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Anorectal Disorders

Satish S. C. Rao,[1] Adil E. Bharucha,[2] Emma V. Carrington,[3] Ugo Grossi,[4] Allison Malcolm,[5,6] Leila Neshatian,[7] and Jose M. Remes-Troche[8]

[1]Division of Gastroenterology and Hepatology, Augusta University, Augusta, Georgia; [2]Department of Gastroenterology and Hepatology, Mayo College of Medicine, Rochester, Minnesota; [3]Department of Colorectal Surgery, Imperial College and Imperial College National Health Service Trust, London, United Kingdom; [4]Division of Proctology and Pelviperineology, Humanitas Gavazzeni and Castelli, Bergamo, Italy; [5]Neurogastroenterology Unit, Royal North Shore Hospital, St Leonards, New South Wales, Australia; [6]Faculty of Medicine and Health, University of Sydney, Sydney, New South Wales, Australia; [7]Division of Gastroenterology and Hepatology, Stanford University, Palo Alto, California; and [8]Instituto de Investigaciones Médico-Biológicas, Universidad Veracruzana, Veracruz, México

This article defines the diagnostic criteria and reviews clinical evaluation and management of fecal incontinence, anorectal pain, dyssynergic defecation, and rectal hyposensitivity and hypersensitivity. Diagnostic evaluation includes anorectal manometry, balloon expulsion test, anal ultrasound, magnetic resonance imaging, defecography, and neurophysiology testing. Fecal incontinence is defined as recurrent uncontrolled passage of fecal material for 3 months. Management includes antidiarrheals, Kegel exercises, biofeedback therapy, dextranomer injection, surgery, sacral nerve stimulation, and translumbosacral neuromodulation therapy. Proctalgia fugax is defined as anorectal pain lasting seconds to minutes and levator ani syndrome is defined as pain lasting more than 30 minutes with puborectalis tenderness. Biofeedback and translumbosacral neuromodulation therapy may be useful. Dyssynergic defecation is defined by both symptoms of difficult defecation and objective evidence of dyssynergia. Biofeedback therapy is efficacious in dyssynergic defecation. Rectal sensory disorders are defined by both anorectal symptoms and increased (hyposensitivity) or decreased (hypersensitivity) sensory thresholds during rectal balloon distention; sensory biofeedback therapy is useful.

Keywords: Fecal Incontinence; Dyssynergic Defecation; Levator Ani Syndrome; Rectal Hyposensitivity.

Anorectal disorders are defined by symptoms and/or abnormal pathophysiology. Concepts regarding anorectal physiology continue to evolve, especially with the use of an increasing array of sophisticated tools.[1]

We discuss the epidemiology, pathophysiology, clinical assessment, diagnostic criteria, and recommendations for testing and management of common anorectal disorders (Table 1). Furthermore, unlike other disorders of gut–brain interaction (DGBI), a diagnosis of dyssynergic defecation (DD) or rectal sensory dysfunction requires symptoms and physiological testing, whereas fecal incontinence (FI) and anorectal pain require symptoms alone.

F1. Fecal Incontinence

Definition

FI is defined as the recurrent uncontrolled passage of fecal material for at least 3 months.[1] Fecal staining of underwear, but not clear mucus secretion and flatus incontinence, is included.

Epidemiology

Prevalence. The prevalence of FI is difficult to establish due to variation in sampling and definitions among studies.[2] A systematic review of 80 studies reported a pooled global prevalence of 8.0%, and 5.4% by Rome Criteria.[3] FI was more common in older (9.3%) than younger (4.9%) people, and in women (9.1%) than men (7.4%).[4]

Impact on quality of life and psychological features. FI significantly impairs quality of life (QoL).[5,6] Patients restrict social activities and report concerns about hygiene, odor, coping, fear, self-esteem, embarrassment, physical activities, sexual relationships, and the unpredictability of bowel habits.[5,6] Anxiety and depression are associated with more severe FI symptoms[7,8] and QoL impairment[5,7] and increased number of overlapping DGBI.[9] Patients with FI mixed with constipation reported lower QoL than isolated FI.[6]

Risk factors and etiology for fecal incontinence. Risk factors include age, female sex, obstetric trauma, diarrhea, cholecystectomy, diabetes, and neurologic and inflammatory diseases.[10–12] Obstetric anal sphincter injury during vaginal or instrument-assisted delivery is the leading cause of anal sphincter trauma.[13] Age-related muscle decline and emergence of other risk factors may explain why FI occurs many decades after vaginal delivery.[14] Smoking, a risk factor for external anal sphincter (EAS) atrophy, and obesity also contribute.[15,16]

Abbreviations used in this paper: **ARM, anorectal manometry; AUS, anal ultrasound; BET, balloon expulsion test; BT, biofeedback therapy; DD, dyssynergic defecation; DGBI, disorders of gut–brain interaction; DRE, digital rectal examination; EAS, external anal sphincter; EMG, electromyography; FI, fecal incontinence; HR, high-resolution; HRM, high-resolution manometry; IAS, internal anal sphincter; IBS, irritable bowel syndrome; LAS, levator ani syndrome; MEP, motor evoked potential; MRI, magnetic resonance imaging; PNTML, pudendal nerve terminal motor latency; QoL, quality of life; RAG, rectoanal gradient; RCT, randomized controlled trial; SAT, sensory adaptation therapy; SNS, sacral nerve stimulation; TNT, translumbosacral neuromodulation therapy.**

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Table 1. Rome V Anorectal Disorders

F. Anorectal disorders
F1. Fecal incontinence
F2. Anorectal pain
F2a. Levator ani syndrome
F2b. Unspecified anorectal pain
F2c. Proctalgia fugax
F3. Dyssynergic defecation
F4. Anorectal sensory dysfunction disorders
F4a. Rectal hyposensitivity
F4b. Rectal hypersensitivity

Structural disorders such as rectocele[12] and higher grades of rectal intussusception[17] are associated with FI. Anorectal surgery for hemorrhoids, fissures, fistula, or cancer can damage the sphincters[18] and proctitis or fecal impaction with overflow diarrhea, especially in older adults, can impair rectal compliance and cause FI.[19]

Justification for Changes in Diagnostic Criteria

Frequency. The term "recurrent FI" was nonspecific. In 1 study, 55% of patients had <1 FI episode per month, 24% had 1 episode per month, and the remainder had more frequent FI.[14] Even infrequent FI can significantly impair QoL[5] and such patients respond equally well to biofeedback therapy (BT).[20] Hence, the term "recurrent" was replaced with "2 or more" episodes of FI.

Developmental age. This was removed as we focus on FI in adults.

Duration. This was increased from 3 to 6 months, as some patients may not have experienced FI in the previous 3 months due to lifestyle changes and to align with other Rome Criteria.

Research studies. Up to 50% of patients have fewer than 1 episode of FI per month with significant impairment of QoL.[5,14,21] Requiring a threshold frequency of 1 FI episode per week may decrease eligibility for clinical and epidemiologic studies. Although therapeutic response may depend on baseline frequency, this can be addressed by using longer baseline and treatment periods and global measures of FI severity.[22] In addition, demonstrable evidence of FI episodes in a 4-week stool diary provides clarity, aligns with other Rome Criteria, and is affirmed by several randomized controlled trials (RCTs).[23–26]

F1. Diagnostic Criteria[a] for Fecal Incontinence

Must include *all* of the following:

1. Two or more episodes of uncontrolled passage of fecal material[b]
2. The individual should report at least 1 episode of fecal incontinence per month on a 4-week stool diary

[a]Criteria fulfilled for the last 3 months with symptom onset at least 6 months before diagnosis.
[b]For clinical purposes, 2 or more episodes of uncontrolled passage of fecal material in the past 12 months.

Pathophysiology

Physiological factors. Fecal continence is maintained by several mechanisms: internal anal sphincter (IAS) and EAS function, the puborectalis, preserved anorectal neurophysiology (ie, pudendal and levator ani nerves, inferior hypogastric plexus, pelvic splanchnic nerves),[1,27] rectoanal sensation, and rectal compliance.[1,28] Anal sphincter weakness is the most frequent abnormality. In older women with FI, approximately 40% had reduced resting and 80% had reduced squeeze pressure.[28,29] IAS dysfunction manifests as decreased resting pressure,[1,29] and results from sphincter defects or thinning or neurologic injury.[1,30,31] Severity of FI is greater in postpartum women with IAS defects.[32] Resting pressure is the strongest predictor of future FI after obstetric anal sphincter injury.[33] IAS weakness is often observed in idiopathic FI and scleroderma,[34] but not in fecal seepage.[35]

EAS damage, neuropathy, myopathy, or decreased corticospinal input may impair the EAS and reduce squeeze pressure.[36] The levator ani, including the puborectalis, contributes to fecal continence via a flap-valve effect,[37] whereas anal cushions and sphincter tone provide closure.[38] Excessive perineal descent leads to a more obtuse anorectal angle, impairing the flap-valve mechanism when intra-abdominal pressure increases.[37]

Up to one-third of patients with FI have normal anal sphincter pressures. Many of these have concurrent constipation[39] and coexisting pelvic organ prolapse.[40] Data in men are sparse, but studies suggest that fecal seepage predominates.[41] A case-control study observed that sphincter defects are less common in men, whereas impaired rectal sensation and evacuation disorders are more frequent than in women.[41]

Rectal hyposensitivity and increased rectal compliance predispose to reduced urge sensation, fecal retention, and allow anal leakage before EAS contraction.[28,42,43] Chronic fecal retention may blunt rectal sensation by altering rectal viscoelastic properties, or afferent signaling.[44] Conversely, rectal hypersensitivity coexists with reduced rectal compliance.[29,45] Rectal capacity is reduced in women with FI and is associated with urgency.[45] Thus, FI is a heterogeneous, disorder with most patients exhibiting multiple deficits.[1,28,29,45,46]

Anal sphincter relaxation during or independent of rectal distention allows sampling of rectal contents to discriminate gas, liquid, or stool.[44] The role of sampling in continence remains unclear,[47] but patients with FI exhibit fewer sampling responses and reduced anal sensation, potentially depriving them of sensory feedback.[44] An impaired rectosigmoid brake may also predispose to FI.[48]

