Published in final edited form as:

Gastroenterology. 2014 January ; 146(1): 37–45.e2. doi:10.1053/j.gastro.2013.10.062.

An Update on Anorectal Disorders for Gastroenterologists

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Abstract

Gastroenterologists frequently encounter pelvic floor disorders, which affect 10–15% 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 (e.g., 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 these advances, current concepts and controversies in the area.

Introduction

Pelvic floor disorders affect 10–15% of the population ^{1, 2}. This mini-review is focused on recent advances in basic science and clinical research in common conditions encountered by practicing gastroenterologists, but is not a comprehensive review of these topics ^{3, 4}.

Advances in Basic Sciences

Anatomy and Physiology of the Anal Sphincters

While the anal sphincters are vital for maintaining continence and defecation ⁵, 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), 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 ⁶. 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.

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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 channels⁷⁻⁹ and by RhoA-ROCK (RhoA kinase), which enhances myofilament sensitivity to calcium¹⁰. Fascinating data suggest that micro RNAs can modulate the RhoA/ROCK pathway and thereby regulate tone in the rat IAS¹¹. For example, miRNA 139-5b repressed the RhoA/ROCK pathway and reduced tone while the corresponding anti-miR had the opposite effect. Whether miRNAs 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 rabbits⁸. 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 but not long term¹². Recently, bioengineered IAS strips were created by co-culturing 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^{13, 14}. 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 unresponsive to laxatives, anorectal testing is necessary to identify defecatory disorder(s) (DD)^{2, 15}. As detailed below, DD may result from disordered function (e.g., rectoanal dyssynergia) or rectal structural disturbances; these may coexist. Defecatory disorders are common in the community with a prevalence of 22 [versus 5.8 for Crohn's disease] per 100,000 person years¹⁶. While DD have been mostly described in patients without underlying colorectal disease, recent studies also report DD in constipated patients with inflammatory bowel disease, either with native anatomy or ileal pouchitis with anal anastomosis^{17, 18}.

Clinical Features

Physicians consider certain symptoms (e.g., excessive straining and anal digitation) as suggestive of disordered defecation. However, questionnaire assessments cannot distinguish DD from other causes of chronic constipation^{19, 20}. Whether an interview, which provides an opportunity to ask follow-on questions, can discriminate between DD and other causes of chronic constipation, is unclear. A recent abstract from Italy suggests that an affirmative response to a different question (i.e., asking patients if they mostly squeezed the anus to defecate) was 82% sensitive and 86% specific for identifying DD²¹.

Few physicians meticulously evaluate anal sphincter tone and pelvic floor motion by a digital rectal examination (DRE), even in patients with chronic constipation²². This is a significant lacuna since a DRE is reasonably accurate relative to manometry for assessing anal resting tone and squeeze function and for identifying dyssynergia^{23, 24}. Thus, a DRE was 75% sensitive and 87% specific versus dyssynergia documented by manometry¹⁸ but 80% sensitive and 56% specific versus the rectal balloon expulsion test. The lower specificity compared to a rectal balloon expulsion test may perhaps 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 exam in patients with chronic constipation. Controlled studies evaluating the utility of a DRE alone and integrated with symptoms for identifying DD 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 (i.e., inadequate relaxation and/or paradoxical contraction of the anal sphincter)²⁵. A principal components (PC) analysis of anorectal pressures in 295 constipated patients and 62 controls identified these 2 patterns and a third (i.e. hybrid) pattern characterized by low rectal and high anal pressures during evacuation¹⁹ (Figure 1). Since these PCs 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 puberty²⁶. However, this framework emphasizes “volitional disturbances” but does not integrate visceral dysfunctions (e.g., rectal hyposensitivity²⁷, increased anal resting pressure [anal hypertension], perineal laxity manifested by excessive perineal descent²⁸, and delayed colonic transit²⁹) into the pathophysiology of DD. Some features (e.g., rectal hyposensitivity and delayed transit) improve after successful biofeedback treatment suggesting they are consequences rather than causes of obstructed defecation³⁰. Studies with high definition manometry confirm that the puborectalis closes the cranial part of the anal canal, thereby preserving fecal continence³⁶.

