digital assessment of anal sphincter function at rest and during squeeze and manometry is excellent (22,76). Other abnormalities in patients with FI include abnormal (i.e., increased or reduced) pelvic floor motion during evacuation, impacted stool in the rectal vault, and perianal soiling with feces. Reduced anal resting tone and/or a weak squeeze response are the most common features in FI.

The next steps are guided by the clinical assessment. For patients with mild symptoms and/or symptoms that are not bothersome, conservative measures alone suffice, often on an as-needed basis (77) (see beginning of Therapy section). If symptoms improve and there are no features to suggest an organic disorder, further testing may not be necessary. If symptoms do not improve, diagnostic testing can guide management (78,79).

Recommendations for diagnostic testing

8. ARM, BET, and rectal sensation should be evaluated in patients who fail to respond to conservative measures (strong recommendation, moderate quality of evidence).
9. Pelvic floor and anal canal imaging, as well as anal EMG, should be considered for patients with reduced anal pressures who have failed conservative therapy, particularly if surgery is being considered (strong recommendation, moderate quality of evidence).

Diagnostic tests should preferably be performed by laboratories with requisite expertise. Testing should begin with an ARM. Rectal sensation is evaluated concurrently, and rectal balloon expulsion and manometric changes during attempted expulsion of the manometric apparatus are performed if there is a suspicion of a defecation abnormality that could contribute to incontinence (**Figure 1**). In incontinent patients, anal sphincter resting and squeeze pressures are the key parameters. As anal sphincter pressures decline with age and are lower in women, age and gender should be taken into consideration when interpreting anal canal pressures (13,27,80,81). The anal cough reflex is also useful, in a qualitative sense, for evaluating the integrity of the lower motor neuron innervation of the external anal sphincter. Rectal sensation in FI may be normal, increased, or decreased. Rectal sensory disturbances and rectal evacuation disorders are potentially amenable to biofeedback therapy (**Figure 2**).

Further testing is guided by the results of initial tests and therapy. Anal imaging with endoanal ultrasound or MRI should be considered in patients with weak pressures if surgery is considered a possible option. Although the findings of endoanal ultrasound and MRI are generally congruent, each of these modalities has unique strengths (62). The internal sphincter is visualized more clearly by endoanal ultrasound, whereas MRI is superior for discriminating between an external anal sphincter tear and a scar and for identifying external sphincter atrophy. Internal sphincter defects probably reflect more severe anorectal injury than do external sphincter injuries alone (82,83). Interpreting the clinical significance of anal sphincter injury can be challenging even for experienced radiologists. Moreover, even asymptomatic women can have postpartum sphincter defects. Two-dimensional ultrasound has identified anal sphincter defects after vaginal delivery in up to one-third of women (84). With three-dimensional ultrasound or MRI, the prevalence is much lower (i.e., ~10%) (70,85).

Further testing (e.g., assessment of rectal compliance and sensation with a barostat, needle electromyography (EMG) of the anal sphincter, and assessment of pelvic floor motion by dynamic MRI or barium proctography) may be considered for patients who have refractory symptoms, especially if surgery is being considered. However, these tests are not widely available. Needle EMG of the anal sphincter should be considered in patients with clinically suspected neurogenic sphincter weakness, particularly if there are features suggestive of proximal (i.e., sacral root) involvement (86).

Recommendations for nonsurgical treatments

10. Gastroenterologists and other providers should manage patients with FI using education, dietary modifications, skin care, and pharmacologic agents to modify stool delivery and liquidity before diagnostic testing, particularly when

symptoms are mild and not bothersome (strong recommendation, moderate quality of evidence).

11. Gastroenterologists and other providers should prescribe antidiarrheal agents for FI in patients with diarrhea (strong recommendation, low quality of evidence).

Patients should be reassured that FI is not uncommon and often responds to simple measures. They should be educated about the contribution of bowel disturbances to FI, including the possible relationship between foods containing incompletely digested sugars (e.g., fructose, lactose) and caffeine to loose stools and urgency. They should also be informed about the effectiveness of behavioral urge resistance programs. A food and symptom diary may identify factors that cause diarrheal stools and incontinence. Although fiber supplements are often advocated to increase stool bulk and reduce watery stools, there is no published evidence to support this approach.

Several drugs to manage diarrhea (e.g., loperamide, diphenoxylate with atropine, bile salt-binding agents such as cholestyramine and colesevelam, anticholinergic agents, and clonidine) are available. A systematic Cochrane review of medical therapy for FI conducted in 2007 identified 13 randomized studies with 473 participants; 11 were crossover trials with short or no washout period between treatments (87). Nine trials included only individuals with FI related to liquid stool and seven tested antidiarrheal drugs (loperamide, diphenoxylate plus atropine, and codeine). In four trials, symptoms were better with active treatment compared with placebo; this improvement was characterized by improved and/or restored fecal continence (88–91), improved fecal urgency (89), more formed stools (89,91), and reduced use of pads (90). In two of these four trials, more individuals reported adverse effects such as constipation, abdominal pain, diarrhea, headache, and nausea when taking active drug (89,91). There were no adverse effects in either arm in one trial (90), and adverse effects were not reported in the other trail (88). Anorectal physiological measurements showed no clear differences between treatment periods.

Concomitant constipation should be managed with fiber supplementation and osmotic and/or stimulant laxatives (86). However, there are limited data based on two studies that used lactulose for FI associated with constipation in geriatric patients. In one study, geriatric patients who received lactulose (15 ml/day) required less nursing aid and had less soiled linen; however, only 57 of 87 participants completed this 3-week study (92). In another study, the number of episodes of FI and soiled laundry did not differ between individuals receiving lactulose and those receiving lactulose along with a rectal stimulant and weekly enemas (93). By facilitating rectal evacuation, glycerine and bisacodyl suppositories are well tolerated and may help some patients. Retrograde rectal washouts with tap water or phosphate solutions are an alternative, but some individuals with poor resting anal tone are unable to retain an enema. Except for patients with spinal cord injury, these treatments have not been formally evaluated in patients with FI.

In controlled clinical trials, conservative measures improve fecal continence. For example, in an RCT of 171 patients with FI, effects on symptoms (i.e., improved in 55% and resolved in 5%) and anal pressures were comparable among four groups: standard medical/nursing care (i.e., advice only), advice plus verbal instruction on sphincter exercises, hospital-based computer-assisted sphincter pressure biofeedback, or hospital biofeedback plus the use of a home EMG biofeedback device (94). Improvement was sustained at 1 year after each therapy. In another RCT of 108 patients, 22% reported adequate relief of FI after 4 weeks of conservative therapy (68). However, in contrast to urinary incontinence, prompted evacuation was not effective for FI in nursing home residents (95–97).

Anal plugs are available in some European countries but not in the United States. In a Cochrane review of four RCTs or quasi-randomized controlled studies involving 136 participants, 48 (35%) participants dropped out before the end of the study (98). However, in two studies that compared plugs with no plugs, stool loss was effectively blocked in six patients who tolerated and continued to use these plugs, at least in the short term. Patients with passive incontinence for small amounts of stool may benefit from a perianal cotton plug to absorb moisture and also perhaps to help with uncontrolled passage of gas. However, there are no formal studies with this intervention.

12. Pelvic floor rehabilitative techniques are effective and superior to pelvic floor exercises alone in patients with FI who do not respond to conservative measures (strong recommendation, moderate quality of evidence).

Patients can be taught to contract their pelvic floor muscles without (e.g., Kegel exercises) or with manometric or EMG-assisted biofeedback therapy. The latter requires a rectal balloon and anal manometry or a surface EMG device. Patients are taught to contract the external anal sphincter when they perceive balloon distention; perception may be reinforced by visual tracings of balloon volume and anal pressure, and the procedure is repeated with progressively smaller volumes. As described above, one study demonstrated that sphincter exercises or pelvic floor retraining were not superior to education alone for managing FI (94). In another study, 6 biweekly sessions of manometric biofeedback were superior (i.e., 77% reported adequate relief and 66% were completely continent) to pelvic floor exercises alone (i.e., 41% reported adequate relief and 48% were completely continent) in patients who did not respond to education alone (99). Biofeedback therapy often improves rectal sensation and may enhance coordination between perception of rectal distention and external sphincter contraction in patients with reduced rectal sensation (100,101). The effects of pelvic floor retraining on anal pressures are variable; anal squeeze pressures improved in some (94,99) but not in all studies (102).