Psychological factors. Some patients experience FI during periods of stress. FI is associated with anxiety and

depression. Because concurrent DGBI worsened FI severity and psychological distress, gut–brain axis dysfunction likely contributes to the pathophysiology of FI alongside anorectal abnormalities.[11]

Clinical Evaluation

History. Because patients may not volunteer FI symptoms, they should be asked directly.[49] Bowel questionnaires are useful,[50] but are limited by recall bias.[50] Prospective stool diaries or digital applications that incorporate pictorial stool scales can improve accuracy.[51,52] Building rapport with patients is essential, along with assessment of predisposing conditions and FI severity, which is determined by the type (ie, solid, liquid, and/or gas), quantity, and frequency of FI and awareness of the urge to defecate before incontinence.[1,4,5] Staining refers to leakage of small amounts of stool (1–2 tablespoons).[35] Soiling refers to more extensive leakage and can be specified further (ie, soiling of underwear, outer clothing, or furnishing/bedding).[35] Coexistent constipation and/or diarrhea are common.[39,40] The timing of incontinence—after meals, bowel movements, exercise, or at night—may provide etiologic clues and guide management.[53]

Symptoms can suggest underlying mechanisms. Incontinence for solid stool suggests more severe sphincter weakness than liquid stool alone.[54] Nocturnal incontinence is uncommon and often reflects IAS dysfunction (eg, diabetes and scleroderma). Urge incontinence is characterized by a strong desire to defecate before leakage, usually associated with reduced squeeze pressure[55] and duration,[56] and/or reduced rectal capacity and rectal hypersensitivity.[29,57] In contrast, passive incontinence occurs without awareness of stooling and is often associated with lower resting pressure.[46,55,56]

Digital Rectal Examination and Physical Examination

Patients must have an abdominal and neurologic examination together with a meticulous digital rectal examination (DRE).[58] Diagnostic testing aims to identify organic diseases or underlying pathophysiology and should be tailored to patient's age, probable etiology, symptom severity, and impact on QoL. These include endoscopic, manometric, radiologic, and neurophysiological tests[1,39,59] (Table 2).

Endoscopy. Endoscopic colorectal assessment may be desirable depending on symptom profile, age, and cancer risk factors, together with biopsies for microscopic colitis.

Manometry. Anorectal manometry (ARM) assesses mechanisms of continence and defecation, including the anal sphincter and puborectalis muscle function, rectoanal reflexes, and rectal sensorimotor functions.[1,58,60–62] Patients with FI may have reduced anal resting and/or squeeze pressures, as detailed above.[35,36,46,62]

Anal ultrasound. Anal ultrasound (AUS) depicts the IAS muscle as a homogeneous hypoechoic ring and the EAS with mixed echogenicity. AUS identifies anal sphincter thinning and/or defects—often clinically unrecognized—that may be amenable to surgical repair.[63,64] Although AUS reliably identifies IAS defects, interpretation of EAS images is operator-dependent and confounded by normal anatomic variation and interobserver variability. Compared with 2-dimensional AUS, 3-dimensional AUS measures the length and volume of EAS[65] and may detect atrophy. Vaginal ultrasound can visualize the anterior EAS.

Defecography. Defecography may be helpful in selected patients with FI, particularly preoperatively, to identify excessive perineal descent, rectocele, enterocele, rectal intussusception, or rectal prolapse.[66] Small rectoceles are relatively common in asymptomatic women.[67]

Pelvic magnetic resonance imaging. Magnetic resonance imaging (MRI) visualizes the anal sphincter structure, global pelvic floor motion, bladder, and genital organs in real-time without radiation exposure.[68,69] It is performed via intra-rectal administration of ultrasound gel, obtaining dynamic images at rest, squeeze, Valsalva, and rectal evacuation, and after emptying the bladder and rectum.[29,69]

Neurophysiology tests. Several tests are available as discussed below.[1,30,31] Others, including somatosensory evoked potentials, positron emission tomography scan, and functional brain MRI, are not used in clinical practice.[70]

Pudendal nerve terminal motor latency. Pudendal nerve terminal motor latency (PNTML) measures the latency time of the pudendal nerve after intrarectal stimulation with a prolonged latency suggesting neuropathy. Due to methodological limitations, PNTML is performed infrequently.[71,72]

Electromyography. Electromyography (EMG) includes surface, needle, and single-fiber methodologies.[72,73] EMG findings are characterized as normal, neurogenic, muscle injury, or nonspecific.[31] Among patients with FI, needle EMG may demonstrate injury to the EAS and/or puborectalis muscle.[29,37] Anal sponge electrode EMG identifies weak sphincters but cannot distinguish among causes of sphincter weakness. Intraluminal EMG may be more accurate and less likely to pick up artifacts.[73] In addition to diagnostic purposes, EMG is also used to provide EAS BT in patients with FI.[74]

Sensory testing. Anal sensation has been tested using electrical or thermal stimulation, but its clinical utility is unclear. Rectal sensation can be evaluated by balloon distention during ARM or with a barostat. Both rectal hyposensitivity[42,44] and hypersensitivity have been documented in FI.[45,75]

Translumbosacral anorectal magnetic stimulation test. This novel test measures the latencies of motor evoked potentials (MEPs) after magnetic stimulation of the lumbar and sacral plexus. Most patients with FI showed prolonged MEPs, and translumbosacral anorectal magnetic stimulation was superior to PNTML.[76] In addition, patients with spinal cord injury and bowel dysfunction had prolonged MEP latencies.[30] Its clinical utility was confirmed in a large series, with 50% of patients showing both lumbar and sacral plexus neuropathy and higher prevalence of anal than rectal neuropathy.[77] Afferent cortico-anorectal evoked

Table 2. Evidence-Based Summary of the Utility of Commonly Performed Diagnostic Tests in Fecal Incontinence and Constipation With Dyssynergic Defecation

Diagnostic tests	Strengths	Weaknesses	Evidence	Recommendation grade[a]	Comments
Clinical utility in FI					
ARM	Quantifies sphincter pressures, rectal sensation and compliance, and recto-anal reflexes	Availability, training	Good	A1	Facilitates diagnosis of FI and DD and rectal sensory disorders
Needle EMG	Quantifies spike potentials and reinnervation pattern indicating neuropathy/ myopathy	Lack of standardization	Fair	B3	Used only in research laboratories, painful
Surface EMG	Displays EMG activity and can provide information on normal or weak muscle tone	Inaccurate, artifacts	Fair	B3	Largely used for biofeedback
PNTML	Measures latency of terminal portion of pudendal nerve	Minimally invasive, low sensitivity, interobserver differences	Fair	B3	Conflicting recommendations, rarely used clinically
Translumbosacral anorectal magnetic stimulation	Quantifies lumbar and sacral plexus nerve innervation of the anorectum using MEPs, useful in monitoring treatment response	Availability, training	Good	B2	Identifies neuropathy in FI, LAS, spinal cord injury, and mixed FI and constipation
AUS	Visualization of the IAS and EAS and puborectalis muscle and quantifies muscle thickness, defects, and scarring	Operator-dependent, availability, cost	Good	B2	Not widely available; rigid ultrasound probe useful but endoscopic AUS not reliable
MR defecography	Lack of standardization	Availability, cost, and assessment in lying position, not physiological	Good	B2	Open magnet and seated assessment is ideal but not widely available
Clinical utility in constipation with DD					
Blood tests (thyroid, calcium, glucose, electrolytes)	Evaluate metabolic disorder causing constipation	Not cost-effective	No evidence	C	Not recommended routinely without alarming features
Plain abdominal radiograph	Identify excessive amount of stool in the colon, simple, inexpensive, widely available	Radiation exposure, lack of controlled studies	Fair	B2	Recent studies describe usefulness
Barium enema	Identify megacolon, megarectum, stenosis, diverticulosis, masses	Lack of standardization, radiation exposure, lack of controlled studies	Poor	C	Not recommended for routine evaluation without alarming features
Fluoroscopic defecography	Identify dyssynergia, rectocele, enterocele, prolapse, excessive perineal descent, megarectum, and Hirschsprung disease	Radiation exposure (modest), interobserver bias, inconsistent methodology	Fair	B3	Performed in seated position. Helps diagnosis of DD when ARM/BET are inconclusive
AUS	Visualization of the anal sphincters	Interobserver bias, availability, limited normative data	Poor	C	Experimental

Table 2. Continued

Diagnostic tests	Strengths	Weaknesses	Evidence	Recommendation grade[a]	Comments
MRI	Simultaneously evaluate global pelvic floor anatomy, sphincter morphology, and dynamic motion, detects rectal intussusception, prolapse, rectocele	Expensive, lack of standardization, availability	Fair	B3	Used as adjunct to ARM
Flexible sigmoidoscopy and colonoscopy	Visualization of mucosal disease	Invasive, risks of procedure and sedation	Poor	C	Lack of prospective study regarding efficacy
Colonic transit with radiopaque markers	Evaluate colonic transit, inexpensive, and widely available	Inconsistent methodology	Good	B2	Useful to identify slow transit constipation
Colonic transit with scintigraphy	Evaluate slow, normal, or rapid colonic transit	Expensive, time-consuming, availability, lack of standardization	Good	B2	Facilitates classification of pathologic subtypes
Wireless motility capsule	Standardized evaluation of slow, normal, or rapid colonic and upper-gastrointestinal transit, no radiation, validated technique	Availability	Excellent	A1	Reliability identifies slow-transit constipation and upper-gut transit abnormalities, 2 new capsule techniques are now available
ARM	Identify DD, weak or hypertonic anal sphincters/puborectalis, rectal hyposensitivity, rectal hypersensitivity, rectal compliance, and Hirschsprung disease	Availability, training	Excellent	A1	International Anorectal Physiology Working Group protocol and London Classification are helpful but needs further refinement
BET	Identify DD and impaired evacuation	Lack of standardization	Good	B2	Different methodologies and balloons with variable normative values; lack of inexpensive commercial balloon

From Rao et al,[59] adapted with permission.