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 sensori-motor response, which manometrically, seems to result from puborectalis contraction, and coincides with the desire to defecate³¹. These observations are reminiscent of prior studies which suggest that the desire to defecate is mediated not by rectal distention but by the rectal contractile response to distention³². Further studies are necessary to ascertain if sensori-motor 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 MR defecography if necessary are recommended in constipated patients when diet and lifestyle modification and empiric laxative therapy have failed^{2, 15} (Table 1). Clinicians can probably diagnose DD with reasonable confidence in patients who have typical symptoms, a confirmatory DRE, and an abnormal balloon expulsion test.

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 maneuver³³. High definition catheters use 256 circumferentially distributed pressure sensors that provide greater definition of sphincter morphology and defects³⁴. 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 systems³³. Data from all sensors is used to assess sphincter symmetry by high definition manometry.

Recent studies have highlighted two challenges with diagnosing DD. First, based on physical principles, the rectoanal gradient (i.e., ratio of or difference between rectal and anal pressure) during evacuation should normally be positive in people without dyssynergia³³. Consistent with these principles, this gradient is low in dyssynergia and increases after

biofeedback therapy³⁵, reflecting improved rectoanal coordination. However, there is considerable overlap in this gradient between asymptomatic subjects, patients with dyssynergia and chronic pelvic pain without constipation^{35,36}. Indeed, with high resolution manometry, this gradient was negative [i.e., anal > rectal pressure] in a majority of asymptomatic women^{19,33}. 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 14/31 patients with incontinence and 12/39 with DD³⁷.

Second, there is limited agreement among tests used to diagnose DD and there is no single criterion standard for diagnosing dyssynergia. For example, 51% of 125 patients with chronic constipation had dyssynergia by defecography³⁸; of those, only approximately 50% had an abnormal balloon expulsion test 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% CI 7.9–26.3) for absent opening of the anorectal angle on defecography to 47.7% (95% CI 39.5–56.1) for a dyssynergic pattern with manometry, and 52.9% (95% CI 44.3–61.3) for a dyssynergic pattern by ultrasound³⁹. Taken together, these observations do not undermine the entity of dyssynergia but rather emphasize the phenotypic heterogeneity of DD²⁸, underscore the influence of the nature of rectal contents as also the sensation evoked by rectal distention on defecation⁴⁰, and suggest there is considerable scope for refining anorectal tests for diagnosing DD. Currently, high resolution manometry is primarily helpful to stratify patients into subpopulations, and is not more useful than standard anorectal manometric techniques for managing DD.

Management

Dyssynergic defecation should be managed by biofeedback therapy⁴¹. Successful protocols have typically employed 5–6 training sessions lasting 30–60 minutes each and spaced two 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.

While these studies demonstrate 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 therapy⁴². The anorectal factors that predict the response to biofeedback therapy, the ingredients of biofeedback therapy which 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 corticoanal function⁴³. Biofeedback therapy was also effective in approximately 60% of patients with inflammatory bowel disease and DD^{17,18}.

Other therapeutic approaches

While sacral nerve stimulation (SNS) has been used to treat chronic constipation, the data are mostly uncontrolled, and the response to treatment is inconsistent⁴⁴. Two uncontrolled studies including 24 patients with DD reported improved outcomes after SNS for constipation^{45,46}. 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 sensation⁴⁶. SNS may also modulate colonic motility; suprasensory but not subsensory stimulation increased colonic propagating sequences⁴⁷. Long term, controlled trials with assessment of subjective and objective features are necessary to clarify the role of SNS in DD, particularly in patients who have failed biofeedback therapy.

The recent AGA 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 constipation². Likewise, a randomized controlled trial observed that botulinum toxin was not useful for levator ani syndrome; biofeedback therapy is effective for this disorder^{36, 48}.