Some patients with FI or fecal seepage have an underlying evacuation disorder (8,62) such as DD. In these patients, biofeedback can be used to train patients to relax their pelvic floor muscles during simulated defecation and to correlate relaxation and pushing to achieve defecation (see section on defecation disorders). Restoring normal coordination improves fecal

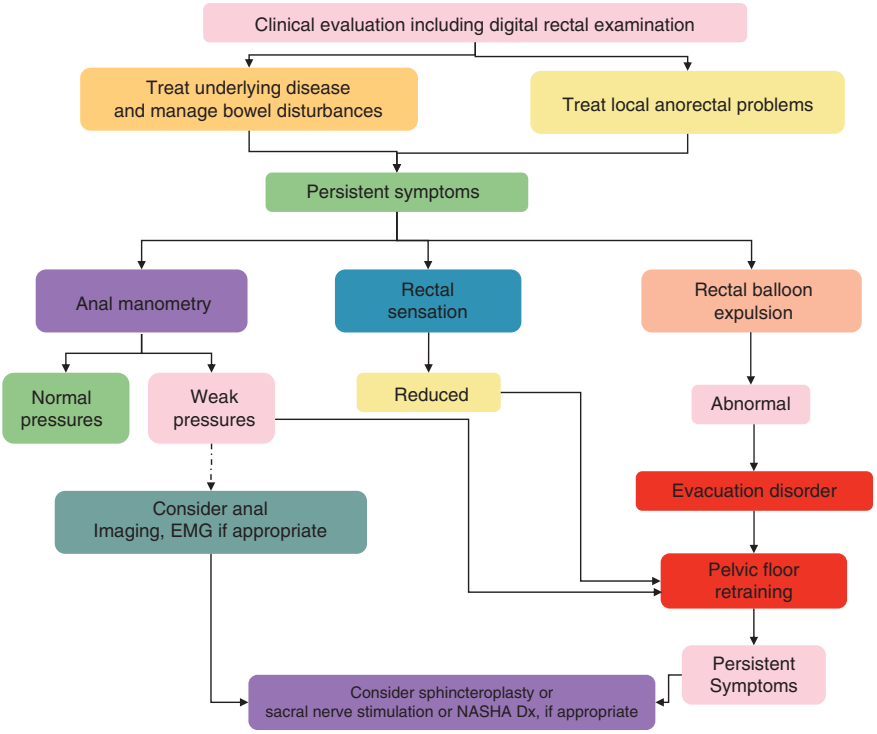

Figure 2. Suggested algorithm for evaluation and management of fecal incontinence.

evacuation. Because less stool is retained in the rectum, patients are less prone to leak. However, this approach has not been evaluated in controlled studies.

Biofeedback is not indicated in patients with isolated internal anal sphincter weakness, overflow incontinence associated with behavioral or psychiatric disorders, neurological disorders associated with substantial loss of rectal sensation and/or the inability to contract the striated muscles, decreased rectal storage capacity from resection, inflammation or fibrosis, or major structural damage to continence mechanisms.

Recommendations for minimally invasive procedures

13. Minimally invasive procedures such as injectable anal bulking agents may have a role in patients with FI who do not respond to conservative therapy (weak recommendation, moderate-quality of evidence).
14. There is insufficient evidence to recommend radiofrequency ablation treatment to the anal sphincter (SECCA) at this time (no recommendation, insufficient evidence).

Injectable bulking agents are used to augment the urethral sphincter and to treat urinary incontinence. One substance was recently approved by the Food and Drug Administration (FDA) for managing FI. In a multicenter, placebo-controlled randomized trial of a perianal bulking agent (nonanimal stabilized hyaluronic acid/dextranomer (NASHA/Dx)) in 206 patients with FI (103), a 50% reduction in incontinence episodes was reported more frequently for NASHA/Dx (52% of patients) than placebo (31%) at 12 months, of whom 6% became completely continent. With the exception of 2 serious adverse events (i.e., rectal abscess and prostatic abscess), most of the 128 adverse events were minor. An accompanying editorial (104) observed that without baseline severity data it is challenging to interpret the 50% reduction in episodes in absolute terms. Treatment did not affect embarrassment scores related to FI. ARM and imaging were not performed; hence, patient characteristics and mechanisms of action were unknown. It is unclear whether 4–8 ml of a substance can mechanically occlude the anus, which has a wide lumen, or whether this substance migrates. In summary, although these results seem promising, further studies to confirm the effects of this and other bulking agents on symptoms and anorectal functions in fecal continence are awaited (105).

Radiofrequency ablation therapy, which delivers temperature-controlled radiofrequency energy to the anal sphincter complex, was approved by the FDA in 2002 for treating FI that had failed conservative measures. The biological (and unproven) rationale is that heat applied to tissues results in collagen deposition and tissue contraction. Small industry-supported studies suggested efficacy (106), but these results were not duplicated by three small studies from Europe and Asia (107–109). All studies reported no changes in anorectal manometric measures, and reported improvements were tempered by the fact that FI persisted at levels that most would consider morbid. Further studies to evaluate the efficacy of this technique for FI are required.

Recommendations for surgical treatment

15. Sacral nerve stimulation should be considered in patients with FI who do not respond to conservative therapy (strong recommendation, moderate quality of evidence).
16. Anal sphincteroplasty should be considered in patients with FI who do not respond to conservative therapy and who have an anatomic sphincter defect (weak recommendation, low quality of evidence).
17. Dynamic graciloplasty and artificial anal sphincter, where available, may possibly allow the occasional patient with FI to avoid colostomy (weak recommendation, insufficient evidence).
18. Colostomy is a last resort procedure that can markedly improve the quality of life in a patient with severe or intractable FI (strong recommendation, low quality of evidence).

A recent Cochrane review identified 13 trials with 440 participants that evaluated the effects of surgery in adults with FI without rectal prolapse (110). Anterior and posterior pelvic floor repair, anterior sphincter repair and sphincter plication (with intact sphincter), antegrade colonic irrigation, and interventions designed to create a neosphincter (i.e., artificial anal sphincter or gracilis transposition) without or with electrical stimulation were included in this review, but sacral nerve stimulation (SNS) and anal bulking agents were not. The review concluded with the impression that the "small number of relevant trials identified together with their small sample sizes and other methodological weaknesses continue to limit the usefulness of this review for guiding practice. It was impossible to identify or refute clinically important differences between the alternative surgical procedures" (110).

SNS is approved for treating FI in both Europe and the United States. Patients whose symptoms respond to temporary SNS for 2–3 weeks proceed to subcutaneous implantation of the device. SNS is technically straightforward and major complications are infrequent. The pivotal North American multicenter study enrolled 133 patients who had FI (i.e., more than 2 incontinent episodes per week) for more than 6 months or for more than 12 months after childbirth and who had failed or were not candidates for conservative therapy (111,112). The success rate for temporary SNS was 90%; 120 patients (110 females) with a mean age of 60.5 years and a mean duration of FI of 6.8 years proceeded to chronic SNS. At 3-year follow-up of 83 patients, 86% achieved therapeutic success, as defined by a 50% reduction in the number of incontinent episodes per week; 40% achieved 100% continence. Incontinent episodes decreased from a mean of 9.4 per week at baseline to 1.7 at 12 months. There was significant improvement in all four scales of the Fecal Incontinence Quality of Life instrument at 12, 24, and 36 months of follow-up. The most common device-related adverse events included implant site pain (28%), paresthesia (15%), change in the sensation of stimulation (12%), and infection (10%). These results are very impressive, but the study was uncontrolled. Despite marked improvement in symptoms, SNS has had relatively minor or no effects on measured anorectal functions (113). However, in a recent study in 11 patients with urge FI, SNS resulted in a substantial increase in retrograde colonic propagating sequences that did not occur with sham stimulation (114). This suggests that SNS may improve FI through alterations in colonic motility rather than a direct effect on anorectal functions. On the basis of these and other studies, SNS should be considered for patients with FI whose symptoms are truly refractory to medical therapy and are otherwise eligible for the procedure.