[a]Grade A1: excellent evidence in favor of the test based on high specificity, sensitivity, accuracy, and positive predictive values; grade B2: good evidence in favor of the test with some evidence on specificity, sensitivity, accuracy, and predictive values; grade B3: fair evidence in favor of the test with some evidence on specificity, sensitivity, accuracy, and predictive values; grade C: poor evidence in favor of the test with some evidence on specificity, sensitivity, accuracy, and predictive values.

potentials were also prolonged in patients with FI, suggesting impaired gut–brain signaling.[78]

Treatment

Management of FI must be individualized and directed toward treating underlying pathophysiology, associated disease, and symptom burden.

Dietary or pharmacologic interventions. The primary goal is to optimize stool form and restore normal bowel habits, especially in patients with loose stools.[68,79,80] Dietary changes (eg, low lactose or low fermentable oligo-, di-, monosaccharides and polyols diets), psyllium but not gum arabic or carboxymethylcellulose, improved FI compared with placebo in a controlled study.[81] For diarrhea without fecal impaction, loperamide or diphenoxylate-atropine can improve stool consistency. Loperamide also increases IAS tone.[82] Amitriptyline may improve FI via anticholinergic effects.[83] Bile acid sequestration with cholestyramine benefits patients with diarrhea and FI.[84] In an RCT, clonidine was not beneficial over placebo.[51] In another RCT, oral clonidine (0.1 mg twice daily) with colesevelam (1875 mg twice daily), but not placebo, improved stool form, decreased FI episodes for semi-formed stools, and FI severity.[85]

Transanal therapies. In patients with FI and abnormal evacuation, transanal therapies including suppositories (eg, glycerine and bisacodyl), low-volume enemas (ie, water or osmotic), and irrigation may be useful, but studies are lacking. In older adult patients, a regimen of lactulose (10 mL twice daily) plus weekly enemas improved FI.[86]

Transanal irrigation (low or high volume) systems have gained popularity. RCTs support its efficacy in patients with neurogenic bowel or spinal cord injury,[87] spina bifida,[88] and anterior resection syndrome.[89] Transanal irrigation improved FI severity, constipation, and bowel satisfaction, although more than one-third of patients discontinued within 1 year[90] due to poor satisfaction and/or adverse effects.[90]

Anal plug devices or mechanical barriers to prevent FI may provide benefit.[91–93] In the pivotal study of an anal insert device, 77% of 73 completers and 62% of 91 intention-to-treat participants reported a ≥50% reduction in FI frequency.[91] One US Food and Drug Administration–approved vaginal bowel control device uses an inflatable balloon to compress the rectum. Among 110 women, 65 were successfully fitted with the device and 79% achieved a ≥50% reduction in FI at 1 month. Another US Food and Drug Administration–approved anal insert device was safe, well tolerated, and effective. More than 75% and 54% of participants reported a 50% and 75% reduction in the frequency of moderate or full bowel movement FI.[92] Further research regarding the long-term efficacy, usability, and safety is needed.[93]

Biofeedback therapy. Biofeedback uses operant conditioning and instrumental learning[43,94,95] along with Kegel exercises.[1] Using a rectal balloon anal manometry device or a surface EMG device, patients are taught to selectively contract the EAS and puborectalis to improve strength and duration of anal squeeze and correction of dyssynergia when present.[74,95] Feedback is provided through visual tracings of balloon volume and/or anorectal pressure (Figure 1). Further reinforcement is provided by the therapist and aided through sensory perception and balloon distention with progressively smaller volumes.[95] In uncontrolled studies, intact baseline sensation and improved sensory discrimination predicted greater success.[43,96] Multiple RCTs have demonstrated the efficacy of BT (Table 3),[26,96–110] with improvements in symptoms and QoL.[26,43,74,95–104,107,108,110] Long-term follow up confirms durability with more than one-half of patients maintaining benefit in a 7-year median follow-up study.[111] Patients failing initial therapy rarely respond later and should be referred for alternative therapies.[20]

Surgical approaches. Surgery may be considered for patients unresponsive to optimal medical and behavioral therapy. Available options range from minimally invasive neuromodulation techniques to reconstructive approaches. High-quality evidence of long-term efficacy is limited.

Neuromodulation and minimally invasive treatment approaches. *Sacral nerve stimulation.* Sacral nerve stimulation (SNS) is typically performed in 2 stages, with permanent implantation being contingent on response to a 2- to 4-week test period (Figure 1). In the pivotal open-label US multicenter study, 90% of 120 patients received permanent stimulation.[112] At 5 years, 76 of 120 (63%) patients were evaluated; 36% achieved complete continence and 89% were deemed a therapeutic success.[113] In another 5-year follow-up of 101 patients with FI with intention-to-treat analysis, 42.6% had favorable outcome; younger age, urge FI, and 6-month response predicted success.[114] A crossover study (n = 27) reported a 90% reduction in FI episodes during SNS vs 76% without, but with significant attrition bias.[112] Despite widespread use, no high-quality, multicenter, sham-controlled RCT has confirmed its efficacy and its mechanisms of action remain uncertain.[115] In addition, sham-controlled trials showed clinically meaningful improvement with sham stimulation.[116]

Percutaneous tibial nerve stimulation. Percutaneous tibial nerve stimulation has not shown significant benefit over sham in multiple RCTs, including a large trial.[23] It may be combined with biofeedback in patients with coexistent FI and obstructive defecation.[117]

Translumbosacral neuromodulation therapy. Translumbosacral neuromodulation therapy (TNT) delivers repetitive magnetic stimulations (Figure 1) to the lumbar and sacral plexus nerves and improves nerve conduction through neuroplasticity. In a dose ranging RCT of 1 vs 5 vs 15 Hz of TNT, the 1-Hz frequency was superior, with 91% responders (>50% reduction in FI episodes).[25] Anal neuropathy, squeeze pressure, and rectal capacity, as well as QoL, improved significantly with good correlation between improved neuropathy and

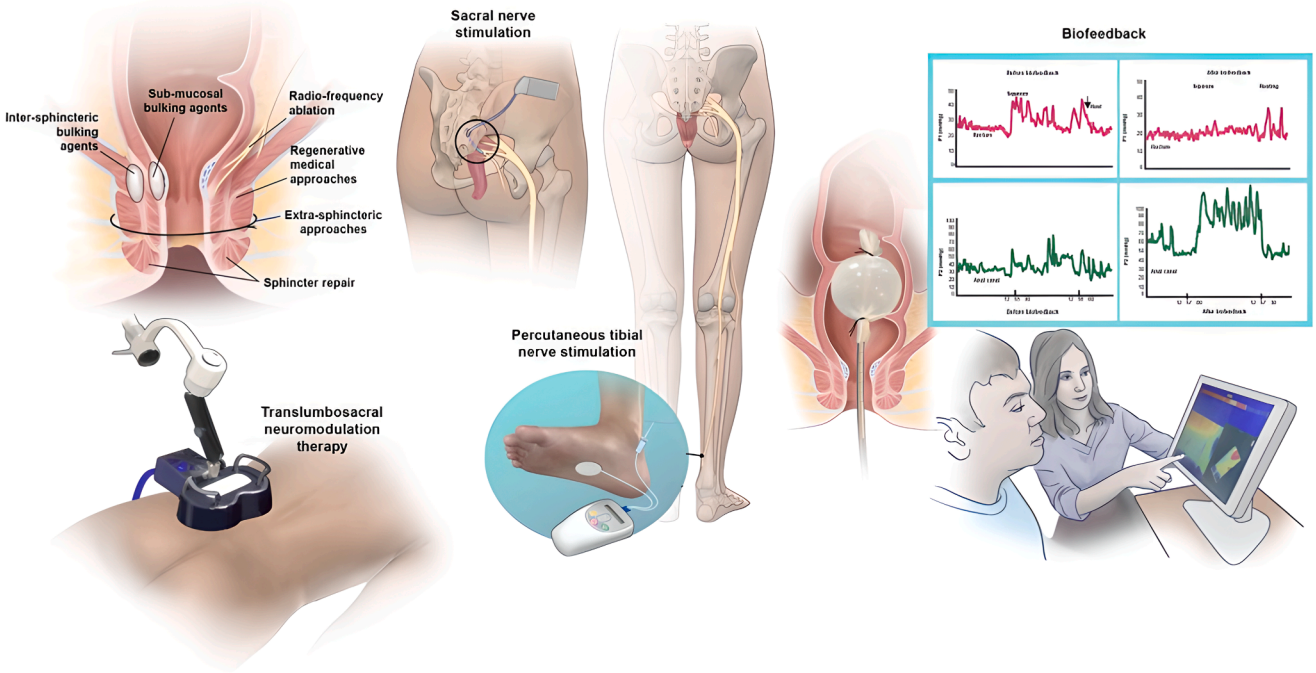

Figure 1. The current and novel neuromodulation therapies and others for the treatment of fecal incontinence. During biofeedback, the *left* panel at baseline shows weak anal squeeze with an abnormal increase in rectal pressure whereas after training, there is significant increase in anal squeeze without rectal pressure change.

FI responders. This suggests that TNT improves FI through neuromodulation.[118] A recent sham-controlled study further confirmed TNT's efficacy.[119]

Sphincter bulking therapies. Biocompatible materials have been injected into the submucosal or intersphincteric space to augment the anal barrier (Figure 1). Five RCTs have evaluated 10 different materials in 382 patients.[120] In a pivotal trial (n = 206), dextranomer in stabilized hyaluronic acid achieved a 52% response (50% reduction in FI episodes) vs 31% with sham (number needed to treat = 4.4).[121] Adverse events were mostly mild (eg, proctalgia and bleeding) with rare abscesses. Although sustained benefit up to 3 years has been suggested,[122] improvement in QoL was not significant and physiological or imaging data are lacking. At 2 years, the dextranomer in stabilized hyaluronic acid outcomes were comparable with BT.[123] Patients with a shorter FI duration, obstetric causes, and no prior therapy, responded best.[124] A recent large, multicenter RCT found that dextranomer and BT had an approximately 29% responder rate.[121]

SphinKeeper (THD SpA) implants 10 self-expandable polyacrylonitrile prostheses (20 mm × 1.8 mm) into the intersphincteric space.[125] Adverse events include prosthetic displacement (14%) and anorectal pain (71%).[125] A recent multicenter retrospective cohort study with a 3-year follow-up confirmed the safety, efficacy, and QoL benefits of SphinKeeper implantation in patients with refractory FI.[126] Long-term outcomes are lacking.

