**BRIEF REVIEW**

**An Update on Anorectal Disorders for Gastroenterologists**

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Gastroenterologists frequently encounter pelvic floor disorders, which affect 10% to 15% of the population. The anorectum is a complex organ that collaborates with the pelvic floor muscles to preserve fecal continence and enable defecation. A careful clinical assessment is critical for the diagnosis and management of defecatory disorders and fecal incontinence. Newer diagnostic tools (eg, high-resolution manometry and magnetic resonance defecography) provide a refined understanding of anorectal dysfunctions and identify phenotypes in defecatory disorders and fecal incontinence. Conservative approaches, including biofeedback therapy, are the mainstay for managing these disorders; new minimally invasive approaches may benefit a subset of patients with fecal incontinence, but more controlled studies are needed. This mini-review highlights advances, current concepts, and controversies in the area.

Keywords: Anorectal Manometry; Dyssynergic Defecation; Fecal Incontinence; Biofeedback Therapy.

**Advances in Basic Sciences**

**Anatomy and Physiology of the Anal Sphincters**

Although the anal sphincters are vital for maintaining continence and defecation,¹ our understanding of their neurophysiology lags behind the rest of the enteric nervous system. Recent studies have shed new light on the structure of the internal anal sphincter (IAS) and its tone and innervation. In monkeys, the IAS is thicker than the rectum and organized into "mini-bundles," which contain nerves and unique stellate-shaped interstitial cells of Cajal (ICC).² Nerves and ICC are not closely associated with one another. These morphological features suggest that intramuscular ICC in the IAS may serve as pacemaker cells rather than as mediators of neuromuscular transmission.

Befitting a sphincter, the IAS has higher resting tone than the rectum. Basal tone in the human IAS is maintained by calcium entry via L-type calcium channels³–⁵ and RhoA–RhoA kinase (ROCK), which enhances myofilament sensitivity to calcium.⁶ Fascinating data suggest that microRNAs can modulate the RhoA/ROCK pathway and thereby regulate tone in the rat IAS.⁷ For example, microRNA 139-5b repressed the RhoA/ROCK pathway and reduced tone while the corresponding anti-miR had the opposite effect. Whether micro-RNAs alter anal sphincter function in humans is unclear.

Sympathetic nerves provide the primary excitatory input to the anal sphincter in monkeys and humans but not in mice or rabbits.³ Perhaps these species-dependent differences explain why some species (eg, mouse, rabbits) that defecate more frequently have less sympathetic excitatory innervation than others (ie, monkeys) that defecate less frequently.

Therapeutic options for restoring anal sphincter function in patients with sphincter injury are limited. Surgical repair of sphincter defects restores continence in the short term but not in the long term.⁸ Recently, bioengineered IAS strips were created by coculturing human IAS circular smooth muscle strips with mouse fetal enteric neurons, and these appear to retain their integrity and functional characteristics after implantation into mice.⁹,¹⁰ Translating these advances to patients with damaged sphincters will require isolation and culture of autologous human enteric neuronal progenitor populations to minimize immune reactions and techniques for implantation that do not disrupt other muscles.

**Defecatory Disorders**

In patients with chronic constipation that is unresponsive to laxatives, anorectal testing is necessary to identify defecatory disorder(s) (DDs).²,¹⁵ As detailed in the following text, DDs may result from disordered function (eg, rectoanal dyssynergia) or rectal structural disturbances; these may coexist. DDs are common in the community, with a prevalence of 22 (vs 5.8 for Crohn’s disease) per 100,000 person-years.¹⁶ Although DDs have been mostly described in patients without underlying colorectal disease, recent studies also report DDs in constipated patients with inflammatory bowel disease, either with native anatomy or ileal pouchitis with anal anastomosis.¹⁷,¹⁸

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**Abbreviations used in this paper:** DD, defecatory disorder; DRE, digital rectal examination; FI, fecal incontinence; IAS, internal anal sphincter; ICC, interstitial cells of Cajal; MRI, magnetic resonance imaging; ROCK, RhoA–RhoA kinase; SNS, sacral nerve stimulation.