Anal sphincteroplasty: Although short-term improvements in FI have been reported in up to 85% of patients after anal sphincteroplasty, continence deteriorates thereafter and there is a 50% failure rate after 40–60 months (115). Hence, anal sphincteroplasty is generally reserved for patients in whom FI and anal sphincter injury are recognized shortly after vaginal delivery and persist despite adequate therapy of coexisting bowel disturbances. Whether anal sphincter defects that are recognized several years after a presumed obstetric insult should be repaired is not clear, because the initial improvement in FI after overlapping anterior sphincteroplasty is not often sustained.

Dynamic graciloplasty: It involves continuous electrical stimulation of the gracilis muscle that is surgically transposed around the anal canal. Electrical stimulation facilitates anal tone by converting type II (fast-twitch, fatigue-prone) to type I (slow-twitch, fatigue-resistant) muscle fibers. The hardware for dynamic graciloplasty is approved in Europe but not in the United States. Although FI may improve in ~50% of patients, this procedure may be complicated by mortality (2% in 1 study), and significant morbidity (i.e., infections (28%), device problems (15%), and leg pain (13%)) for which reoperation may be required. Constipation has been reported in 13–90% of patients (116).

Artificial anal sphincter: The experience with implantation of an artificial anal sphincter, which is approved for use in Europe and the United States, is similar to that with dynamic graciloplasty. A systematic review of 14 studies with the artificial anal sphincter emphasized that most studies were case series with little or no follow-up of patients in whom the device failed (117). Moreover, complications were common, and the device was explanted in about one-third of patients. However, most patients with a functioning device reported clinically significant improvement in continence and quality of life.

Colostomy: A colostomy, typically an end sigmoid colostomy, is considered the last resort for patients with severe FI. It should be discussed early in the evaluation and management of a patient with severe FI, so that the patient knows that the option exists. It can be done with low morbidity even in very frail patients, and it may lead to marked improvement in the quality of life. In one study, the median score for the ability to live with a stoma was 8 and satisfaction with the stoma was rated as a median of 9, both on a scale of 0–10 (118). The majority (83%) felt that the stoma restricted their life "a little" or "not at all," and this was significantly improved from the perceived former restriction owing to incontinence. Also, 84% would "probably" or "definitely" choose to have the stoma again. Quality of life (short form (SF)-36) was poor, but neither depression nor anxiety was a prominent feature. Patients have a wide variety of reactions to the prospect of colostomy. Understanding the patient's

informed views toward the possibility of colostomy helps the gastroenterologist navigate the various options for severe FI.

ANAL FISSURE

Definitions and epidemiology

Anal fissure is an ulcer-like, longitudinal tear in the midline of the anal canal, distal to the dentate line. In almost 90% of cases, an idiopathic fissure is located in the posterior midline, but it can also occur in the anterior midline. Fissures in lateral positions should raise suspicion for disease processes such as Crohn's disease, tuberculosis, syphilis, HIV/AIDS, dermatologic conditions (e.g., psoriasis), and anal carcinoma. An acute fissure looks like a simple tear in the anoderm, whereas a chronic fissure, defined as lasting more than 8 to 12 weeks, is further characterized by edema and fibrosis. Fibers of the internal anal sphincter may be visible at the fissure base. Typical accompanying features of chronic fissures include a 'sentinel pile' (skin tag) at the distal fissure margin, and a hypertrophied anal papilla in the anal canal proximal to the fissure. The former is often described by patients as a "painful hemorrhoid," and the latter may be seen on endoscopic retroflexion. The clinical hallmark of anal fissure is pain during defecation and often persisting after defecation. Often, there is the history of a tearing sensation during passage of a hard stool or diarrhea. Rectal bleeding, usually limited to minimal bright red blood on toilet tissue, is frequent.

Chronic anal fissure is maintained as a nonhealing ulcer by sphincter spasm and consequent ischemia. Treatment for chronic anal fissure is typically directed toward relieving spasm, and as such it is a predominantly medical condition, with surgery reserved for medically refractory cases.

Recommendations for treatment of acute anal fissure

1. Gastroenterologists and other providers should use nonoperative treatments such as sitz baths, psyllium fiber, and bulking agents as the first step in therapy of acute fissure (strong recommendation, moderate quality of evidence).

Almost half of all patients with acute anal fissure will heal with supportive measures, i.e., sitz baths, psyllium fiber, and bulking agents, with or without the addition of topical anesthetics or anti-inflammatory ointments (119). In addition to fissure healing, symptomatic relief of pain and bleeding can be achieved with virtually no side effects.

Recommendations for treatment of chronic anal fissure

2. Gastroenterologists and other providers should treat chronic anal fissure with topical pharmacologic agents such as a calcium channel blockers or nitrates (strong recommendation, moderate quality of evidence).
3. Gastroenterologists and other providers should refer patients who do not respond to conservative or pharmacologic treatment for local injections of botulinum toxin (strong recommendation, low quality of evidence) or surgical internal anal sphincterotomy (strong recommendation, high quality of evidence).

Chronic anal fissure may be treated with topical nitrates, although nitrates are marginally superior to placebo with regard to healing. Topical nitrate medications such as 0.2% nitroglycerin ointment applied twice daily for 6–8 weeks have been associated with healing in at least 50% of treated chronic fissures, and the use of topical nitroglycerin significantly decreases pain during the therapy period. A Cochrane review of medical treatment of chronic anal fissure concluded that topical nitroglycerin remains only marginally better than placebo in healing anal fissures (120). Dose escalation does not improve healing rates and does increase side effects. The principal side effect is headache that is dose related and occurs in 20–30% of treated patients, often leading to cessation of therapy. Recurrence rates are significantly higher compared with surgery, although morbidity is lower.

Chronic anal fissure may also be treated with topical calcium channel blockers, with a lower incidence of adverse effects than topical nitrates. There are insufficient data to conclude whether they are superior to placebo in healing anal fissures. Topical calcium channel blockers such as 2% diltiazem applied twice daily for 6–8 weeks have been associated with healing of chronic anal fissure in 65 to 95% of patients (121). There is no clear consensus on dosing. Side effects include headache (in up to 25%) but occur less frequently than with topical nitrates (122). There are fewer RCTs of topical calcium channel blockers than of topical nitrate medications. Because topical diltiazem has a lower incidence of headache and fissure recurrence than topical nitroglycerin, it may be the preferred topical treatment (123). Oral calcium channel blockers may be as good as topical calcium channel blockers, suggesting that it is the drug rather than the route of administration that is important (124).

Botulinum toxin injection has superior healing rates compared with placebo, although it has the disadvantage of requiring a needle injection in a sensitive area. There is no consensus on dosage, precise site of administration, number of injections, or efficacy (125). Injection of botulinum toxin into the internal anal sphincter allows healing in 60% to 80% of fissures, and at a higher rate than placebo (126). The most common side effect is temporary incontinence of flatus in up to 18%, and of stool in 5%. Recurrence may occur in up to 42%, but patients may be retreated with similar results to initial treatment (127). Higher doses may improve healing and are as safe as lower doses (128). Topical nitrate medications may potentiate the effects of botulinum toxin in patients with refractory anal fissure (129). Patients in whom botulinum toxin injection fails should be recommended for lateral internal sphincterotomy (LIS) (130).

Novel nonsurgical fissure treatments under study include both new medications and support devices (131). Gonyautoxin injection has been reported in uncontrolled case series to be effective in decreasing resting pressure, improving pain, and healing chronic fissures (132), and thus it could become an alternative to botulinum toxin injection. However, there are no comparative studies between gonyautoxin and botulinum toxin injection. A posterior perineal support device, used as a modification of a toilet seat, decreased fissure-related symptoms in another case series (133);

this could become a stand-alone treatment or an adjunct to standard fissure treatments.