Intrarectal botulinum toxin. Intrarectal injection of botulinum A toxin has been shown to reduce frequency of FI episodes plus urgency episodes in a randomized placebo-controlled study.[127] This approach deserves further evaluation, especially for urge FI.

Direct reconstructive approaches. Historically, most surgery for FI has been directed toward repair of anal sphincter defects.[128] In short-term studies, up to 60%–85% of patients had improved after an overlapping anterior sphincteroplasty.[128] However, long-term results are disappointing; failure rates of approximately 50% are seen after 40–60 months.[129] Recent innovations include autologous skeletal muscle–derived cell implantation[130] and implantation of a bioengineered IAS from autologous cells.[80,131]

Ostomy. A colostomy remains a last resort for patients with severe FI and improves QoL.[128]

F2. Anorectal Pain Disorders

The following 3 anorectal pain syndromes have been described: levator ani syndrome (LAS), unexplained anorectal pain, and proctalgia fugax. Although there is an overlap, they are primarily distinguished based on pain duration and presence or absence of levator ani tenderness during a DRE.

F2a. Levator Ani Syndrome and F2b. Unexplained Anorectal Pain

Definition

LAS is often described as a vague, dull ache, or pressure sensation in the rectum that is worse with sitting than standing or lying down. DRE reveals tenderness on palpation of the levator ani muscles. Although left-sided tenderness was felt to be more common,[132] a recent

Table 3. Summary of Randomized Controlled Trials of Biofeedback Therapy for Fecal Incontinence and Constipation With Dyssynergic Defecation

Study, first author (year)	Comparison	Technique	Primary outcome	Follow-up duration	Lost to follow-up	Result
Fecal incontinence						
Miner[97] (1990)	Manometric biofeedback with rectal sensory training (n = 13) vs sham (crossover) (n = 12)	Manometry weekly, 4 wk	Symptoms assessment, no. of FI episodes, sensory thresholds	1 mo	0%	Favors biofeedback Active sensory retraining reduced the sensory threshold from 32 ± 8 mL to 7 ± 2 mL ($P < .001$), corrected sensory delay that was present ($P < .004$), and reduced the frequency of incontinence episodes from 5 ± 1 to 1 ± 1 episodes per week ($P < .01$). Sham training caused a modest reduction in the sensory threshold (from 29 ± 9 to 20 ± 8; $P < .05$) but did not significantly reduce the frequency of incontinence.
Fynes[98] (1999)	EMG biofeedback (n = 20) vs vaginal manometric biofeedback + Kegel (n = 20)	EMG, weekly 30-min sessions for 12 wk	Symptoms global assessment, physiological parameters	3 mo	1/20 (5%) vs 0/20 (0%)	Favors biofeedback Continence scores improved in both treatment groups, but the results were better for those who received biofeedback. ARM was vaginal biofeedback, whereas anal resting and squeeze pressures increased with EMG feedback.
Heymen[99] (2000)	1. Outpatient intra-anal EMG biofeedback training (n = 10) 2. EMG plus intrarectal balloon training (n = 10) 3. EMG plus a home trainer (n = 10) 4. EMG, balloon training, and home trainer (n = 10)	EMG, weekly, 60 min, 4 sessions	Reduction in incontinent episodes (days/week)	End of treatment	2/10 (20%) vs 2/10 (20%) vs 2/10 (20%) vs 0/10 (0%)	Similar efficacy BT significantly improves FI. Moreover, EMG training was as effective alone as when combined with home trainer, balloon training, or both for the treatment of FI.

Table 3. Continued

Study, first author (year)	Comparison	Technique	Primary outcome	Follow-up duration	Lost to follow-up	Result
Solomon[100] (2003)	1. Biofeedback with anal manometry (n = 39) 2. Biofeedback with transanal ultrasound (n = 40) 3. Pelvic floor exercises with feedback from digital examination (n = 41)	Manometry, 5 monthly, 30 min/session	Symptoms improvement	5 mo	8/39 (21%) vs 4/40 (10%) vs 4/41 (15%)	Similar efficacy No significant differences between groups in compliance, physiologic sphincter strength, and clinical or QoL measures.
Norton[96] (2003)	1. Standard care (advice) (n = 37) 2. Advice plus instruction on sphincter exercises (n = 43) 3. Hospital-based computer-assisted sphincter pressure biofeedback (n = 49) 4. Hospital biofeedback plus home EMG biofeedback (n = 42)	EMG, up to 9 sessions over 3–6 mo, 40–60 min/session	Patient's own view of the effectiveness of treatment	12 mo	31/171 (18%)	Similar efficacy No significant difference in global rating of symptom changes between the 4 groups. Approximately 53% improved with hospital-based or home biofeedback.
Mahony[101] (2004)	Biofeedback (n = 30) vs biofeedback + electrical stimulation (n = 30)	EMG intraanal, weekly for 12 wk, 30 min/session	Symptom questionnaire to determine continence score, anal manometry, and endoanal ultrasound scanning	3 mo	4/30 (13%) vs 2/30 (6%)	Similar efficacy After treatment, both groups demonstrated significant improvement in continence score ($P < .001$) and in anal squeeze pressures ($P < .04$). Resting anal pressures did not alter significantly.
Davis[102] (2004)	Sphincter repair alone (n = 20) vs sphincter repair + biofeedback (n = 18)	Manometry, weekly 6 sessions, 60 min/session	Subjective analysis of outcome and change in continence status	6 wk	3/20 (15%) vs 4/18 (22%)	Favors biofeedback After surgery continence function improves in all patients but adjuvant BT improves QoL and maintains symptomatic improvement over time.
Ilnyckyj[103] (2005)	Pelvic exercises (verbal education) (n = 11) vs BT (n = 7), all women	Manometry, 4 weekly, 30–45 min/session	Success in bowel control	1 mo	5/23 (22%)	Similar efficacy Responders for BT (86%) was more than exercises (45%) but not significant, $P = .15$.

Table 3. Continued

Study, first author (year)	Comparison	Technique	Primary outcome	Follow-up duration	Lost to follow-up	Result
Naimy[104] (2007)	Home biofeedback (n = 24) vs electrical stimulation (n = 25)	EMG, 2 face-to-face sessions (30 min) to ensure that the patient had understood the treatment; then 30-min sessions twice daily during 8 wk at home, with the standard equipment	Subjective global assessment, QoL	2 mo	5/24 (21%) vs 4/25 (16%)	Similar efficacy Neither biofeedback nor electrostimulation treatments improved Wexner incontinence score, reduced QoL, or FI QoL scores. Both treatments resulted in improvement of patients' subjective perception of incontinence control.
Heymen[105] (2009)	Biofeedback plus pelvic floor exercise (n = 45) vs pelvic floor exercises alone (n = 63)	Manometry, 6 biweekly, 50 min/session	FI severity index	3 mo	10/63 (16%) vs 5/45 (12%) at 3 mo	Favors biofeedback 76% (biofeedback) vs 41% (exercises) reported adequate relief of symptoms ($P < .001$).
Damon[106] (2014)	Biofeedback (n = 77) vs standard of care (n = 80)	EMG, 20 sessions of 30 min by physiotherapist, over 4-mo period at an initial rate of 2–3 sessions per week for the first 4 wk, and 1–2 sessions per week thereafter	Each patient rated his view of the treatment's effectiveness as a change on an ordinal scale from –5 to +5; success was defined as a rating $\geq +3$	4 mo	10/77 (13%) vs 5/80 (6%)	Favors biofeedback After a 4-mo follow-up. The success rate was significantly higher in the biofeedback group (57% vs 37%; $P < 0.021$). In the biofeedback group, daily stool frequency, leakage, and fecal urgency significantly decreased, and daily nonurgent perception of stool increased.
Young[107] (2018)	1. Face-to-face biofeedback (n = 111) 2. One face-to-face vs biofeedback followed by telephone biofeedback (2 groups, n = 6 and n = 66) 3. One-off face-to-face biofeedback (n = 68)	Manometry, 4 monthly sessions, 60/min session; telephone biofeedback was one 20-min session; 1 face-to-face biofeedback was one 120-min session	Severity of FI and QoL as assessed by the 36-item Short Form Health Survey	4 mo	23/111 (21%) vs 15/106 (14%) and 20/66 (30%) Vs 18/68 (26%)	Similar efficacy All groups had significant improvements in FI, QoL incontinence score and mental status ($P > .001$ each). There were no differences in improvements in FI between groups although patient satisfaction was less with reduced face-to-face contact.

Table 3. Continued

Study, first author (year)	Comparison	Technique	Primary outcome	Follow-up duration	Lost to follow-up	Result
Ussing[108] (2019)	Biofeedback plus PMFT (n = 49) vs conservative treatment (n = 49)	EMG, 6 sessions over 16 wk, 45 min/session	Symptom changes, based on score from the Patient Global Impression of Improvement Scale	4 mo	1/49 (2%) vs 2/49 (2%)	Favors biofeedback + PFMT Patients in biofeedback + PFMT group were significantly more likely to report improvement in incontinence symptoms based on Patient Global Impression of Improvement Scale scores (unadjusted odds ratio, 5.16; 95% CI, 2 18–12.19; $P = .0002$).
Jelovsek[109] (2019)	1. Oral placebo + education only (n = 42) 2. ARM-assisted biofeedback + placebo (n = 84) 3. Loperamide + education only (n = 88) 4. ARM-assisted biofeedback + loperamide (n = 86)	Manometry, 6 biweekly, duration nonspecified	St Marks score, ≥50% reduction in FI episodes (secondary)	3 mo	4/42 (10%) vs 8/84 (10%) vs 6/88 (7%) vs 8/86 (9%)	Similar efficacy Loperamide is equivalent to placebo. Anal exercises with biofeedback is equivalent to an educational pamphlet. Loperamide and biofeedback are equivalent to oral placebo and biofeedback or loperamide plus an educational pamphlet. 50% reduction in FI episodes was significantly different between biofeedback + exercise vs exercise alone (83.7% vs 57.5%, $P < .0009$).
Mundet[110] (2021)	1. Kegel (n = 45) 2. Biofeedback + Kegel (n = 45) 3. Electrostimulation + Kegel (n = 45) 4. Neuromodulation + Kegel (n = 45)	Manometry, 6 biweekly, 45 min/session	FI severity index, physiological parameters, and QoL	3 mo	9/45 (20%) vs 9/45 (20%) vs 6/45 (13%) vs 5/45 (20%) vs	Similar efficacy The treatments for FI have a strong and similar efficacy on severity and QoL but affect specific pathophysiological mechanisms. Response rate was 38.50%, 46.70%, 65.60%, and 56.30% for, Kegel, biofeedback + Kegel, electrostimulation + Kegel, and neuromodulation + Kegel, respectively.