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Clinical Features

Physicians consider certain symptoms (eg, excessive straining and anal digitation) to be suggestive of disordered defecation. However, questionnaire assessments cannot distinguish DDs from other causes of chronic constipation. Whether an interview, which provides an opportunity to ask follow-on questions, can discriminate between DDs and other causes of chronic constipation is unclear. A recent abstract from Italy suggests that an affirmative response to a different question (ie, asking patients if they mostly squeezed the anus to defecate) was 82% sensitive and 86% specific for identifying DDs.

Few physicians meticulously evaluate anal sphincter tone and pelvic floor motion by a digital rectal examination (DRE), even in patients with chronic constipation. This is a significant lacuna because a DRE is reasonably accurate relative to manometry for assessing anal resting tone and squeeze function and for identifying dyssynergia. A DRE was 75% sensitive and 87% specific versus dyssynergia documented by manometry but 80% sensitive and 56% specific versus the rectal balloon expulsion test. The lower specificity compared with a rectal balloon expulsion test may reflect the inability of some people with normal pelvic floor function to simulate the process of defecation during a DRE. Hence, a normal DRE is more useful than an abnormal examination in patients with chronic constipation. Controlled studies evaluating the utility of a DRE alone and integrated with symptoms for identifying DDs are necessary.

Pathophysiology

Visual inspection of anorectal manometry tracings suggests that impaired rectal evacuation can be attributed to weak rectal propulsive forces or increased anal outlet resistance (ie, inadequate relaxation and/or paradoxical contraction of the anal sphincter). A principal components analysis of anorectal pressures in 295 constipated patients and 62 controls identified these 2 patterns and a third (ie, hybrid) pattern characterized by low rectal and high anal pressures during evacuation (Figure 1). Because these principal components were, by design, uncorrelated to each other, they reflect distinct underlying pathophysiological mechanisms. The concept that dyssynergia results from “maladaptive learning” of sphincter contraction, perhaps stemming from neglecting the call to defecate in childhood, is plausible. For example, one-third of children with childhood constipation continue to have severe symptoms beyond puberty. However, this framework emphasizes “volitional disturbances” but does not integrate visceral dysfunctions (eg, rectal hyposensitivity, increased anal resting pressure [anal hypertension], perineal laxity

<table>
<thead>
<tr>
<th>During evacuation</th>
<th>Rectal evacuation</th>
<th>Anal relaxation versus rest</th>
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<tbody>
<tr>
<td>High anal</td>
<td>Normal</td>
<td>No</td>
</tr>
<tr>
<td>Low rectal</td>
<td>Low</td>
<td>Yes</td>
</tr>
<tr>
<td>Hybrid</td>
<td>Low</td>
<td>No</td>
</tr>
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</table>

Figure 1. Representative examples of anorectal pressure phenotypes identified by high-resolution manometry in DDs. Pressures at rest and during squeeze and evacuation were recorded by 12 sensors (2 in the rectal balloon and 10 in the anal canal) and are depicted in color; the numbers reflect the distance of the sensors from the anal verge. High anal, low rectal, and hybrid phenotypes are defined by anal, rectal, and combined rectoanal dysfunction, respectively. During evacuation: (1) anal relaxation was normal in the rectal phenotype but absent in the high anal and hybrid patterns and anal resting pressure was also higher in the anal phenotype and (2) rectal (balloon) pressure increased, as evidenced by the change in color from blue to green in the rectal balloon, in the high anal phenotype only.
manifested by excessive perineal descent, and delayed colonic transit into the pathophysiology of DD. Some features (eg, rectal hyposensitivity and delayed transit) improve after successful biofeedback treatment, suggesting they are consequences rather than causes of obstructed defecation. Studies with high-definition manometry confirm that the puborectalis closes the cranial part of the anal canal, thereby preserving fecal continence.