LIS, a procedure that can be performed under general, spinal, or local anesthesia, remains the surgical treatment of choice for refractory anal fissures (134). LIS is clearly superior to uncontrolled manual anal dilation, with better healing rates and less incontinence (134). It is also more efficacious than any topical or injectable treatment (135). There is no outcome difference between open and "closed" sphincterotomy, and thus a minimal-incision approach is probably preferred (136). Because of the low but real incidence of incontinence from LIS, surgeons continue to explore alternatives to LIS (137,138), but none is standard.

Controlled pneumatic balloon dilation has shown promise as an alternative to LIS in one small series (139), suggesting that an interested gastroenterologist, using the tools at his or her disposal, may treat even medically refractory anal fissures without resort to surgical consultation. However, surgical referral remains prudent for most cases of medical treatment failure in chronic anal fissure, because LIS is a safe and effective operation.

HEMORRHOIDS

Definitions and epidemiology

Hemorrhoids are among the most common problems encountered in the industrialized world. The normal proximal anal canal structures, called the anal cushions, are renamed internal hemorrhoids when they bleed and/or protrude. Hemorrhoids are not well understood, and a large number of diverse symptoms may be attributed to them by patients and referring physicians. The cardinal signs of internal hemorrhoids are hemorrhoid-pattern bleeding—defined as painless bleeding with bowel movements—and intermittent, reducible protrusion. It is often the role of the gastroenterologist to provide the "diagnosis of exclusion" of symptomatic internal hemorrhoids by ruling out other sources of bleeding and protrusion. The loosely related condition called thrombosed external hemorrhoid involves a clot in a vein under the anoderm that is the skin of the anal verge.

Recommendations for diagnostic assessment

1. Gastroenterologists and other providers should diagnose hemorrhoids by history and physical examination. If there is bleeding, the source often requires confirmation by endoscopic studies (strong recommendation, moderate quality of evidence).

Physical examination should include visual inspection of the anus, both at rest and while straining, and digital examination for other anal pathology. Internal hemorrhoids can be assigned a functional grade based on their history: first-degree hemorrhoids do not prolapse, second-degree hemorrhoids prolapse but self-reduce, third-degree hemorrhoids protrude and require manual reduction, and fourth-degree hemorrhoids protrude and cannot be reduced. Laboratory testing is almost never helpful. The clinical diagnosis of hemorrhoids usually includes hemorrhoid-pattern bleeding that mandates at least sigmoidoscopy to rule out other sources of bleeding. In patients over the age of 50 years or with a suggestive family history, this may be the occasion for evaluation of the entire colon, usually by colonoscopy (140,141).

Hemorrhoids without symptoms are not diagnosed by flexible endoscopy. Redundant anal cushions seen on sigmoidoscopy or colonoscopy may or may not be symptomatic. Recalling the functional definition of hemorrhoids as anal cushions that bleed and/or protrude, one can say that "hemorrhoids" seen endoscopically are not hemorrhoids until the patient defines them as such by describing protrusion or bleeding.

A thrombosed external hemorrhoid is easily recognized on physical examination as a usually tender blue lump at the anal verge, and no other workup or classification is needed. The only questions are whether it is identified early or late and whether symptoms are increasing or abating.

Recommendations for treatment of thrombosed external hemorrhoid

2. Most patients who present urgently (within ~3 days of onset) with a thrombosed external hemorrhoid benefit from excision (strong recommendation, low quality of evidence).

Although thrombosed external hemorrhoids treated without excision will eventually resolve their symptoms, excision of thrombosed external hemorrhoids gives more rapid symptom resolution, a lower incidence of recurrence, and longer remission intervals (142). Most excisions can be safely performed in the office or emergency room, with an injection of a local anesthetic. The thrombosis should be excised along with overlying skin to leave a wide open wound, rather than simply incised and drained that allows local recurrence. Thrombosed external hemorrhoids seen late, with symptoms improving and clot already resorbing, may be allowed to resolve without excision.

Recommendation for treatment of internal hemorrhoids

3. Gastroenterologists and other providers should treat patients with symptomatic hemorrhoids first with increased fiber intake and adequate fluids (strong recommendation, moderate quality of evidence).

A Cochrane review demonstrated the benefit of increased fiber intake in reducing both prolapse and bleeding (143). Laxatives have a limited role in the initial treatment of hemorrhoids (144). Patients should be counseled to avoid straining and limit their time spent on the commode, because both are associated with higher rates of symptomatic hemorrhoids (145). Because longstanding habits are hard to break, this advice may not be efficacious. Topical treatments for hemorrhoids, e.g., astringents and anti-inflammatories, are readily available over the counter and are commonly used by patients. They are of unclear value, although the use of an astringent enema to relieve symptoms has intuitive appeal.

4. Gastroenterologists and other providers should consider patients with first- to third-degree hemorrhoids that remain symptomatic after dietary modifications for office procedures such as banding, sclerotherapy, and infrared coagulation. Ligation is probably the most effective option (strong recommendation, moderate-quality of evidence).

All office-based procedures attempt to reduce redundancy, reduce vascularity, and increase fixation of the anal cushion to its underlying muscle. Many destructive techniques exist to downsize, devascularize, and scar anal cushions to decrease bleeding and protrusion. These office procedures are all relatively well tolerated, all display variable recurrence rates, and all may require repeated application (146). Because of the risk of significant bleeding, office procedures should generally be avoided in patients with thrombocytopenia or on warfarin, heparin products, and antiplatelet agents such as clopidogrel.

Rubber-band ligation (banding) is the most popular and effective office treatment for internal hemorrhoids. Ligation can be accomplished through a rigid anoscope or using a retroflexed flexible endoscope with a ligation attachment. In a meta-analysis of 18 randomized prospective studies of office treatments, banding had a lower need for repeated treatment compared with injection sclerotherapy and infrared coagulation, in the treatment of first- to third-degree hemorrhoids (146). It also had a higher, although still minuscule, complication rate, and it caused more pain. This flagship office treatment has also been compared with excisional hemorrhoidectomy for third-degree hemorrhoids. As expected, banding proved less effective, less painful, and had fewer complications than surgery (147). Ligation is probably the treatment of choice for second-degree hemorrhoids, and it is a reasonable first-line treatment for third-degree hemorrhoids. The use of suction to position the hemorrhoid for ligation is somewhat less painful and causes less bleeding than forceps, although both methods are acceptable (148). The most common complications of banding are anorectal pain, bleeding, thrombosis of external hemorrhoids, and vasovagal symptoms that occur in 1–3% of patients (149). Life-threatening septic complications have been reported (150) but are vanishingly rare.

Sclerotherapy involves the injection of a sclerosant into the apex of an internal hemorrhoid. It is successful in treating 75–90% of patients with first- to third-degree hemorrhoids (151). Recurrence is frequent, but retreatment is safe, with complications similar to ligation. Rarely, serious complications have resulted from erroneous injection or systemic effects of the sclerosant (152,153).

Infrared coagulation involves the contact application of infrared radiation, essentially cauterizing the hemorrhoid. This is most commonly used for first- and second-degree hemorrhoids. Randomized trials have demonstrated outcomes similar to banding (154). Both infrared coagulation and sclerotherapy can treat bleeding hemorrhoids that are too small to ligate.

5. Gastroenterologists and other providers should refer for surgical operations (hemorrhoidectomy, stapled hemorrhoidopexy, and Doppler-assisted hemorrhoidal artery ligation) those patients who are refractory to or cannot tolerate office procedures, who have large, symptomatic external tags along with their hemorrhoids, who have large third-degree hemorrhoids, or who have fourth-degree hemorrhoids (strong recommendation, moderate quality of evidence).

Traditional hemorrhoidectomy remains very effective. When compared with office procedures, hemorrhoidectomy was more effective for grade III hemorrhoids, but more painful, and had a higher complication rate (146). Standard hemorrhoidectomy leaves open or closed wounds (155), and it may be performed with a variety of surgical devices, none of which displays a clear advantage over the others (156).