Table 3. Continued

Study, first author (year)	Comparison	Technique	Primary outcome	Follow-up duration	Lost to follow-up	Result
Xiang[26] (2021)	Office biofeedback (n = 10) vs home biofeedback (n = 20)	Office: manometric biofeedback, 6 sessions, weekly, 30 min/ sessions. Home: biofeedback 1–2 sessions/d for 6 d/wk, 6 wk	Change in weekly incontinence episodes, responder = >50% reduction in FI episodes	6 wk	0/10 (0%) vs 2/29 (19%)	Favors biofeedback with similar efficacy Weekly FI episodes/wk decreased significantly in home biofeedback group (4.7 + 1.8; $P = .003$) and office group (3.7 + 1.6; $P = .0003$) compared with baseline but no difference between groups; $P = .2$. Home is noninferior to office treatment. Responder rate = ∼65% in both groups. Home biofeedback is safe, effective, improves QoL and through increased access could facilitate improved management of FI.
Dyssynergic defecation						
Chiarioni[201] (2006)	Biofeedback (n = 54) vs polyethylene glycol (n = 55)	EMG, 5 weekly, 30 min/session	Global symptoms improvement	24 mo	2/54 (3.7%) vs 4/55 (7.3%)	Favors biofeedback Global symptom improvement 80% vs 22%; $P < .01$. More pelvic floor relaxation, improved evacuation and urge threshold; vs polyethylene glycol; $P < .01$.
Heymen[202] (2007)	Biofeedback (n = 30) vs diazepam (n = 30) or placebo pill (n = 24)	EMG, 6 biweekly, 50 min/session	Adequate relief of constipation	3 mo	7/30 (23.3%), 7/30 (23.3%), 4/24 (16.7%)	Favors biofeedback Adequate relief of constipation 70% vs 23% and 38%; $P < .01$. More pelvic floor relaxation in biofeedback vs diazepam and placebo; $P = .001$.

Table 3. Continued

Study, first author (year)	Comparison	Technique	Primary outcome	Follow-up duration	Lost to follow-up	Result
Rao[198] (2007)	Biofeedback (n = 28) vs sham feedback (n = 25) vs standard treatment (n = 24)	Biofeedback = 6 manometric biweekly, 60 min/session; sham = 6 visits, biweekly with probe placement and listening to mindful relaxation 60 min/session; Standard = laxatives, diet, counseling	CSBMs, global bowel satisfaction	3 mo	7/28 (25%); 7/30 (23.5%); 4/24 (16.7%)	Favors biofeedback Global bowel satisfaction 75% vs 23% vs 38%; $P < .01$. Higher no. of CSBM/wk ($P = .05$), proportion of patients with corrected dyssynergia ($P < .0001$), improved defecation index ($P < .0001$), and decreased balloon expulsion time ($P < .05$) in the biofeedback group compared with sham and standard groups.
Simon[203] (2009)	Biofeedback (n = 15) vs counseling focused on behavioral mechanisms involved in defecation (n = 15)	EMG, once per week, 45 min/session	Clinical (symptoms) and psychological assessment before treatment	1 mo	0%	Favors biofeedback Significant improvements in physiological measures (EMG activity during straining to defecate and anismus index), as well as in clinical variables (frequency of defecations per week, sensation of incomplete evacuation, difficulty evacuation level and perianal pain) only in the EMG-biofeedback group.
Farid[204] (2009)	Biofeedback (n = 24) vs botulinum toxin A injection 100 U into puborectalis muscle (n = 24)	Pressure probe biofeedback, 8 sessions, biweekly, 1 mo	Not clearly defined	1 mo	3/24 (12.5%) vs 0%	Similar efficacy Overall satisfaction 25% vs 33%; $P = .23$. Both treatments significantly improved dyssynergic pattern (54% vs 70%; $P = .54$) and balloon expulsion time (29% vs 37%; $P = .32$) but no difference between groups.

Table 3. Continued

Study, first author (year)	Comparison	Technique	Primary outcome	Follow-up duration	Lost to follow-up	Result
Pourmomeny[205] (2011)	Biofeedback (n = 34) vs balloon defecation training (n = 31)	EMG, 1 session, 30 min, vs Foley balloon expulsion training	Patients' satisfaction with their defecation ranked on a 3-point scale (low, moderate, or high), time to expel balloon during defecation	12 mo	0%	Favors biofeedback Patient satisfaction after treatment reached 52% with balloon training and 79% in the biofeedback group ($P > .05$). The change in balloon expulsion time for the biofeedback group was 16 s vs 1 s for the balloon-trained group ($P = .03$).
Hart[206] (2012)	Biofeedback (n = 10) vs control (trapezius or temporal muscle relaxation) (n = 11)	EMG, 6 sessions, biweekly, 30 min	Overall severity scores, symptoms, and QoL	3 mo	4/10 (40%) vs 2/11 (18%)	Favors biofeedback Overall constipation severity scores decreased by 35% in the biofeedback arm vs 15.3% in control. Obstructive defecation symptom scores decreased by 37.9% vs 19.7%.
Simon[207] (2017)	Biofeedback (n = 10) vs counseling sessions (n = 10) in community-dwelling older adult women (mean age, 74 y; range, 69–81 y)	EMG, 8 biweekly, 45 min/session	Clinical and physiological parameters	3 mo	0%	Favors biofeedback Significant improvements in all clinical parameters (stool frequency, sensation of incomplete evacuation, and difficulty evacuation) and mean EMG-activity of the EAS during straining to defecate and the anismus index (EMG-activity during straining/EMG activity during squeezing).
Rao[208] (2018)	Home biofeedback (n = 50) vs office biofeedback (n = 50)	Home device 20 min, twice daily vs manometry 6 biweekly, 60-min session	≥1 CSBM + correction of dyssynergia	3 mo	12/50 (24%) in home and 5/50 (10%) in office biofeedback	Favors biofeedback Home and office biofeedback equally efficacious. Home and office significant improvement. Responders were 68% in home and 70% in office biofeedback.

Table 3. Continued

Study, first author (year)	Comparison	Technique	Primary outcome	Follow-up duration	Lost to follow-up	Result
Simon[209] (2019)	Biofeedback (n = 10) vs counseling focused on behavioral mechanisms involved in defecation (n = 10)	EMG twice per week 45-min/session	Clinical symptoms and QoL (PAC-SYM and PAC-QoL)	3 mo	0%	Favors biofeedback Overall constipation severity scores decreased by 35% in the EMG biofeedback arm vs 15.3% in behavioral arm. Obstructive defecation symptom scores decreased by 37.9% vs 19.7%.

CSBM, complete spontaneous bowel movements; PAC-SYM, Patient Assessment of Constipation Symptom questionnaire; PAC-QoL, Patient Assessment of Constipation Quality of Life questionnaire; PMFT, pelvic muscle floor therapy.

study found that DRE reproduced pain in 1 or more anal quadrants with similar pain severity.[133] Patients with no tenderness are classified as having unexplained anorectal pain.

Epidemiology

The prevalence of anorectal pain from all causes is estimated at 11.6% in the community and is similar across genders.[134] The prevalence of LAS in the Rome Foundation Global Epidemiology Study was 1.1% in an internet survey and 0.7% in a household survey.[4] Because DRE was not possible in this questionnaire study, unexplained anorectal pain was also included.

Justification for Changes in Diagnostic Criteria

The following 4 changes were made: (1) the order of other conditions was changed to be consistent with the frequency of these disorders; (2) the phrase "structural alterations" was replaced with "disorders"; (3) "posterior traction" was removed because traction at other quadrants also evoked pain[133]; and (4) the term "functional" was removed. Although we have distinguished LAS from unexplained anorectal pain, largely based on 1 study.[135] The following discussion applies to both conditions.

Pathophysiology

Physiological factors. LAS is hypothesized to result from excessive pelvic floor contraction, reflected by elevated resting pressures.[136] In an RCT of 157 patients with chronic anorectal pain (Rome II Criteria), 85% of patients with levator tenderness had dyssynergia with manometry but not constipation; reversal with biofeedback strongly predicted symptom relief, implying dyssynergia as a mechanism.[135] Similarly, 3-dimensional AUS showed a more contracted puborectalis at rest and straining in chronic proctalgia compared with healthy controls,[137] and 34% of patients with LAS met criteria for DD. Recent studies demonstrate impaired anorectal afferent pathways[138] and a high prevalence of lumbo-anal and sacro-anal plexus neuropathy,[133] suggesting that neurophysiological dysfunction may cause pain in LAS.

Psychological factors. LAS is associated with psychological disturbances.[139] In a case-control study, women with a history of sexual abuse had a higher prevalence of LAS and proctalgia fugax.[140]

Clinical Evaluation

Diagnosis relies on characteristic episodes of anorectal pain with levator muscle tenderness on palpation.[133] In addition to excluding a fissure, an anoscopy and sigmoidoscopy may identify mucosal disease and if perianal disease (abscess or fistula) is suspected, cross-sectional imaging should be performed. Next, ARM, defecography, and translumbosacral anorectal magnetic stimulation test should be considered.[77,133] MEP latencies were prolonged in patients with LAS indicating lumbar or sacral plexus neuropathy.[133] Patients with LAS also have weak anal

F2a. Diagnostic Criteria[a] for Levator Ani Syndrome

Must include *all* of the following:

1. Chronic or recurrent anorectal pain
2. Episodes last 30 minutes or longer
3. Tenderness during traction on the puborectalis
4. Exclusion of other causes of anorectal pain, such as anal fissure, thrombosed hemorrhoids, malignancy, and infectious or inflammatory causes, such as inflammatory bowel disease, perianal abscess, fistula, prostatitis, coccygodynia, and gynecologic causes of pelvic pain

[a]Criteria fulfilled for the last 3 months with symptom onset at least 6 months before diagnosis.