Rectal distention induces involuntary relaxation of the IAS. At a higher volume, rectal distention is perceived and subsequently evokes contraction of the external sphincter. Likewise, distention induces a sensorimotor response that manometrically seems to result from puborectalis contraction and coincides with the desire to defecate. These observations are reminiscent of prior studies suggesting that the desire to defecate is mediated not by rectal distention but by the rectal contractile response to distention. Further studies are necessary to ascertain if the sensorimotor response is a cause or consequence of the desire to defecate.

**Diagnostic Testing**

Anorectal manometry and a rectal balloon expulsion test, followed by barium or magnetic resonance defecography if necessary, are recommended in constipated patients when diet and lifestyle modification and empiric laxative therapy have failed (Supplemental Table 1). Clinicians can probably diagnose DDs with reasonable confidence in patients who have typical symptoms, a confirmatory DRE, and an abnormal balloon expulsion test result.

Until the advent of high-resolution manometry catheters 5 years ago, anal manometry was performed with water-perfused or solid-state sensors. High-resolution catheters provide a single pressure, averaged around the circumference, at 6-mm intervals and straddle the entire length of the anal canal, obviating the need for a station pull-through maneuver. High-definition catheters use 256 circumferentially distributed pressure sensors that provide greater definition of sphincter morphology and defects. Because they have more sensors, these new systems provide better resolution relative to traditional (water-perfused or solid-state) systems. However, only the highest pressure recorded by the catheter at any instant is used to calculate average or maximum resting or squeeze pressure, which is why normal values are higher than for traditional systems. Data from all sensors are used to assess sphincter symmetry by high-definition manometry.

Recent studies have highlighted 2 challenges with diagnosing DDs. First, based on physical principles, the rectoanal gradient (ie, the ratio of or difference between rectal and anal pressure) during evacuation should normally be positive in people without dyssynergia. Consistent with these principles, this gradient is low in dyssynergia and increases after biofeedback therapy, reflecting improved rectoanal coordination. However, there is considerable overlap in this gradient between asymptomatic subjects, patients with dyssynergia, and patients with chronic pelvic pain without constipation. Indeed, with high-resolution manometry, this gradient was negative (ie, anal > rectal pressure) in a majority of asymptomatic women. Perhaps these findings are partly explained by the challenge of replicating the process of defecation in the left lateral position with an empty rectum. Coaching patients while they perform maneuvers might be useful; in one study, coaching changed the diagnosis based on manometry from “pathologic” to “normal” values in 14 of 31 patients with incontinence and 12 of 39 patients with DDs.

![Figure 2. Algorithm for managing DDs. IBS, irritable bowel syndrome; p.r.n., as needed. Reproduced with permission from Bharucha et al.](image-url)
Second, there is limited agreement among tests used to diagnose DDs and there is no single criterion standard for diagnosing dyssynergia. For example, 51% of 125 patients with chronic constipation had dyssynergia by defecography; of those, only approximately 50% had an abnormal balloon expulsion test result and only 50% had abnormal pelvic floor relaxation by surface electromyography. A meta-analysis of 79 studies with 7591 patients who had chronic constipation reported that the prevalence of findings suggestive of abnormal defecation ranged from 14.9% (95% confidence interval, 7.9–26.3) for absent opening of the anorectal angle on defecography to 47.7% (95% confidence interval, 39.5–56.1) for a dysynergic pattern with manometry and 52.9% (95% confidence interval, 44.3–61.3) for a dysynergic pattern by ultrasonography. Taken together, these observations do not undermine the entity of dyssynergia but rather emphasize the phenotypic heterogeneity of DDs, underscore the influence of the nature of rectal contents as also the sensation evoked by rectal distention on defecation, and suggest there is considerable scope for refining anorectal tests for diagnosing DDs. Currently, high-resolution manometry is primarily helpful to stratify patients into subpopulations and is not more useful than standard anorectal manometric techniques for managing DDs.