Stapled hemorrhoidopexy uses a circular stapler to resect a ring of tissue rostral to the anal cushions, and to remove redundancy in the remaining anal cushions. Being highly effective for prolapsing internal hemorrhoids and less painful than hemorrhoidectomy, it may not adequately address external hemorrhoids. Systematic reviews demonstrated slightly lower complication rates and higher long-term recurrence rates with stapled hemorrhoidopexy compared with standard hemorrhoidectomy (157,158). Stapled hemorrhoidopexy is an established alternative to hemorrhoidectomy in most cases.

Doppler-assisted hemorrhoidal artery ligation uses a Doppler-equipped anoscope to identify and ligate the arteries supplying internal hemorrhoids. A potential comparative benefit is that less tissue is excised, although this may not address the problem of redundancy, as well as other operations. Success rates are comparable to those reported for both hemorrhoidectomy and stapled hemorrhoidopexy (159), although there have yet to be comparative studies.

CONFLICT OF INTEREST

Guarantor of the article: Arnold Wald, MD, MACG.
Specific author contributions: All authors played a role in writing individual sections and correcting and editing the final manuscript. Dr. Wald was responsible for the integration of the sections and overall organization of the manuscript.
Financial support: The ACG provided a modest stipend to support secretarial services for writing the manuscript.
Potential competing interests: None.

REFERENCES

1. Camilleri M, Parkman HP, Shafi MA *et al.* Clinical guideline: management of gastroparesis. Am J Gastroenterol 2013;108:18–37.
2. Bharucha AE, Dorn SD, Lembo A *et al.* American Gastroenterological Association medical position statement on constipation. Gastroenterology 2013;144:211–7.
3. Falck-Ytter Y, Kunz R, Guyatt GH *et al.* How strong is the evidence? Am J Gastroenterol 2008;103:1334–8.
4. Wald A, Bharucha AE, Enck P *et al.* Functional anorectal disorders. In: Drossman DA, Corazziari E, Delvaux M, *et al.* (eds). Rome III: The Functional Gastrointestinal Disorders. McLean, Virginia: Degnon Associates, 2006, pp. 639–85.
5. Bharucha AE, Wald A, Enck P *et al.* Functional anorectal disorders. Gastroenterology 2006;130:1510–8.

6. Ratuapli S, Bharucha AE, Harvey D *et al.* Comparison of rectal balloon expulsion test in seated and left lateral positions. Neurogastroenterol Motil 2013;25:e813–20.
7. Rao SS, Mudipalli RS, Stessman M *et al.* Investigation of the utility of colorectal function tests and Rome II criteria in dyssynergic defecation (Anismus). Neurogastroenterol Motil 2004;16:589–96.
8. Rao SS, Ozturk R, Laine L. Clinical utility of diagnostic tests for constipation in adults: a systematic review. Am J Gastroenterol 2005;100:1605–15.
9. Chiarioni G, Whitehead WE, Pezza V *et al.* Biofeedback is superior to laxatives for normal transit constipation due to pelvic floor dyssynergia. Gastroenterology 2006;130:657–64.
10. Rao SS, Seaton K, Miller M *et al.* Randomized controlled trial of biofeedback, sham feedback, and standard therapy for dyssynergic defecation. Clin Gastroenterol Hepatol 2007;5:331–8.
11. Heymen S, Scarlett Y, Jones K *et al.* Randomized, controlled trial shows biofeedback to be superior to alternative treatments for patients with pelvic floor dyssynergia-type constipation. Dis Colon Rectum 2007;50:428–41.
12. Ratuapli SK, Bharucha AE, Noelting J *et al.* Phenotypic identification and classification of functional defecatory disorders using high-resolution anorectal manometry. Gastroenterology 2013;144:314–22.
13. Noelting J, Ratuapli SK, Bharucha AE *et al.* Normal values for high-resolution anorectal manometry in healthy women: effects of age and significance of rectoanal gradient. Am J Gastroenterol 2012;107:1530–6.
14. Rao SS, Welcher KD, Leistikow JS. Obstructive defecation: a failure of rectoanal coordination. Am J Gastroenterol 1998;93:1042–50.
15. Rao SS, Kavlock R, Rao S. Influence of body position and stool characteristics on defecation in humans. Am J Gastroenterol 2006;101:2790–6.
16. Whitehead WE, di Lorenzo C, Leroi AM *et al.* Conservative and behavioural management of constipation. Neurogastroenterol Motil 2009;21 (Suppl 1): 55–61.
17. Leroi AM, Berkelmans I, Denis P *et al.* Anismus as a marker of sexual abuse. Consequences of abuse on anorectal motility. Dig Dis Sci 1995;40: 1411–6.
18. Glia A, Lindberg G, Nilsson LH *et al.* Clinical value of symptom assessment in patients with constipation. Dis Colon Rectum 1999;42:1401–8.
19. Grotz RL, Pemberton JH, Talley NJ *et al.* Discriminant value of psychological distress, symptom profiles, and segmental colonic dysfunction in outpatients with severe idiopathic constipation. Gut 1994;35:798–802.
20. Minguez M, Herreros B, Sanchiz V *et al.* Predictive value of the balloon expulsion test for excluding the diagnosis of pelvic floor dyssynergia in constipation. Gastroenterology 2004;126:57–62.
21. Chiarioni G, Pieramico O, Vantini I *et al.* Utility of physical examination and questionnaire for identifying patients who do not have dyssynergic defecation. Gastroenterology 2011;140:S 800.
22. Tantiphlachiva K, Rao P, Attaluri A *et al.* Digital rectal examination is a useful tool for identifying patients with dyssynergia. Clin Gastroenterol Hepatol 2010;8:955–60.
23. Raza N, Bielefeldt K. Discriminative value of anorectal manometry in clinical practice. Dig Dis Sci 2009;54:2503–11.
24. Chiarioni G, Whitehead WE. Biofeedback therapy for constipation. In: Parkman HP, McCallum RW, Rao SSC (eds). GI Motility Testing: A Laboratory and Office Handbook. 2011, pp. 179–87.
25. Chiarioni G, Pieramico O, Vantini I *et al.* Utility of the balloon-evacuation test for identifying patients with dyssynergic defecation. Gastroenterology 2011;140:S–797.