Fecal Incontinence

Fecal incontinence (FI) refers to the recurrent uncontrolled passage of feces not related to a temporary diarrheal illness (eg, acute gastroenteritis). In non-institutionalized adults, the prevalence is 2.2% to 15.3%. FI substantially impairs quality of life^{1, 49, 50}. Risk factors include age, diarrhea, urgency to defecate, obstetric injury and a variety of medical conditions^{23–26}.

Etiology and Pathophysiology

Initial studies with endoanal ultrasound uncovered a high prevalence (i.e., up to 30%) of obstetric anal sphincter injury in women with FI, prompting the concept that obstetric trauma is an important risk factor in women⁵¹. 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 injury⁵². These findings suggest that healing after sphincter injury may not improve function.

On average, FI begins in the 5–7th decade^{21,50}, which suggests that factors in addition to obstetric anal injury play a role in FI. Indeed, data from community-based studies demonstrate that diarrhea, and other conditions (e.g., cholecystectomy, smoking, and increased BMI) rather than a complicated obstetric history (e.g., forceps use) are risk factors in community women with late-onset FI^{53–56}. Of interest, the risk of FI in current smokers is comparable to patients with irritable bowel syndrome or a cholecystectomy⁵⁵. Moreover, smoking was the only risk factor for external sphincter atrophy by MRI⁵⁶.

Nerve injury also contributes to FI. Pudendal nerve terminal motor latencies are not recommended for identifying pudendal nerve injury⁵⁷. 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 previously⁵⁸. Moreover, even in asymptomatic nulliparous women, increased age was associated with neurogenic injury, which partly explained weak squeeze pressures.

Using techniques that are widely used 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^{59, 60}. Confirming proof of concept, motor evoked potentials after anal and rectal stimulation were prolonged in patients with spinal cord injury and bowel dysfunction⁶¹ and also in FI⁶². These techniques may enhance our understanding of anorectal dysfunctions, in particular the mechanisms of impaired voluntary relaxation in DD or unexplained anal weakness in FI. However, further validation is

necessary. For example, the precise cortical target of transcranial magnetic stimulation is unclear⁶³. Spinal magnetic stimulation usually activates spinal nerves at the neuroforamina but not in the spinal canal, i.e., the cauda equina⁶⁴. 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 cortico-conus motor-conduction times.

In addition to anal weakness, rectal distention by a barostat demonstrates 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 FI⁶⁵ (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 FI⁶⁶.

Diagnostic Testing

Endoscopy, and when microscopic colitis is considered with colonic biopsies, should be considered⁶⁷. A rigorous trial of conservative measures is justified before diagnostic testing, particularly in older patients, those with mild symptoms, and bowel disturbances⁶⁷. 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 EMG are useful (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 40–60-minute sessions over 3–6 months by a specialist nurse⁶⁸. In another RCT of 108 patients, 22% of patients responded to conservative therapy for 4 weeks⁶⁹. Among non-responders to conservative therapy, EMG-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 treated with placebo alone for 4 weeks⁷⁰. 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 (e.g., poorly absorbed carbohydrates such as fructose, sorbitol, and others, caffeine) that can cause or aggravate diarrhea and/or rectal urgency, urge suppression techniques, and anti-diarrheal agents [e.g., loperamide].

Clonidine increases rectal compliance and reduces rectal sensation and improves symptoms in patients with diarrhea-predominant IBS^{71,72}. In an uncontrolled study, clonidine improved continence⁷³. In a controlled study, clonidine reduced diarrhea and tended to reduce the number of days with FI, but overall effects were not significant⁷⁰.

Biofeedback Therapy—For those failing medical therapy, biofeedback therapy designed to improve anal sphincter and puborectalis tone, strength and endurance, and anorectal coordination remains the mainstay. Randomized controlled trials demonstrate that biofeedback therapy is superior to Kegel exercises⁶⁹. 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 maneuvers⁷⁴.

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 study¹². Sacral nerve stimulation (SNS) and anal submucosal injection of a “bulking agent” (dextranomer in stabilized hyaluronic acid [NASHA Dx]) are now FDA-approved for the treatment of FI.