F2b. Diagnostic Criteria[b] for Unexplained Anorectal Pain

[b]Symptom criteria for levator ani syndrome but no tenderness during traction on the puborectalis muscle

sphincters, and features of DD with prolonged balloon expulsion time.[133]

Treatment

In an RCT, 87% of patients with levator tenderness reported adequate pain relief after BT compared with 45% after electrical stimulation and 22% after massage; patients without tenderness did not benefit from any therapy.[135] Electrogalvanic stimulation may be considered if BT is not available. Retrospective studies suggested symptom improvement after intra-anal or intra-levator injection of botulinum A toxin injection,[141] but 1 RCT found no benefit over placebo.[142] An open-label study of TNT reported significant improvement in anorectal pain and lumbosacral neuropathy.[133] Other treatments (eg, muscle relaxants, diazepam, SNS, and sitz baths) are ineffective, as are radiofrequency, pudendal infiltration, transgluteal decompression, TENS, and laparoscopic decompression.[143]

F2c. Proctalgia Fugax

Definition

Proctalgia fugax is defined as sudden, severe pain in the rectal area, lasting for a few seconds to several minutes, and then disappearing completely.[144,145] Attacks are infrequent, typically occurring fewer than 5 times per year in 51% of patients.[144,145] The pain has been described as cramping, gnawing, aching, or stabbing ranging from uncomfortable to unbearable.[144,145] Although patients often recall nocturnal attacks, it can happen anytime, interrupting activities in 50% of patients.[144,145]

Epidemiology

The prevalence was 5.6% in an internet survey and 1.5% (range, 1.4%–1.7%) in household survey.[4,135] Because these were questionnaire-based, other anorectal disorders (eg, anal fissure) were not excluded. Proctalgia fugax is 1.3-fold more common in women,[4] and occurs in children.[144] Although few patients seek health care, it can be disabling.[134]

F2c. Diagnostic Criteria[a] for Proctalgia Fugax

Must include *all* of the following:

1. Recurrent episodes of pain localized to the anorectum and unrelated to defecation
2. Episodes last from seconds to minutes
3. There is no anorectal pain between episodes
4. Exclusion of other causes of anorectal pain, such as anal fissure, thrombosed hemorrhoids, malignancy, and infectious or inflammatory causes, such as inflammatory bowel disease, perianal abscess and fistula, prostatitis, coccygodynia, and major structural disorders of the pelvic floor

[a]Criteria fulfilled for the last 3 months with symptom onset at least 6 months before diagnosis.

Pathophysiology

Physiological factors. Several studies suggest that abnormal smooth muscle contractions may be responsible for the pain.[146] Hypertrophy of the IAS has been reported in a hereditary form of proctalgia fugax.[147]

Psychological factors. Attacks of proctalgia fugax are often triggered by stressful life events or anxiety.[148] In an uncontrolled study, most patients were perfectionistic, anxious, and/or hypochondriacal[149] and association with sexual abuse has been reported.[140]

Clinical Evaluation

Diagnosis is based on characteristic symptoms and exclusion of anorectal and pelvic pathophysiology. Chronic pelvic pain pathologies such as endometriosis (women) and chronic prostatitis (men) may mimic these symptoms.

Treatment

Proctalgia fugax has been described as "harmless, unpleasant, and incurable."[150] Reassurance is often sufficient, but frequent or severe attacks may require therapy. In an RCT, inhaled salbutamol shortened duration of severe and prolonged attacks compared with placebo.[151] Other options such as clonidine,[152] amylnitrite, nitroglycerine,[1] neuromodulators, or behavioral therapies have been suggested, although no comparative studies exist.

F3. Dyssynergic Defecation

Definition

DD is characterized by paradoxical contraction or inadequate relaxation of the pelvic floor muscles and/or inadequate propulsive forces during attempted defecation.[68,153] Because symptoms alone do not distinguish DD from other causes of evacuation disorders,[154–157] and some asymptomatic controls show dyssynergia pattern,[156–158]

the diagnosis requires constipation symptoms and abnormal anorectal tests. We prefer the term "dyssynergic defecation" over pelvic floor dyssynergia because most patients with DD report only difficult defecation, not urinary or sexual dysfunctions.[1]

Epidemiology

Because the diagnosis of DD requires physiologic testing, its population prevalence is uncertain. Of 11,112 constipated patients in a community, 516 had undergone anal manometry; 245 (47%) of these (209 women, 36 men) had DD.[159] The age-adjusted incidence per 100,000 person-years was greater ($P < .0001$) in women (31.8; 95% CI, 27.4–36.1) than in men (6.6; 95%, CI, 4.4–8.9). Before diagnosis, nearly 30% of patients had irritable bowel syndrome (IBS), 48% had a psychiatric diagnosis, 18% had a history of abuse, and 21% reported urinary incontinence and/or FI. At tertiary centers, DD prevalence among constipated patients ranges from 20% to 81%.[160,161]

Justification for Changes in Diagnostic Criteria

The term "functional" was removed. "Defecation disorders" is a broad term that includes both functional and structural disorders (eg, rectal prolapse), whereas DD is more specific. Other updates include removal of type 3 dyssynergia and EMG; latter is rarely used clinically.[162]

Many constipated patients report difficult evacuation (eg, straining, use of digital maneuvers, sensation of blockage, and/or incomplete evacuation),[155,163,164] but do not satisfy Rome IV Criteria for functional constipation or IBS. Under Rome V, such patients can be diagnosed with DD if $\geq$1 symptom occurs >25% of the time and physiology criteria are fulfilled. These symptoms are common in DD[163,164] and are associated with prolonged balloon expulsion time.[155,165] The Rome V Criteria for DD broaden the symptom spectrum for DD, recognizing that patients with disordered defecation and increased stool frequency or loose stools who also satisfy objective criteria for dyssynergia respond to biofeedback.[164]

The Rome IV Criteria required abnormalities in $\geq$2 of 3 modalities (ie, balloon expulsion, manometry, and imaging), but concordance among tests was limited.[166] For example, it was unclear whether patients with a prolonged balloon expulsion time but a normal rectoanal gradient (RAG) had DD. A large study (474 constipated patients, 184 healthy participants) observed that high-resolution (HR)-ARM, balloon expulsion test (BET), and defecography findings were concordant, and a reduced RAG, which was often associated with a dyssynergic pattern of defecation, best predicted prolonged BET or reduced rectal evacuation.[167] Hence, the Rome V Criteria propose that any 1 abnormal finding (eg, prolonged BET time, reduced gradient, abnormal pattern, or reduced rectal evacuation by defecography) is sufficient to diagnose DD in patients with difficult evacuation. This modification also avoids unnecessary tests, which may include radiation exposure. Defecography provides supportive evidence when only gradient or BET is abnormal. However, defecography may not be required because an abnormal BET and gradient are 81% and 86% specific, respectively, for predicting reduced rectal evacuation. Hence, patients with 1 abnormal test are classified as probable DD; those with $\geq$2 are classified as definite DD.

Rome V Criteria also specify that an assessment of rectoanal coordination such as the RAG is the best manometry measure of abnormal evacuation. A reduced gradient or defecation index may reflect inadequate rectal propulsive force and/or impaired anal relaxation.[155,157] However, precise criteria for inadequate propulsion and paradoxical contraction vary across techniques[168] and they do not reliably discriminate healthy people from DD patients[169] or predict response to BT.[170] Hence, the Rome V Criteria do not subclassify DD.

Pathophysiology

DD is an acquired behavioral disorder in which patients "unlearn" normal defecation,[153,163] which can often be relearned through BT.[168,170,171]

Physiological factors. Normal defecation is characterized by increased rectal pressure coordinated with relaxation and opening of the anal canal and perineal descent.[153,156,172] In DD, the cardinal abnormality is incoordination characterized by either inadequate rectal propulsive force and/or impaired relaxation or paradoxical contraction of the EAS/puborectalis muscle.[153,172] Earlier studies that were conducted with isolated left lateral[152,155] and seated manometry[173] suggested that patients with DD had an inadequate propulsive force and/or impaired pelvic

F3. Diagnostic Criteria[a] for Dyssynergic Defecation

Must include *all* of the following:

1. The patient reports 1 or more symptoms suggestive of difficult evacuation (ie, excessive straining, use of digital maneuvers to evacuate, sensation of anorectal blockage, and/or feeling of incomplete evacuation) with at least 25% of bowel movements and may satisfy diagnostic criteria for chronic constipation or irritable bowel syndrome.
2. During attempted defecation, they show features of impaired evacuation, as demonstrated by any 1 of the following 3 tests[b]:
   a. Reduced rectoanal pressure gradient or abnormal anorectal evacuation pattern with anorectal manometry
   b. Abnormal balloon expulsion test
   c. Impaired rectal evacuation with defecography

[a]Criteria fulfilled for the last 3 months with symptom onset at least 6 months before diagnosis.
[b]These abnormalities are identified by comparing the data obtained with age- and gender-appropriate normal values for that particular manometry system. Structural abnormalities such as rectocele, rectal mucosal intussusception, and/or rectal sensory disorders may coexist with dyssynergic defecation.

relaxation. However, when pressures and evacuation were assessed simultaneously (ie, proctomanometry), 78% of nonevacuators had global anorectal dysfunction, that is, reduced rectal pressures, pelvic floor relaxation, and puborectalis relaxation during defecation.[172] Volitional control of defecation requires activation of multiple brain regions. Functional MRI shows altered activation of several brain regions (eg, primary and supplementary motor and somatosensory cortices, and emotional arousal networks [hippocampus and prefrontal cortex]) during defecation in patients with DD vs healthy controls.[174] Abnormal colonic motor function[175] may also contribute to DD.