26. Whitehead WE, Schuster MM. Anorectal physiology and pathophysiology. Am J Gastroenterol 1987;82:487–97.
27. Diamant NE, Kamm MA, Wald A *et al.* AGA technical review on anorectal testing techniques. Gastroenterology 1999;116:735–60.
28. Chiarioni G, Kim SM, Vantini I *et al.* Validation of the balloon evacuation test: reproducibility and agreement with findings from anorectal manometry and electromyography. Clin Gastroenterol Hepatol 2014 (e-pub ahead of print).
29. Pelsang RE, Rao SS, Welcher K. FECOM: a new artificial stool for evaluating defecation. Am J Gastroenterol 1999;94:183–6.
30. Agachan F, Pfeifer J, Wexner SD. Defecography and proctography. Results of 744 patients. Dis Colon Rectum 1996;39:899–905.
31. Bordeianou L, Savitt L, Dursun A. Measurements of pelvic floor dyssynergia: which test result matters? Dis Colon Rectum 2011;54:60–5.
32. Bharucha AE, Fletcher JG, Seide B *et al.* Phenotypic variation in functional disorders of defecation. Gastroenterology 2005;128:1199–210.
33. Kaufman HS, Buller JL, Thompson JR *et al.* Dynamic pelvic magnetic resonance imaging and cystocolpoproctography alter surgical management of pelvic floor disorders. Dis Colon Rectum 2001;44:1575–83.
34. Kelvin FM, Maglinte DD, Hale DS *et al.* Female pelvic organ prolapse: a comparison of triphasic dynamic MR imaging and triphasic fluoroscopic cystocolpoproctography. AJR Am J Roentgenol 2000;174:81–8.
35. Nullens S, Nelsen T, Camilleri M *et al.* Regional colon transit in patients with dys-synergic defaecation or slow transit in patients with constipation. Gut 2012;61:1132–9.
36. Chiarioni G, Salandini L, Whitehead WE. Biofeedback benefits only patients with outlet dysfunction, not patients with isolated slow transit constipation. Gastroenterology 2005;129:86–97.
37. Simon MA, Bueno AM. Behavioural treatment of the dyssynergic defecation in chronically constipated elderly patients: a randomized controlled trial. Appl Psychophysiol Biofeedback 2009;34:273–7.
38. Rao SS, Valestin J, Brown CK *et al.* Long-term efficacy of biofeedback therapy for dyssynergic defecation: randomized controlled trial. Am J Gastroenterol 2010;105:890–6.
39. Pourmomeny AA, Emami MH, Amooshahi M *et al.* Comparing the efficacy of biofeedback and balloon-assisted training in the treatment of dyssynergic defecation. Can J Gastroenterol 2011;25:89–92.
40. Koutsomanis D, Lennard-Jones JE, Roy AJ *et al.* Controlled randomised trial of visual biofeedback versus muscle training without a visual display for intractable constipation. Gut 1995;37:95–9.
41. Farid M, El Monem HA, Omar W *et al.* Comparative study between biofeedback retraining and botulinum neurotoxin in the treatment of anismus patients. Int J Colorectal Dis 2009;24:115–20.
42. Faried M, El Nakeeb A, Youssef M *et al.* Comparative study between surgical and non-surgical treatment of anismus in patients with symptoms of obstructed defecation: a prospective randomized study. J Gastrointest Surg 2010;14:1235–43.
43. Loening-Baucke V. Functional fecal retention with encopresis in childhood. J Pediatr Gastroenterol Nutr 2004;38:79–84.
44. Wald A, Chandra R, Gabel S *et al.* Evaluation of biofeedback in childhood encopresis. J Pediatr Gastroenterol Nutr 1987;6:554–8.
45. Loening-Baucke V. Modulation of abnormal defecation dynamics by biofeedback treatment in chronically constipated children with encopresis. J Pediatr 1990;116:214–22.
46. van der Plas RN, Benninga MA, Buller HA *et al.* Biofeedback training in treatment of childhood constipation: a randomised controlled study. Lancet 1996;348:776–80.
47. Nolan T, Catto-Smith T, Coffey C *et al.* Randomised controlled trial of biofeedback training in persistent encopresis with anismus. Arch Dis Child 1998;79:131–5.
48. Sunic-Omejc M, Mihanovic M, Bilic A *et al.* Efficiency of biofeedback therapy for chronic constipation in children. Coll Antropol 2002; 26 (Suppl): 93–101.
49. Pieczarkowski S, Fyderek K. Usefulness of biofeedback treatment in children with chronic functional constipation. Przegl Lek 2005;62: 851–4.
50. Kajbafzadeh AM, Sharifi-Rad L, Ghahestani SM *et al.* Animated biofeedback: an ideal treatment for children with dysfunctional elimination syndrome. J Urol 2011;186:2379–84.
51. Hart SL, Lee JW, Berian J *et al.* A randomized controlled trial of anorectal biofeedback for constipation. Int J Colorectal Dis 2012;27:459–66.
52. Wald A BA, Enck P *et al.* Functional Anorectal Disorders. Degnon Associates:, 2006.
53. Chiarioni G, Nardo A, Vantini I *et al.* Biofeedback is superior to electrogalvanic stimulation and massage for treatment of levator ani syndrome. Gastroenterology 2010;138:1321–9.
54. Bharucha AE, Trabuco E. Functional and chronic anorectal and pelvic pain disorders. Gastroenterol Clin North Am 2008;37:685–96.
55. Jeyarajah S, Chow A, Ziprin P. Proctalgia fugax: an evidence based management pathway. Int J Colorectal Dis 2010;25:1037–46.
56. Norton C, Whitehead WE, Bliss DZ *et al.* Management of fecal incontinence in adults. Neurourol Urodynam 2010;29:199–206.
57. Bharucha AE, Zinsmeister AR, Locke GR *et al.* Symptoms and quality of life in community women with fecal incontinence. Clin Gastroenterol Hepatol 2006;4:1004–9.
58. Grover M, Whitehead WE. Is fecal incontinence a risk factor for institutionalization in the elderly? Am J Gastroenterol 2011;106:366–7.
59. Nelson R, Furner S, Jesudason V. Fecal incontinence in Wisconsin nursing homes: prevalence and associations. Dis Colon Rectum 1998;41: 1226–9.
60. Drossman DA, Li Z, Andruzzi E *et al.* U.S. householder survey of functional gastrointestinal disorders. Prevalence, sociodemography, and health impact. Dig Dis Sci 1993;38:1569–80.