For both studies, success was defined by a 50% reduction in the number of incontinent episodes per week. SNS is a staged procedure, i.e., when symptoms respond to temporary stimulation for 3 weeks, the device is implanted subcutaneously (i.e., “permanent stimulation”). In the pivotal US multicenter trial, 90% of 120 patients proceeded from temporary to permanent stimulation⁷⁵. Five-year follow up was available in 76 of 120 (63%) patients; 36% reported complete continence and 89% were deemed a therapeutic success⁷⁶.

However, most studies with SNS were uncontrolled. In one cross-over study of 34 patients, the number of episodes of FI declined by 90% during stimulation versus 76% without stimulation⁷⁷. The discrepancy between symptom-improvement and relatively minor effects on anorectal functions is puzzling⁷⁸. Recent data suggest that SNS but not sham stimulation increased the frequency of retrograde propagated sequences throughout the colon. Similar to anti-diarrheal agents, these effects may be anticipated to delay colonic transit⁷⁸. In contrast SNS increased colonic propagating sequences in constipation⁴⁷. Perhaps differences in baseline colonic motor activity partly explain why SNS may have different effects on colonic motility in constipation and FI but this needs more work.

In the pivotal trial with 206 patients, the 6 month response as defined above was higher for NASHA Dx (52%) than sham injections (31%)⁷⁹, yielding a NNT 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 (i.e., rectal abscess, prostatic abscess), most adverse events were minor. Treatment did not significantly improve FI quality of life; data for complete continence and effects on anorectal physiology or imaging were not provided^{80, 81}. Another controlled trial with 126 patients reported significant improvement in FI symptom severity and QOL in patients who were randomized to pelvic floor biofeedback therapy and separately to NASHA Dx⁸²; 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.

Defecatory disorders are a common cause of chronic constipation. While symptoms and a careful digital rectal examination are very useful for identifying defecatory disorders, anorectal tests are necessary to confirm the diagnosis. In most patients, anorectal manometry and a rectal balloon expulsion test suffice. In some patients, defecography, either with barium or MRI are necessary to confirm or exclude the diagnosis. Pelvic floor retraining by biofeedback therapy represents the mainstay for managing defecatory disorders.

Fecal incontinence is a common and often distressing symptom. Bowel dysfunctions and anorectal sensorimotor dysfunctions are the key pathophysiological mechanisms. Management relies upon conservative measures, pelvic floor retraining by biofeedback

therapy in patients who do not respond to conservative measures, and sacral nerve stimulation or other surgical approaches for patients who are refractory to medical therapy.

In DD, 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, and develop new approaches for managing these disorders based on our understanding of the underlying mechanisms, and to compare the efficacy of various therapeutic approaches in rigorously controlled clinical trials.

Acknowledgments

This study was supported in part by USPHS NIH Grants R01 DK78924 and by R01DK 57100-07

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Figure 1. Representative examples of anorectal pressure phenotypes identified by high resolution manometry in defecatory disorders
Pressures at rest, 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 sensors from anal verge. High anal, low rectal and hybrid phenotypes are defined by anal, rectal and combined rectoanal dysfunction respectively. During evacuation: (i) anal relaxation was normal in the rectal phenotype but absent in the “high anal” and “hybrid” patterns. Anal resting pressure was also higher in the anal phenotype, (ii) rectal (balloon) pressure increased, as evidenced by color change from blue to green in the rectal balloon, in the high anal phenotype only.

Figure 2.

Algorithm for managing defecatory disorders Reproduced with permission from Bharucha AE, Dorn SD, Lembo A, Pressman A. American Gastroenterological Association Medical Position Statement on Constipation Gastroenterology 2013; 144(1):211–17 (left panel)

Figure 3.
Algorithm for managing fecal incontinence.