Psychological factors. DD is an acquired behavioral disorder. Withholding behavior in childhood may lead to DD that persists into adulthood, creating a cycle of hard stools, difficult and painful defecation, and stool retention.[176] Patients with DD report more anxiety, depression, paranoid ideation, hostility, and obsessive-compulsive traits than those with slow transit constipation.[177] Psychological distress adversely affects biofeedback outcomes.[178] Approximately 85% of patients with DD have ≥1 additional DGBI (eg, 36% have rectal pain and 41% have functional dyspepsia), which is associated with poorer mental health, QoL, and social functioning.[179] In uncontrolled studies, 22% of women with DD reported sexual abuse.[163,180] In these patients, rectal fullness may trigger traumatic memories and involuntary pelvic floor contraction.[180]

Clinical Evaluation

Key symptoms include excessive straining, hard stools, feeling of incomplete evacuation, use of digital maneuvers, infrequent bowel movements (<3 per week), prolonged time on toilet, and a sensation of anorectal blockage during defecation. DRE findings[1] are critical to the diagnosis.[58,163,181] Bowel diaries overcome the recall bias inherent to questionnaires and interviews.[182,183]

Diagnostic Testing of Patients With Suspected Defecation Disorders

Testing is recommended in patients with suspected DD or laxative-refractory constipation.[66,68,155,184] ARM, BET, magnetic resonance or fluoroscopic defecography, and a colonic transit provide complementary information[59] (Table 2). Guidance on conducting and interpreting ARM is detailed elsewhere.[2,71]

Balloon expulsion test. Patients attempt to expel a water-filled rectal balloon (eg, a commercial balloon) with either 50 mL of water[153,184–186] or inflated until the individual's defecatory desire[184] in privacy and when seated on a commode.[153,184,185] Normal balloon expulsion times range from 20 seconds[186,187] to 2 minutes, although a 1-minute cutoff is recommended.[188] BET results are highly reproducible in 90% of individuals across days.[187,189] Although useful as a screening test, it does not reveal DD pathophysiology and results may be normal in some patients who have constipation and features of DD by defecography or manometry.[155,167]

A rectal expulsion device that mimics Bristol Stool Scale type 4 has been evaluated.[190] In 52 patients, rectal expulsion device in the left lateral position—with abnormal defined as <5 seconds or >120 seconds—predicted response to pelvic floor physical therapy.[190] Seated rectal expulsion devices enhanced the ability to predict response to pelvic floor physical therapy but whether this included biofeedback techniques is unclear.

Anorectal manometry. During ARM, intrarectal and anal pressures are measured during attempted defecation.[2,62,191] Earlier studies used water-perfused or solid-state manometry catheters. Newer options include HR-ARM catheters, which interpolate pressures between multiple circumferential sensors, and air-charged, disposable catheters.[2,62] A normal pattern is characterized by increased intrarectal pressure associated with anal relaxation. An early study identified the following 4 patterns of dyssynergia in 35 constipated patients[153]: type I: adequate propulsion (≥45 mm Hg) with paradoxical anal contraction (Figure 2); type II: inadequate propulsion (<45 mm Hg) with paradoxical contraction or insufficient relaxation; type III: adequate propulsion (≥45 mm Hg) with absent/insufficient anal relaxation (<20%); and type IV: inadequate propulsion (<45 mm Hg) with inadequate relaxation (<20%).[192] Thereafter, a controlled study observed that 87% of healthy participants had "abnormal" patterns suggestive of DD.[169] However, that inference was based on interpretation of data acquired using HR-ARM with (older) normal values assessed with non–high-resolution manometry (HRM) and in the seated position. We now recognize that normal values differ considerably between HRM and non-HRM catheters, as well as among different types of HRM catheters.[2] Even among healthy people with a normal BET time, the 10th–90th percentile range for the RAG during left lateral position HR-ARM is –70 to 12 mm Hg in women and –128 to 1 mm Hg in men.[193] The RAG is abnormal only when it falls below the lower limit of normal. When interpreted relative to sex-appropriate and, in women, age-appropriate, normal values, an abnormal RAG measured with the Manoscan (Medtronic Inc) catheter is 36% sensitive and 85% specific for predicting a prolonged BET time.[167] A lower RAG and prolonged BET time were independently associated with reduced evacuation.[167] Among constipated patients, the probability of reduced rectal evacuation was 14% when both measures were normal, 45% when either was abnormal, and 75% when both variables were abnormal.[167] To conclude, when interpreted rigorously, HR-ARM is a useful test for diagnosing DD. A prolonged BET, reduced gradient, and incomplete evacuation, are each sufficient to diagnose DD.[68,167]

The 4 abnormal patterns associated with DD in constipated patients may be observed in healthy volunteers.[169] In addition, interobserver agreement is substantial for types I and IV dyssynergia, moderate for normal/dyssynergic patterns, and fair for types II and III.[169] Hence, by themselves, patterns may have limited utility.[169]

In clinical practice, manometry is generally performed in the left lateral position. The rectoanal pressures and the

RAG during evacuation were greater in the seated than the left lateral position.[173] Seated HRM is more useful than left lateral HRM for distinguishing between healthy individuals and patients with DD. Ideally, attempted defecation should be performed in the more physiological seated position, although, it can be challenging because of catheter displacement during evacuation.

Anorectal pressures and defecography can now be measured concurrently using fluoroscopy[172] or MRI.[194] Among other findings, these studies disclose that defecation is characterized by the following 2 phases: preparation and evacuation. During the preparatory phase, rectal and anal pressures increased early and concurrently, even in healthy people. Thereafter, the anorectal junction descends, indicating relaxation of the pelvic floor. Once rectal pressure exceeds anal pressure, a positive RAG is established that opens the anal canal and enables rectal evacuation. MRI discloses that normal defecation begins with abdominal wall expansion.[194] Rectal pressure, anorectal descent, and widening of anorectal angle independently predicted rectal evacuation. Most patients have a global disturbance characterized by reduced rectal pressures and pelvic floor relaxation, and/or paradoxical anal or puborectalis contraction during defecation.[172]

Defecography. Defecography evaluates anus and pelvic floor relaxation, rectal evacuation, and structural abnormalities (eg, rectal intussusception) during defecation.[66,68,167,195] Small rectoceles and intrarectal but not external intussusception also occur in asymptomatic controls.[196] Defecography is most useful when ARM and BET are equivocal, discordant with clinical findings or in patients with DD who fail BT.[187,197]

MR defecography shows anorectal motion and rectal evacuation without radiation and provides better resolution than fluoroscopic defecography. Endoanal MRI depicts anal sphincter and levator ani injury. It is helpful in patients with normal BET time to identify structural lesions and guide surgery.[1] MRI disclosed abnormalities in 94% of patients with constipation.[169] However, it is usually performed supine, and may underestimate rectal intussusception compared with seated barium defecography.[1]

Whole gut transit time. Up to two-thirds of patients with DD also have slow colonic transit.[171] Thus, slow colon transit does not exclude the need for anorectal testing. In 1 study, colonic transit improved after BT, suggesting that DD delayed colon transit.[198] Some[199] but not all studies[200] suggest that DD is associated with a selective delay in rectosigmoid transit.

Treatment

Management begins with explanation, education, and lifestyle measures. BT is the mainstay, as it restores bowel function by improving rectoanal coordination (Figure 3).[171,198] BT is ineffective for isolated slow transit constipation.[1,171] A multicomponent BT is preferred[1,171] (Supplementary Table 1).

Large RCTs confirm that BT is effective in both the short term and long term (Table 3).[198,201–209] It is superior to sham feedback, pelvic floor exercises, laxatives, and muscle relaxant drugs.[198,201,202] In 2 RCTs, surgery was superior to BT for improving DD symptoms. However, 1 trial included patients who had symptoms due to structural pelvic floor abnormalities[210] and the other used a suboptimal biofeedback protocol.[204]

Barriers include labor-intensiveness, lack of dedicated equipment and standardized protocol, limited therapist availability, and poor insurance coverage. Potential solutions include training more therapists and home-based BT. Predictors for favorable outcomes include anal digitation,[168] hard stools,[211] and greater patient motivation,[211] undergoing $\geq$5 sessions,[171,212] and prolonged BET.[168,211] Pelvic floor physical therapy is a widely used alternative, although evidence and standardization is lacking and differs significantly from BT.[213] Home-based BT achieves success rates comparable with office BT (68% vs 70%) and at lower cost.[208]

F4. Anorectal Sensory Dysfunction Disorders

The normal process of defecation involves awareness of stool in the rectum that can evoke a desire or an urge to defecate. Maintaining continence also requires normal rectal sensation. The following 2 types of rectal sensory dysfunction disorders have been identified: (1) rectal hyposensitivity and (2) rectal hypersensitivity.

F4a. Rectal Hyposensitivity

Definition

Awareness of stooling is essential for both defecation and maintenance of continence. Rectal hyposensitivity is characterized by abnormal (higher) sensory thresholds to rectal balloon distention, and often associated with chronic constipation, FI, or IBS.

Epidemiology

Rectal hyposensitivity affects up to 25% of patients with chronic constipation.[214,215] It has been reported in 25%–53% of patients with DD,[153,214,215] 28% with slow transit constipation,[215] 17%–22% with constipation-predomination IBS,[214,215] and 12%–19% with FI.[214,216] In a meta-analysis of patients undergoing ARM for FI, the prevalence was 7% in women and 19% in men.[216]

Diagnostic Criteria

Rectal hyposensitivity is defined by 2 or more abnormal thresholds of rectal sensation, such as a first sensation, desire to defecate or urge to defecate, or maximum tolerable sensation. Using this definition, it is present in 14% of patients undergoing ARM, 24% with constipation, and 12% with FI.[214]

Pathophysiology

Physiological factors. Rectal innervation includes visceral afferents (autonomic nervous system), somatic

fibers from the pudendal nerve and inferior hypogastric plexus,[217] and the gut–brain axis comprising chemosensitive and mechanosensitive receptors, mostly unmyelinated C fibers, and some Aδ fibers.[27] Extrinsic sacral afferents synapse in the sacral dorsal root ganglia (S1–S2)[27] project to the spinal cord, thalamus, and sensory cortex. Cortical processing is critical for perception.[27]

Rectal hyposensitivity may be primary or secondary to an enlarged rectum and/or increased rectal compliance.[218,219] Rectal hyposensitivity may be secondary to opioids, Parkinson disease, spinal cord injury, and diabetes[214] or prolonged stool retention.[220] Electrolyte and renal metabolic dysfunction should be excluded before diagnosis.