61. Leigh RJ, Turnberg LA. Faecal incontinence: the unvoiced symptom. Lancet 1982;1:1349–51.
62. Bharucha AE, Fletcher JG, Harper CM *et al.* Relationship between symptoms and disordered continence mechanisms in women with idiopathic fecal incontinence. Gut 2005;54:546–55.
63. Andrews CN, Bharucha AE. The etiology, assessment, and treatment of fecal incontinence. Nat Clin Pract Gastroenterol Hepatol 2005;2: 516–25.
64. Quander CR, Morris MC, Melson J *et al.* Prevalence of and factors associated with fecal incontinence in a large community study of older individuals. Am J Gastroenterol 2005;100:905–9.
65. Kalantar JS, Howell S, Talley NJ. Prevalence of faecal incontinence and associated risk factors; an underdiagnosed problem in the Australian community? Med J Aust 2002;176:54–7.
66. Bharucha AE, Zinsmeister AR, Locke GR *et al.* Risk factors for fecal incontinence: a population-based study in women. Am J Gastroenterol 2006;101:1305–12.
67. Bharucha AE, Seide BM, Zinsmeister AR *et al.* Relation of bowel habits to fecal incontinence in women. Am J Gastroenterol 2008;103:1470–5.
68. Heymen S, Scarlett Y, Jones K *et al.* Randomized controlled trial shows biofeedback to be superior to alternative treatments for fecal incontinence. Dis Colon Rectum 2009;52:1730–7.
69. Bharucha AE, Zinsmeister AR, Schleck CD *et al.* Bowel disturbances are the most important risk factors for late onset fecal incontinence: a population-based case-control study in women. Gastroenterology 2010;139:1559–66.
70. Bharucha AE, Fletcher JG, Melton III LJ *et al.* Obstetric trauma, pelvic floor injury and fecal incontinence: a population-based case-control study. Am J Gastroenterol 2012;107:902–11.
71. Degen LP, Phillips SF. How well does stool form reflect colonic transit? Gut 1996;39:109–13.
72. Saad RJ, Rao SS, Koch KL *et al.* Do stool form and frequency correlate with whole-gut and colonic transit? Results from a multicenter study in constipated individuals and healthy controls. Am J Gastroenterol 2010;105:403–11.
73. Engel AF, Kamm MA, Bartram CI *et al.* Relationship of symptoms in faecal incontinence to specific sphincter abnormalities. Int J Colorectal Dis 1995;10: 152–5.
74. Chiarioni G, Scattolini C, Bonfante F *et al.* Liquid stool incontinence with severe urgency: anorectal function and effective biofeedback treatment. Gut 1993;34:1576–80.
75. Bharucha AE, Wald A. Anorectal disorders. Am J Gastroenterol 2010;105:786–94.
76. Orkin BA, Sinykin SB, Lloyd PC. The digital rectal examination scoring system (DRESS). Dis Colon Rectum 2010;53:1656–60.
77. Wald A. Clinical practice. Fecal incontinence in adults. N Engl J Med 2007;356:1648–55.
78. Bharucha AE. Pro: Anorectal testing is useful in fecal incontinence. Am J Gastroenterol 2006;101:2679–81.
79. Wald A. Con: Anorectal manometry and imaging are not necessary in patients with fecal incontinence. Am J Gastroenterol 2006;101:2681–3.
80. Bharucha AE. Update of tests of colon and rectal structure and function. J Clin Gastroenterol 2006;40:96–103.
81. Bharucha AE, Fletcher JG. Recent advances in assessing anorectal structure and functions. Gastroenterology 2007;133:1069–74.
82. Richter HE, Fielding JR, Bradley CS *et al.* Endoanal ultrasound findings and fecal incontinence symptoms in women with and without recognized anal sphincter tears. Obstet Gynecol 2006;108:1394–401.
83. Bradley CS, Richter HE, Gutman RE *et al.* Risk factors for sonographic internal anal sphincter gaps 6-12 months after delivery complicated by anal sphincter tear. Am J Obstet Gynecol 2007;197:310 e1-5.
84. Bharucha AE. Fecal incontinence. Gastroenterology 2003;124:1672–85.
85. Williams AB, Bartram CI, Halligan S *et al.* Alteration of anal sphincter morphology following vaginal delivery revealed by multiplanar anal endosonography. BJOG 2002;109:942–6.
86. Bharucha AE, Daube J, Litchy W *et al.* Anal sphincteric neurogenic injury in asymptomatic nulliparous women and fecal incontinence. Am J Physiol Gastrointest Liver Physiol 2012;303:G256–62.
87. Cheetham M, Brazzelli M, Norton C *et al.* Drug treatment for faecal incontinence in adults. Cochrane Database Syst Rev 2003;(3):CD002116.
88. Harford WV, Krejs GJ, Santa Ana CA *et al.* Acute effect of diphenoxylate with atropine (Lomotil) in patients with chronic diarrhea and fecal incontinence. Gastroenterology 1980;78:440–3.
89. Read M, Read NW, Barber DC *et al.* Effects of loperamide on anal sphincter function in patients complaining of chronic diarrhea with fecal incontinence and urgency. Dig Dis Sci 1982;27:807–14.
90. Hallgren T, Fasth S, Delbro DS *et al.* Loperamide improves anal sphincter function and continence after restorative proctocolectomy. Dig Dis Sci 1994;39:2612–8.
91. Sun WM, Read NW, Verlinden M. Effects of loperamide oxide on gastrointestinal transit time and anorectal function in patients with chronic diarrhoea and faecal incontinence. Scand J Gastroenterol 1997;32:34–8.
92. Ryan D, Wilson A, Muir TS *et al.* The reduction of faecal incontinence by the use of "Duphalac" in geriatric patients. Curr Med Res Opin 1974;2:329–33.
93. Chassagne P, Jego A, Gloc P *et al.* Does treatment of constipation improve faecal incontinence in institutionalized elderly patients? Age Ageing 2000;29:159–64.
94. Norton C, Chelvanayagam S, Wilson-Barnett J *et al.* Randomized controlled trial of biofeedback for fecal incontinence. Gastroenterology 2003;125:1320–9.
95. Ouslander JG, Simmons S, Schnelle J *et al.* Effects of prompted voiding on fecal continence among nursing home residents. J Am Geriatr Soc 1996;44:424–8.
96. Schnelle JF, Leung FW, Rao SS *et al.* A controlled trial of an intervention to improve urinary and fecal incontinence and constipation. J Am Geriatr Soc 2010;58:1504–11.
97. Simmons SF, Schnelle JF. Effects of an exercise and scheduled-toileting intervention on appetite and constipation in nursing home residents. J Nutr Health Aging 2004;8:116–21.
98. Deutekom M, Dobben AC. Plugs for containing faecal incontinence. Cochrane Database Syst Rev 2012;4:CD005086.
99. Heymen S, Scarlett Y, Jones K *et al.* Randomized controlled trial shows biofeedback to be superior to pelvic floor exercises for fecal incontinence. Dis Colon Rectum 2009;52:1730–7.
100. Wald A, Tunuguntla AK. Anorectal sensorimotor dysfunction in fecal incontinence and diabetes mellitus. Modification with biofeedback therapy. N Engl J Med 1984;310:1282–7.
101. Chiarioni G, Bassotti G, Stanganini S *et al.* Sensory retraining is key to biofeedback therapy for formed stool fecal incontinence. Am J Gastroenterol 2002;97:109–17.
102. Bharucha AE. Outcome measures for fecal incontinence: anorectal structure and function. Gastroenterology 2004;126:S90–8.
103. Graf W, Mellgren A, Matzel KE *et al.* Efficacy of dextranomer in stabilised hyaluronic acid for treatment of faecal incontinence: a randomised, sham-controlled trial. Lancet 2011;377:997–1003.
104. Norton C. Treating faecal incontinence with bulking-agent injections. Lancet 2011;377:971–2.
105. Maeda Y, Laurberg S, Norton C. Perianal injectable bulking agents as treatment for faecal incontinence in adults. Cochrane Database Syst Rev 2013;2:CD007959.
106. Efron JE, Corman ML, Fleshman J. Safety and effectiveness of temperature controlled radio-frequency energy delivery to the anal canal (Secca procedure) for the treatment of fecal incontinence. Dis Colon Rectum 2003;46:1606–16.
107. Felt-Bersma RJ, Szojda MM, Mulder CJ. Temperature controlled radiofrequency energy (SECCA) to the anal canal for the treatment of faecal incontinence offers moderate improvement. Eur J Gastroenterol Hepatol 2007;19:575–80.
108. Lefebure B, Tuech JJ, Bridoux V. Temperature controlled radio frequency energy delivery (Secca procedure) for the treatment of fecal incontinence: results of a prospective study. Int J Colorectal Dis 2008;23:993–7.
109. Kim DW, Yoon HM, Park JS. Radiofrequency energy delivery to the anal canal: is it a promising new approach to the treatment of fecal incontinence? Am J Surg 2009;197:14–8.
110. Brown SR, Wadhawan H, Nelson RL. Surgery for faecal incontinence in adults. Cochrane Database Syst Rev 2010;7:CD001757.
111. Wexner SD, Coller JA, Devroede G *et al.* Sacral nerve stimulation for fecal incontinence: results of a 120-patient prospective multicenter study. Ann Surg 2010;251:441–9.
112. Mellgren A, Wexner SD, Coller JA *et al.* Long-term efficacy and safety of sacral nerve stimulation for fecal incontinence. Dis Colon Rectum 2011;54:1065–75.
113. Michelsen HB, Worsoe J, Krogh K *et al.* Rectal motility after sacral nerve stimulation for faecal incontinence. Neurogastroenterol Motil 2010;22:36–41.
114. Patton V, Wiklendt L, Arkwright JW *et al.* The effect of sacral nerve stimulation on distal colonic motility in patients with faecal incontinence. Br J Surg 2013;100:959–68.
115. Cheung O, Wald A. Review article: the management of pelvic floor disorders. Aliment Pharmacol Ther 2004;19:481–95.
116. Koch SM, Uludaq O, El Naggar K *et al.* Colonic irrigation for defecation disorders after dynamic graciloplasty. Int J Colorectal Dis 2008;23: 195–200.