Summary of Anorectal Tests

Table 1

Anorectal Parameter (Method)	Methodological Issues	Clinical Utility
<ul style="list-style-type: none"> Internal sphincter function (anal resting pressure) External sphincter function (anal squeeze pressure) Rectoanal coordination (rectoanal pressure gradient) 	<ul style="list-style-type: none"> Manometry utilizes 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 ⁷⁸. Normal values are technique-dependent, lower in women than men, and decline with age, especially resting pressure. Age- and gender- adjusted normal values are not universally used to interpret patient data in clinical practice. 	<ul style="list-style-type: none"> Women with FI have reduced anal resting (~ 40%) or squeeze (~ 80%) pressures ⁶¹. Rectoanal gradient during evacuation is used for identifying DD. However, it's utility is limited by overlap between asymptomatic subjects and patients with DD ^{17, 22, 24}. Some patients with DD have anal hypertension (high resting pressure) ^{17, 28}.
Anal sphincter structure (Ultrasound or MRI)	<ul style="list-style-type: none"> Endoanal probes provide superior visualization of the anal sphincters; transperineal US has also been described ⁷⁹. 	<ul style="list-style-type: none"> Ultrasound and MRI are probably equivalent for visualizing the internal sphincter. MRI is better for identifying external sphincter and puborectalis atrophy but more expensive ⁶¹. Anal sphincter defects may be clinically unrecognized and amenable to surgical repair.
Puborectalis structure and function (Manometry and MRI)	<ul style="list-style-type: none"> High definition manometry uses pressures exerted on the posterior aspect of the upper anal canal to assess puborectalis function ⁸⁰ MRI can visualize puborectalis structure and motion ⁶¹. 	<ul style="list-style-type: none"> 25% of women with FI but no controls had puborectalis atrophy by MRI ⁶¹.
Rectal evacuation (Rectal balloon expulsion and proctography with fluoroscopy or MRI)	<ul style="list-style-type: none"> Generally, rectal balloon expulsion time is measured with a balloon inflated to 50-ml of water ⁷⁸. Normally, subjects take less than 1 minute to expel a balloon; patients with a DD require longer. In women 50 years, the upper limit of normal for balloon expulsion time is 15 seconds ²². Alternatively, the balloon is inflated until patients report urgency ⁸¹. Another approach is to assess the traction required to expel a balloon in the left lateral position. Proctography is described below. 	<ul style="list-style-type: none"> Abnormal rectal balloon expulsion is a useful, highly sensitive and specific, first-line test for diagnosing DD in clinical practice. The positive and negative predictive value of this test against DD documented by barium defecography was 64% and 97% respectively ⁸¹; in this study the balloon was inflated until patients experienced the desire to defecate.
Anorectal and pelvic floor motion (Proctography with fluoroscopy or MRI)	<ul style="list-style-type: none"> 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, pelvic floor, and other organs; hence measurements are more reproducible. 	<ul style="list-style-type: none"> Useful when anorectal manometry and rectal balloon expulsion are equivocal in patients with suspected DD ⁶³. Also useful 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 ²⁸.

Anorectal Parameter (Method)	Methodological Issues	Clinical Utility
	<ul style="list-style-type: none"> Most magnets only permit supine imaging. Supine and seated MRI are generally comparable for identifying pelvic organ prolapse⁸². Rectal intussusception is more frequently documented by seated than by supine MRI. 	
Anal sphincter innervation (Needle or surface EMG)	<ul style="list-style-type: none"> 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. 	<ul style="list-style-type: none"> 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 injury⁵⁴. Surface EMG cannot identify neurogenic injury. Rather, it can assess anal and pelvic floor activation and relaxation, diagnose DD and provide biofeedback therapy²⁷.
Rectal compliance and sensation (Manual or barostat-driven distention of a rectal balloon)	<ul style="list-style-type: none"> Manual distention is typically performed at the same time as anorectal manometry; typically employs a latex balloon, and measures volume thresholds⁷⁸. Normal values are technique dependent and vary across laboratories⁷⁸. Barostat distention employs 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. 	<ul style="list-style-type: none"> Rectal sensation may be reduced in DD and reduced or increased in FI^{61, 83}. Whether reduced sensation causes or is a consequence of DD is unknown. Increased sensation is associated with reduced rectal capacity and with the symptom or urgency⁶¹. Modulation of rectal sensory disturbances by biofeedback therapy can improve fecal continence⁸².