**Management**

Dyssynergic defecation should be managed by biofeedback therapy (Figure 2). Successful protocols have typically used 5 to 6 training sessions lasting 30 to 60 minutes each and spaced 2 weeks apart. The goals of therapy are to (1) educate patients about disordered defecation, (2) coordinate increased intra-abdominal pressure with pelvic floor muscle relaxation during evacuation, and (3) practice simulated defecation with a balloon, aided by a therapist. Some centers also provide sensory retraining for restoring the sensation of rectal filling.

Although these studies show the efficacy of biofeedback therapy in tertiary centers, more studies regarding its effectiveness in clinical practice are necessary. The skill and experience of the therapist are critical factors influencing the response to biofeedback therapy; this expertise is not widely available. In a randomized controlled trial, home biofeedback therapy was equally effective and cheaper than office-based biofeedback therapy. The anorectal factors that predict the response to biofeedback therapy, the ingredients of biofeedback therapy that are critical for its success, and the mechanism(s) by which biofeedback therapy improves symptoms and pathophysiology in dyssynergia are unclear. The latency of cortical evoked potentials in response to rectal or anal stimulation is prolonged and declines after biofeedback therapy in patients with dyssynergic defecation, suggesting improved corticofunctional action. Biofeedback therapy was also effective in approximately 60% of patients with inflammatory bowel disease and DDs.

**Other Therapeutic Approaches**

Although sacral nerve stimulation (SNS) has been used to treat chronic constipation, the data are mostly uncontrolled and the response to treatment is inconsistent. Two uncontrolled studies including 24 patients with DDs reported improved outcomes after SNS for constipation. The mechanisms by which SNS might improve symptoms in DD are unclear. A small study in patients with DD and rectal hyposensitivity reported that rectal sensory thresholds were lower when SNS was on than off, suggesting improved sensation. SNS may also modulate colonic motility; suprasensory but not subsensory stimulation increased colonic propagating sequences. Long-term controlled trials with assessment of subjective and objective features are necessary to clarify the role of SNS in DDs, particularly in patients who have failed to respond to biofeedback therapy.

The recent American Gastroenterological Association technical review on constipation concluded there was insufficient evidence to recommend the stapled transanal resection procedure or injection of botulinum toxin into the pelvic floor muscles for managing chronic constipation. Likewise, a randomized controlled trial observed that botulinum toxin was not useful for levator ani syndrome; biofeedback therapy is effective for this disorder.

**Fecal Incontinence**

Fecal incontinence (FI) refers to the recurrent uncontrolled passage of feces not related to a temporary diarrheal illness (eg, acute gastroenteritis). In noninstitutionalized adults, the prevalence is 2.2% to 15.3%. FI substantially impairs quality of life. Risk factors include age, diarrhea, urgency to defecate, obstetric injury, and a variety of medical conditions.

**Etiology and Pathophysiology**

Initial studies with endoanal ultrasonography uncovered a high prevalence (ie, up to 30%) of obstetric anal sphincter injury in women with FI, prompting the concept that obstetric trauma is an important risk factor in women. After external anal sphincter myotomy in a rabbit model, there was progressive fibrosis, impaired muscle length-tension relationships, and disorganized muscle fiber distribution, which persisted up to 12 weeks after injury. These findings suggest that healing after sphincter injury may not improve function.

On average, FI begins in the fifth to seventh decade of life, which suggests that factors in addition to obstetric anal injury play a role in FI. Indeed, data from community-based studies show that diarrhea and other conditions (eg, cholecystectomy, smoking, and increased body mass index) rather than a complicated obstetric history (eg, use of forceps) are risk factors in community women with late-onset FI. Of interest, the risk of FI in current smokers is comparable to that of patients with irritable bowel syndrome or a cholecystectomy. Moreover, smoking was the only risk factor for external sphincter atrophy by magnetic resonance imaging (MRI).