117. Mundy L, Merlin TL, Maddern GJ *et al.* Systematic review of safety and effectiveness of an artificial bowel sphincter for faecal incontinence. Br J Surg 2004;91:665–72.
118. Norton C, Burch J, Kamm MA. Patients' views of a colostomy for fecal incontinence. Dis Colon Rectum 2005;48:1062–9.
119. Gupta P. Randomized, controlled study comparing sitz-bath and no-sitz-bath treatments in patients with acute anal fissures. ANZ J Surg 2006;76:718–21.
120. Nelson RL, Thomas K, Morgan J *et al.* Non surgical therapy for anal fissure. Cochrane Database Syst Rev 2012;2:CD003431.
121. Shrivastava UK, Jain BK, Kumar P *et al.* A comparison of the effects of diltiazem and glyceryl trinitrate ointment in the treatment of chronic anal fissure: a randomized clinical trial. Surg Today 2007;37:482–5.
122. Bielecki K, Kolodziejczak M. A prospective randomized trial of diltiazem and glyceryltrinitrate ointment in the treatment of chronic anal fissure. Colorectal Dis 2003;5:256–7.
123. Sajid MS, Whitehouse PA, Sains P *et al.* Systematic review of the use of topical diltiazem compared with glyceryltrinitrate for the nonoperative management of chronic anal fissure. Colorectal Dis 2013;15:19–26.
124. Agrawal V, Kaushal G, Gupta R. Randomized controlled pilot trial of nifedipine as oral therapy vs. topical application in the treatment of fissure-in-ano. Am J Surg 2013;206:748–51.
125. Yiannakopoulou E. Botulinum toxin and anal fissure: efficacy and safety systematic review. Int J Colorectal Dis 2012;27:1–9.
126. Arroyo A, Perez F, Serrano P *et al.* Long-term results of botulinum toxin for the treatment of chronic anal fissure: prospective clinical and manometric study. Int J Colorectal Dis 2005;20:267–71.
127. Lindsey I, Smilgin-Humphreys MM, Cunningham C *et al.* A randomized, controlled trial of fibrin glue vs. conventional treatment for anal fistula. Dis Colon Rectum 2002;45:1608–15.
128. Madalinski MH, Slawek J, Zbytek B *et al.* Topical nitrates and the higher doses of botulinum toxin for chronic anal fissure. Hepatogastroenterology 2001;48:977–9.
129. Lysy J, Israelit-Yatzkan Y, Sestiery-Ittah M *et al.* Topical nitrates potentiate the effect of botulinum toxin in the treatment of patients with refractory anal fissure. Gut 2001;48:221–4.
130. Shao WJ, Li GC, Zhang ZK. Systematic review and meta-analysis of randomized controlled trials comparing botulinum toxin injection with lateral internal sphincterotomy for chronic anal fissure. Int J Colorectal Dis 2009;24:995–1000.
131. Poh A, Tan KY, Seow-Choen F. Innovations in chronic anal fissure treatment: a systematic review. World J Gastrointest Surg 2010;2:231–41.
132. Garrido R, Lagos N, Lagos M *et al.* Treatment of chronic anal fissure by gonyautoxin. Colorectal Dis 2007;9:619–24.
133. Tan KY, Seow-Choen F, Hai CH *et al.* Posterior perineal support as treatment for anal fissures--preliminary results with a new toilet seat device. Tech Coloproctol 2009;13:11–5.
134. Nelson RL, Chattopadhyay A, Brooks W *et al.* Operative procedures for fissure in ano. Cochrane Database Syst Rev 2011;(11).
135. de Rosa M, Cestaro G, Vitiello C *et al.* Conservative versus surgical treatment for chronic anal idiopathic fissure: a prospective randomized trial. Updates Surg 2013;65:197–200.
136. Mentes BB, Tezcaner T, Yilmaz U *et al.* Results of lateral internal sphincterotomy for chronic anal fissure with particular reference to quality of life. Dis Colon Rectum 2006;49:1045–51.
137. Singh M, Sharma A, Gardiner A *et al.* Early results of a rotational flap to treat chronic anal fissures. Int J Colorectal Dis 2005;20:339–42.
138. Pelta AE, Davis KG, Armstrong DN. Subcutaneous fissurotomy: a novel procedure for chronic fissure-in-ano. a review of 109 cases. Dis Colon Rectum 2007;50:1662–7.
139. Renzi A, Izzo D, Di Sarno G *et al.* Clinical, manometric, and ultrasonographic results of pneumatic balloon dilatation vs. lateral internal sphincterotomy for chronic anal fissure: a prospective, randomized, controlled trial. Dis Colon Rectum 2008;51:121–7.
140. Ko C, Hyman NH, Standards Committee of The American Society of Colon and Rectal Surgeons Practice parameter for the detection of colorectal neoplasms: an interim report (revised). Dis Colon Rectum 2006;49:299–301.
141. Levin B, Lieberman DA, McFarland B *et al.* Screening and surveillance for the early detection of colorectal cancer and adenomatous polyps, 2008: a joint guideline from the American Cancer Society, the US Multi-Society Task Force on Colorectal Cancer, and the American College of Radiology. Gastroenterology 2008;134:1570–95.
142. Greenspon J, Williams SB, Young HA *et al.* Thrombosed external hemorrhoids: outcome after conservative or surgical management. Dis Colon Rectum 2004;47:1493–8.
143. Alonso-Coello P, Mills E, Heels-Ansdell D *et al.* Fiber for the treatment of hemorrhoids complications: a systematic review and meta-analysis. Am J Gastroenterol 2006;101:181–8.
144. Alonso-Coello P, Guyatt G, Heels-Ansdell D *et al.* Laxatives for the treatment of hemorrhoids. Cochrane Database Syst Rev 2005;(4):CD004649.
145. Johannsson HO, Graf W, Pahlman L. Bowel habits in hemorrhoid patients and normal subjects. Am J Gastroenterol 2005;100:401–6.
146. MacRae HM, McLeod RS. Comparison of hemorrhoidal treatment modalities. A meta-analysis. Dis Colon Rectum 1995;38:687–94.
147. Shanmugam V, Thaha MA, Rabindranath KS *et al.* Rubber band ligation versus excisional haemorrhoidectomy for haemorrhoids. Cochrane Database Syst Rev 2005;(3):CD005034.
148. Ramzisham AR, Sagap I, Nadeson S *et al.* Prospective randomized clinical trial on suction elastic band ligator versus forceps ligator in the treatment of haemorrhoids. Asian J Surg 2005;28:241–5.
149. El Nakeeb AM, Fikry AA, Omar WH *et al.* Rubber band ligation for 750 cases of symptomatic hemorrhoids out of 2200 cases. World J Gastroenterol 2008;14:6525–30.
150. Sim HL, Tan KY, Poon PL *et al.* Life-threatening perineal sepsis after rubber band ligation of haemorrhoids. Tech Coloproctol 2009;13:161–4.
151. Kanellos I, Goulimaris I, Vakalis I *et al.* Long-term evaluation of sclerotherapy for haemorrhoids. A prospective study. Int J Surg Investig 2000;2: 295–8.
152. Schulte T, Fandrich F, Kahlke V. Life-threatening rectal necrosis after injection sclerotherapy for haemorrhoids. Int J Colorectal Dis 2008;23: 725–6.
153. Murray-Lyon IM, Kirkham JS. Hepatic abscesses complicating injection sclerotherapy of haemorrhoids. Eur J Gastroenterol Hepatol 2001;13: 971–2.
154. Marques CF, Nahas SC, Nahas CS *et al.* Early results of the treatment of internal hemorrhoid disease by infrared coagulation and elastic banding: a prospective randomized cross-over trial. Tech Coloproctol 2006;10:312–7.
155. You SY, Kim SH, Chung CS *et al.* Open vs. closed hemorrhoidectomy. Dis Colon Rectum 2005;48:108–13.
156. Nienhuijs S, de Hingh I. Conventional versus LigaSure hemorrhoidectomy for patients with symptomatic hemorrhoids. Cochrane Database Syst Rev 2009;(1):CD006761.
157. Tjandra JJ, Chan MK. Systematic review on the procedure for prolapse and hemorrhoids (stapled hemorrhoidopexy). Dis Colon Rectum 2007;50:878–92.
158. Jayaraman S, Colquhoun PH, Malthaner RA. Stapled hemorrhoidopexy is associated with a higher long-term recurrence rate of internal hemorrhoids compared with conventional excisional hemorrhoid surgery. Dis Colon Rectum 2007;50:1297–305.
159. Ratto C, Donisi L, Parello A *et al.* Evaluation of transanal hemorrhoidal dearterialization as a minimally invasive therapeutic approach to hemorrhoids. Dis Colon Rectum 2010;53:803–11.
160. Bharucha AE. Fecal incontinence. In Talley NJ, Locke GR III, Saito YA (eds). GI Epidemiology. Blackwell: Malden, 2013.
161. Bharucha AE, Rao SSC. An update on anorectal disorders for gastroenterologists. Gastroenterol 2014;146:37–45.