Nerve injury also contributes to FI. Pudendal nerve terminal motor latencies are not recommended for identifying pudendal nerve injury. Hence, needle electromyography is
the only established technique for identifying anal neurogenic injury. A recent controlled study observed neurogenic or muscle injury in 55% of a selected cohort of 20 women with FI, which is comparable to that reported previously. Moreover, even in asymptomatic nulliparous women, increased age was associated with neurogenic injury, which partly explained weak squeeze pressures.

Using techniques that are widely utilized to assess somatic pathways, evoked potentials elicited by peripheral or central, electrical, or magnetic stimulation can be used to assess the neural pathways mediating anorectal functions. Depending on the site of stimulation and recording, afferent and efferent pathways can be evaluated. Confirming proof of concept, motor evoked potentials after anal and rectal stimulation were prolonged in patients with spinal cord injury and bowel dysfunction and also in FI. These techniques may enhance our understanding of anorectal dysfunctions, in particular the mechanisms of impaired voluntary relaxation in DDs or unexplained anal weakness in FI. However, further validation is necessary. For example, the precise cortical target of transcranial magnetic stimulation is unclear. Spinal magnetic stimulation usually activates spinal nerves at the neuroforamina but not in the spinal canal (ie, the cauda equina). With supramaximal stimulation, which is now possible with a novel coil, the magnetic augmented translumbosacral stimulation coil, the most proximal part of the cauda equina can also be reliably activated. Hence, it should be possible to measure the cauda equina and corticonucous motor conduction times.

In addition to anal weakness, rectal distention by a barostat shows increased rectal stiffness and reduced rectal capacity, which is associated with the symptom of rectal urgency and with increased rectal sensitivity, in a subset of women with FI (Supplemental Table 1). A barostat measures rectal volumes and pressures but not diameter. Rectal diameter, hence rectal stress-strain relationships (or stiffness), can be directly measured by integrating MRI with rectal balloon distention. These studies confirmed increased rectal stiffness in FI.

Diagnostic Testing
Endoscopy, and when microscopic colitis is considered with colonic biopsies, should be considered. A rigorous trial of conservative measures is justified before diagnostic testing, particularly in older patients, those with mild symptoms, and those with bowel disturbances. Anorectal manometry, rectal sensation, and rectal balloon expulsion are useful initial tests. In selected patients with reduced anal pressures, anal imaging and/or anal sphincter electromyography are useful (Supplemental Table 1).

Management
Conservative therapy. Three key studies highlight the role of conservative therapy and placebo responses in FI. Norton et al observed that symptoms improved in approximately 54% of patients with FI who were instructed in diet, fluids, techniques to improve evacuation, a bowel training program, titration of antidiarrheal medication if necessary, and practical management in nine 40- to 60-minute sessions over 3 to 6 months by a specialist nurse. In another randomized controlled trial of 108 patients, 22% of patients responded to conservative therapy for 4 weeks. Among nonresponders to conservative therapy, electromyography-assisted biofeedback was superior to pelvic floor exercises alone. More recently, a >50% reduction in the number of days and episodes of FI was reported by 36% and 32%, respectively, of women with FI who received placebo alone for 4 weeks. However, these conservative therapies were administered by specialized therapists and not busy practicing physicians. Nonetheless, conservative therapies will benefit approximately 25% of patients and should be tried first. These conservative measures include reduced intake of foods (eg, poorly absorbed carbohydrates such as fructose, sorbitol, and others, caffeine) that can cause or aggravate diarrhea and/or rectal urgency, urge suppression techniques, and antidiarrheal agents (eg, loperamide).

Clonidine increases rectal compliance, reduces rectal sensation, and improves symptoms in patients with diarrhea-predominant irritable bowel syndrome. In an uncontrolled study, clonidine improved continence. In a controlled study, clonidine reduced diarrhea and tended to reduce the number of days with FI, but the overall effects were not significant.

Biofeedback therapy. For those failing to respond to medical therapy, biofeedback therapy designed to improve anal sphincter and puborectalis tone, strength and endurance, and anorectal coordination remains the mainstay. Randomized controlled trials show that biofeedback therapy is superior to Kegel exercises. A small study did not identify significant differences between sustained squeeze maneuvers, which is the standard approach, and a combination of rapid and sustained squeeze maneuvers.

Minimally invasive/surgical options. With the widespread recognition that success rates decline with time after the procedure, anal sphincteroplasty is primarily reserved for women with postpartum FI; for example, only 21% were continent at 40 months in one study. SNS and anal submucosal injection of a “bulking agent” (dextranomer in stabilized hyaluronic acid [NASHA/Dx]) are now approved by the Food and Drug Administration for the treatment of FI.

For both SNS and NASHA studies, success was defined by a ≥50% reduction in the number of incontinent episodes per week. SNS is a staged procedure; that is, when symptoms respond to temporary stimulation for 3 weeks, the device is implanted subcutaneously (ie, permanent stimulation). In the pivotal US multicenter trial, 90% of 120 patients proceeded from temporary to permanent stimulation. Five-year follow-up was available in 76 of 120 patients (63%); 36% reported complete continence, and 89% were deemed a therapeutic success.

However, most studies with SNS were uncontrolled. In one crossover study of 34 patients, the number of episodes of FI declined by 90% during stimulation versus 76% without stimulation. The discrepancy between symptom improvement and relatively minor effects on anorectal functions is puzzling. Recent data suggest that SNS but not
Sham stimulation increased the frequency of retrograde propagated sequences throughout the colon. Similar to antidiarrheal agents, these effects may be anticipated to delay colonic transit.\textsuperscript{78} In contrast, SNS increased colonic propagating sequences in constipation.\textsuperscript{47} Perhaps differences in baseline colonic motor activity partly explain why SNS may have different effects on colonic motility in constipation and FI, but more work is needed.

In the pivotal trial with 206 patients, the 6-month response as defined in the preceding text was higher for NASHA/Dx (52\%) than sham injections (31\%),\textsuperscript{79} yielding a number needed to treat of 4.4. Eighty percent of patients in the active treatment group required a second injection 1 month after the initial procedure. With 2 exceptions (ie, rectal abscess, prostatic abscess), most adverse events were minor. Treatment did not significantly improve quality of life for patients with FI; data for complete continence and effects on anorectal physiology or imaging were not provided.\textsuperscript{80,81} Another controlled trial with 126 patients reported significant improvement in FI symptom severity and quality of life in patients who were randomized to pelvic floor biofeedback therapy and separately to treatment with NASHA/Dx\textsuperscript{82}; the efficacy of these approaches was not different. Biofeedback therapy increased anal squeeze pressure, but NASHA/Dx did not increase anal resting or squeeze pressure. Hence, the magnitude of benefit, mechanisms of action, long-term effects, and factors that predict response to therapy merit further study.

**Summary**

Significant advances in basic science studies and development of newer diagnostic techniques in humans have advanced our understanding of the multifaceted dysfunctions that contribute to pelvic floor disorders. DDs are a common cause of chronic constipation. Although symptoms and a careful DRE are very useful for identifying DDs, anorectal tests are necessary to confirm the diagnosis. In most patients, anorectal manometry and a rectal balloon expulsion test suffice. In some patients, defecography with either barium or MRI is necessary to confirm or exclude the diagnosis. Pelvic floor retraining by biofeedback therapy represents the mainstay for managing DDs.

FI is a common and often distressing symptom. Bowel dysfunctions and anorectal sensorimotor dysfunctions are
the key pathophysiological mechanisms. Management relies on conservative measures, pelvic floor retraining by biofeedback therapy in patients who do not respond to conservative measures, and SNS or other surgical approaches for patients who are refractory to medical therapy.

In DDs, the scientific and clinical priorities are to refine diagnostic tests, our understanding of phenotypes in these disorders, and the impact of these phenotypes on therapy. Moreover, there is an urgent need to increase access and coverage for pelvic floor retraining by biofeedback therapy and to develop alternative approaches for these disorders. In FI, the emphasis is on identifying the symptom and the likely cause, followed by targeted therapy. Future studies should also apply advances from basic science to humans, refine our understanding of phenotypes in these disorders, develop new approaches for managing these disorders based on our understanding of the underlying mechanisms, and compare the efficacy of various therapeutic approaches in rigorously controlled clinical trials.

Supplementary Material

Note: To access the supplementary material accompanying this article, visit the online version of Gastroenterology at www.gastrojournal.org, and at http://dx.doi.org/10.1053/j.gastro.2013.10.062.

References

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cation can be diagnosed by questionnaire and physical examination (abstr). Gastroenterology 2013;144:S-366.


## Supplementary Material

### Supplemental Table 1. Summary of Anorectal Tests

<table>
<thead>
<tr>
<th>Anorectal parameter (method)</th>
<th>Methodological issues</th>
<th>Clinical utility</th>
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</thead>
<tbody>
<tr>
<td>Internal sphincter function (anal resting pressure)</td>
<td>Manometry uses traditional (water-perfused or solid-state) or newer (high-resolution and high-definition) techniques. Methods for anal manometry and definitions of parameters (e.g., maximum squeeze pressure) are poorly standardized. Normal values are technique dependent, lower in women than in men, and decline with age, especially resting pressure. Age- and sex-adjusted normal values are not universally used to interpret patient data in clinical practice.</td>
<td>Women with FI have reduced anal resting (~40%) or squeeze (~80%) pressures. Rectoanal gradient during evacuation is used for identifying DD, but its utility is limited by overlap between asymptomatic subjects and patients with DD. Some patients with DD have anal hypertension (high resting pressure).</td>
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<tr>
<td>External sphincter function (anal squeeze pressure)</td>
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<tr>
<td>Rectoanal coordination (rectoanal pressure gradient)</td>
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<tr>
<td>Anal sphincter structure (ultrasonography or MRI)</td>
<td>Endoanal probes provide superior visualization of the anal sphincters; transperineal ultrasonography has also been described.</td>
<td>Ultrasoundography and MRI are probably equivalent for visualizing the internal sphincter; MRI is better for identifying external sphincter and puborectalis atrophy but more expensive. Anal sphincter defects may be clinically unrecognized and amenable to surgical repair.</td>
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<tr>
<td>Puborectalis structure and function (manometry and MRI)</td>
<td>High-definition manometry uses pressures exerted on the posterior aspect of the upper anal canal to assess puborectalis function. MRI can visualize puborectalis structure and motion.</td>
<td>25% of women with FI but no controls had puborectalis atrophy on MRI.</td>
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<td>Rectal evacuation (rectal balloon expulsion and proctography with fluoroscopy or MRI)</td>
<td>Generally, rectal balloon expulsion time is measured with a balloon inflated with 50 mL of water. Normally, subjects take less than 1 minute to expel a balloon; patients with a DD require longer. In women &gt;50 years, the upper limit of normal for balloon expulsion time is 15 seconds. Alternatively, the balloon is inflated until patients report urgency. Another approach is to assess the traction required to expel a balloon in the left lateral position. Proctography is described in the following text.</td>
<td>Abnormal rectal balloon expulsion is a useful, highly sensitive and specific, first-line test for diagnosing DDs in clinical practice. The positive and negative predictive value of this test against DDs documented by barium defecography was 64% and 97%, respectively; in this study, the balloon was inflated until patients experienced the desire to defecate.</td>
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## Supplemental Table 1. Continued

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<th>Anorectal parameter (method)</th>
<th>Methodological issues</th>
<th>Clinical utility</th>
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<tr>
<td><strong>Anorectal and pelvic floor motion (proctography with fluoroscopy or MRI)</strong></td>
<td>After adding barium paste (defecography) or ultrasound gel (MRI) to the rectum, images are acquired during pelvic floor contraction and rectal evacuation. Barium is added to the bladder, small intestine, and vagina to visualize these organs by defecography. Advantages of MRI include lack of radiation, ability to perform multiplanar imaging, and better visualization of bony landmarks, the pelvic floor, and other organs; hence, measurements are more reproducible. Most magnets only permit supine imaging. Supine and seated MRI are generally comparable for identifying pelvic organ prolapse. Rectal intussusception is more frequently documented by seated than by supine MRI.</td>
<td>This is useful when anorectal manometry and rectal balloon expulsion are equivocal in patients with suspected DD and in patients with pelvic organ prolapse. Patients with DD may have impaired rectal evacuation, pelvic organ prolapse (rectoceles), and normal, reduced, or increased perineal descent.</td>
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<td><strong>Anal sphincter innervation (needle or surface EMG)</strong></td>
<td>Needle EMG requires considerable technical expertise and is available at selected centers only. Neurogenic EMG can identify not only neurogenic but also muscle injury (e.g., due to local trauma). Surface EMG provides a global index of muscle activity.</td>
<td>Needle EMG may be useful in a small proportion of patients with FI (e.g., with unexplained anal weakness), particularly when proximal neurogenic injury is suspected. In selected cohorts, up to 50% of patients with FI have neurogenic or muscle injury. Surface EMG cannot identify neurogenic injury. Rather, it can assess anal and pelvic floor activation and relaxation, diagnose DDs, and provide biofeedback therapy.</td>
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<td><strong>Rectal compliance and sensation (manual or barostat-driven distention of a rectal balloon)</strong></td>
<td>Manual distention is typically performed at the same time as anorectal manometry, typically uses a latex balloon, and measures volume thresholds. Normal values are technique dependent and vary across laboratories. Barostat distention uses a polyethylene balloon and also measures pressure thresholds. In contrast to a latex balloon, the polyethylene balloon is infinitely compliant over the range of distending pressures; hence, rectal compliance and sensation are most reliably measured by a barostat.</td>
<td>Rectal sensation may be reduced in DDs and reduced or increased in FI. Whether reduced sensation causes or is a consequence of DDs is unknown. Increased sensation is associated with reduced rectal capacity and with the symptom of urgency. Modulation of rectal sensory disturbances by biofeedback therapy can improve fecal continence.</td>
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EMG, electromyography.
Bharucha AE, Rao SS. *An update on anorectal disorders for gastroenterologists.* Gastroenterology 2014;146:37-45

1. Risk factors for fecal incontinence include
   a. Smoking
   b. cholecystectomy
   c. constipation
   d. diarrhea
   e. decreased BMI

True or False

2. In most cases, a careful history is unable to differentiate defecatory disorders from other causes of chronic constipation

3. Stimulant laxatives are the mainstay of therapy for dyssynergic defecation

4. In humans, the primary excitatory input to the anal sphincter is provided by parasympathetic nerves

5. Botulinum toxin injection in the anal sphincter to treat chronic constipation is not recommended

6. Surgical correction of anal sphincter injury usually does not provide long-term continence

7. During defecation, anal sphincter pressure that is higher than rectal pressure is diagnostic for dyssynergia and found only in symptomatic patients.

8. A smoking history is a risk factor for external anal sphincter atrophy

9. FDA approved therapies for fecal incontinence include sacral nerve stimulation and injection of hyaluronic acid, data demonstrating efficacy for either technique is underwhelming