In patients with IBS and a history of physical and sexual abuse, hyposensitivity may reflect psychological dissociation; "screening out" painful sensations that trigger traumatic memories.[221] Prolonged rectal and anal cortical evoked potentials[222] and altered rectal sensory-motor responses (ie, reflex contraction of the puborectalis associated with desire to defecate)[223] also support impaired anorectal–brain signaling. Large reviews confirm a strong association between constipation and rectal hyposensitivity.[214,215]

Psychological factors. Behavioral patterns such as stool withholding may contribute to hyposensitivity, FI, and constipation.[224] Psychological dysfunction is also strongly associated with rectal sensory disorders.[9,179,214]

Clinical Evaluation

Rectal hyposensitivity may present with symptoms of lack of awareness of stooling, excessive straining, incomplete evacuation, use of digital maneuvers, <3 weekly bowel movements, or prolonged toileting. DRE is important, and if stool is present in the rectum and the patient is unaware, hyposensitivity should be suspected.

Diagnostic testing. Rectal hyposensitivity is identified by rectal sensory testing[191,225] using a handheld syringe or an electronic barostat to distend a rectal balloon. Threshold volumes for first sensation, desire or urgency to defecate, and maximum tolerance are recorded; higher than normal values indicate hyposensitivity.[2,186,226] Because techniques (eg, inflation rate, increments, and volumes) vary, the method should be specified.[191,220,225,227] Rectal compliance,[227] gender, age, and body mass index may affect results.[191,225,227] The barostat method, which uses an infinitely compliant rectal balloon connected to a computer-driven pump, provides reproducible thresholds and accurately measures rectal compliance.[226] Patients should be familiarized with potential sensations, ideally via sensory charts.[191,227] Other modalities to assess rectal sensation include cortical evoked potentials,[78,222] functional MRI,[228] or electrical stimulation.[78]

Treatment. Because rectal hyposensitivity may coexist with stool retention, improving rectal emptying with laxatives, enemas, suppositories, or BT for dyssynergia or FI may help.[171,214,226]

Sensory BT is particularly useful.[43,226,229] Training involves repeated balloon inflations, starting at a volume that induces urge, then reducing by 5–10 mL with each inflation.[43,226,229] Patients are trained to recognize progressively smaller balloon volumes.[171,226] If a sensation is not perceived, the volume is increased slightly, encouraging patients to focus on rectal sensations and not ignore the subtle feelings. This titration helps to establish lower sensory thresholds.

Syringe-assisted sensory BT improves hyposensitivity in DD[226] and in FI,[229] especially in patients with diabetes.[43] In an RCT, a novel barostat-assisted sensory training technique was superior to syringe-assisted training in patients with 2 or more abnormal sensory thresholds.[226] Both methods improved symptoms and were rated equally by patients, but therapists preferred barostat-assisted sensory training. Rectal electrical stimulation also enhanced sensation, whereas biofeedback without sensory training did not,[230] underscoring the need for sensory training.

F4a. Diagnostic Criteria for Rectal Hyposensitivity

Must include *all* of the following:

1. Patients fulfill Rome V symptom criteria[a] for fecal incontinence or dyssynergic defecation
2. Demonstration of rectal hyposensitivity with rectal balloon distention sensory testing. Hyposensitivity is defined as 2 or more threshold values (volume/pressure) of rectal sensation (first sensation, desire to defecate, urgency to defecate, or maximum tolerable sensation) that are greater than 2 SDs of the upper limit of normal range using either a simple balloon (anorectal manometry[b]) or rectal barostat
3. No evidence of structural disease on colonoscopy/barium enema, computed tomography scan, or metabolic abnormalities

[a]Criteria fulfilled for the last 3 months with symptom onset at least 6 months before diagnosis.
[b]When using anorectal manometry balloon system, such as a latex balloon, rectal sensory thresholds can be affected by rectal compliance, and this factor should be considered when interpreting the results. Patients with chronic constipation and IBS may have rectal sensory dysfunction.

F4b. Rectal Hypersensitivity

Definition

Rectal hypersensitivity is defined by lower than normal sensory thresholds to rectal balloon distention and is associated with urgency and/or rectal pain. It encompasses both allodynia (ie, pain from normally nonpainful stimuli) and hyperalgesia (ie, exaggerated pain perception). Hypersensitivity to rectal distention[191,225,231] and electrical and chemical stimulation have been described.[232,233] Rectal hypersensitivity is common in IBS[234,235] and FI.[28,29,42,57] It is associated with urgency, abdominal pain, and bloating and its recognition clarifies symptoms, and supports new treatments and research.

Epidemiology

In a meta-analysis of 13 studies in patients with FI, the pooled prevalence of rectal hypersensitivity was 10% in

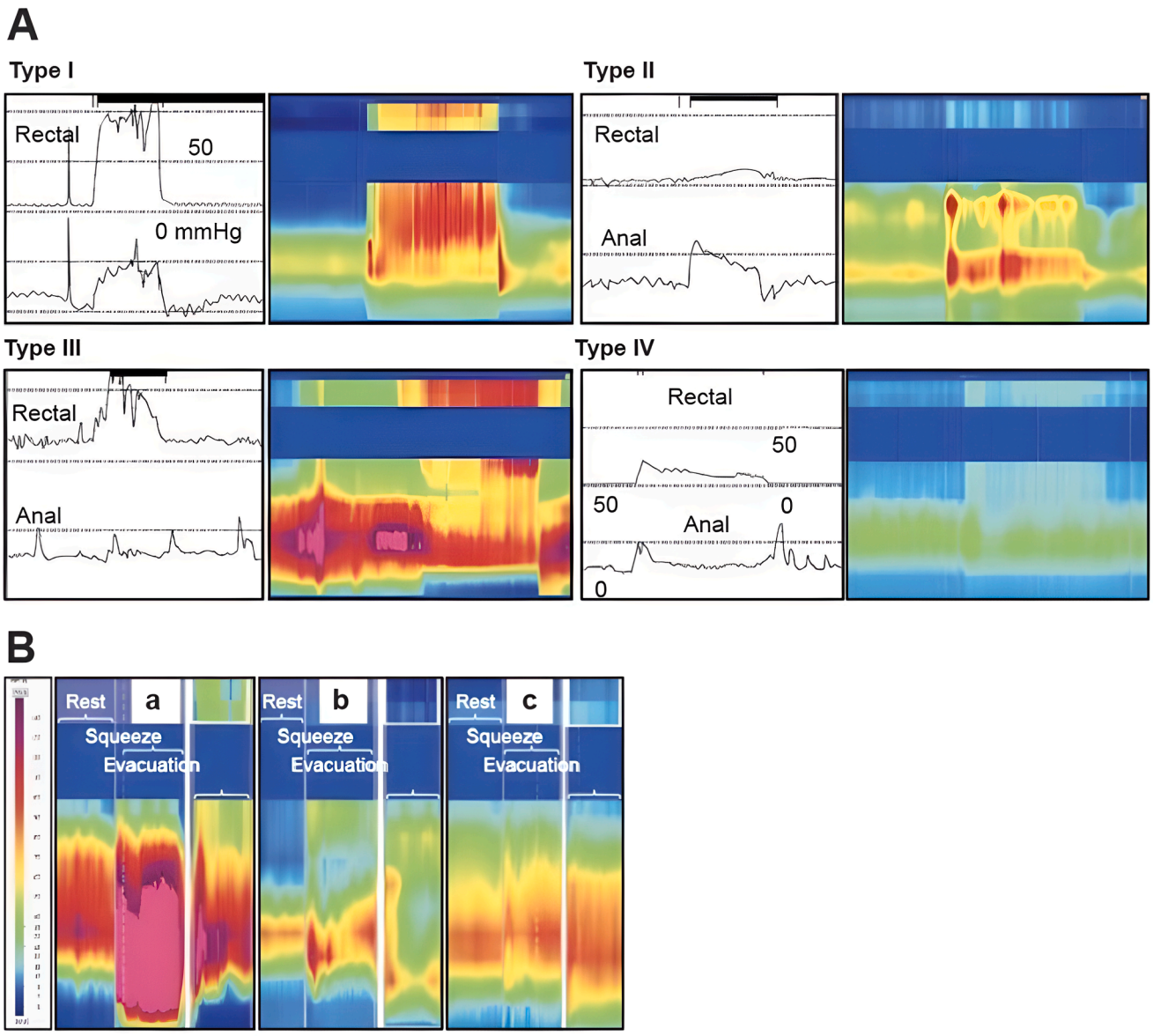

Figure 2. (*A*) The 4 types (I–IV) of DD patterns previously described by Rao et al[1,59,155] are illustrated in conventional wave form manometry tracings as well as in pressure topography. (*B*) The 3 defecatory subtypes based on principal components analysis recently described by Ratuapli et al,[157] (*a*) high anal, (*b*) hybrid, and (*c*) low rectal. From Lee et al,[192] reproduced with permission.

women and 4% in men; rectal hypersensitivity was associated with IBS.[216] We recommend documenting hypersensitivity by at least 1 or more reduced sensory thresholds and, using this definition, 38% of all patients undergoing ARM, 30% with constipation, and 44% with FI, had hypersensitivity.[214] Hypersensitivity to maximum tolerated threshold (or pain) is the most clinically relevant phenotype.[214]

Pathophysiology

Physiologic factors. Rectal hypersensitivity may be primary or secondary to reduced rectal capacity.[45,236] In urge FI, rectal urgency has been associated with reduced rectal capacity and rectal hypersensitivity.[29] Previous infectious sensitization of visceral afferents, especially intraganglonic laminar nerve endings, and alterations in smooth muscle cells, interstitial cells of Cajal, and platelet-derived growth factor receptor α syncytium, may amplify neuronal discharge with luminal distention,[237] causing abdominal and rectal pain, typifying IBS symptoms.[236] Altered gut–brain–gut axis interactions, including activation of the amygdala (emotional) and hippocampus,[238] and hypervigilance, anxiety, depression, and somatization may all contribute.[239]

Psychologic factors. Rectal hypersensitivity is strongly associated with many DGBI, especially IBS.[236,239] Stress, early life adversity (eg, sexual abuse), anxiety, and depression are implicated.[240] Mental stress lowers sensory

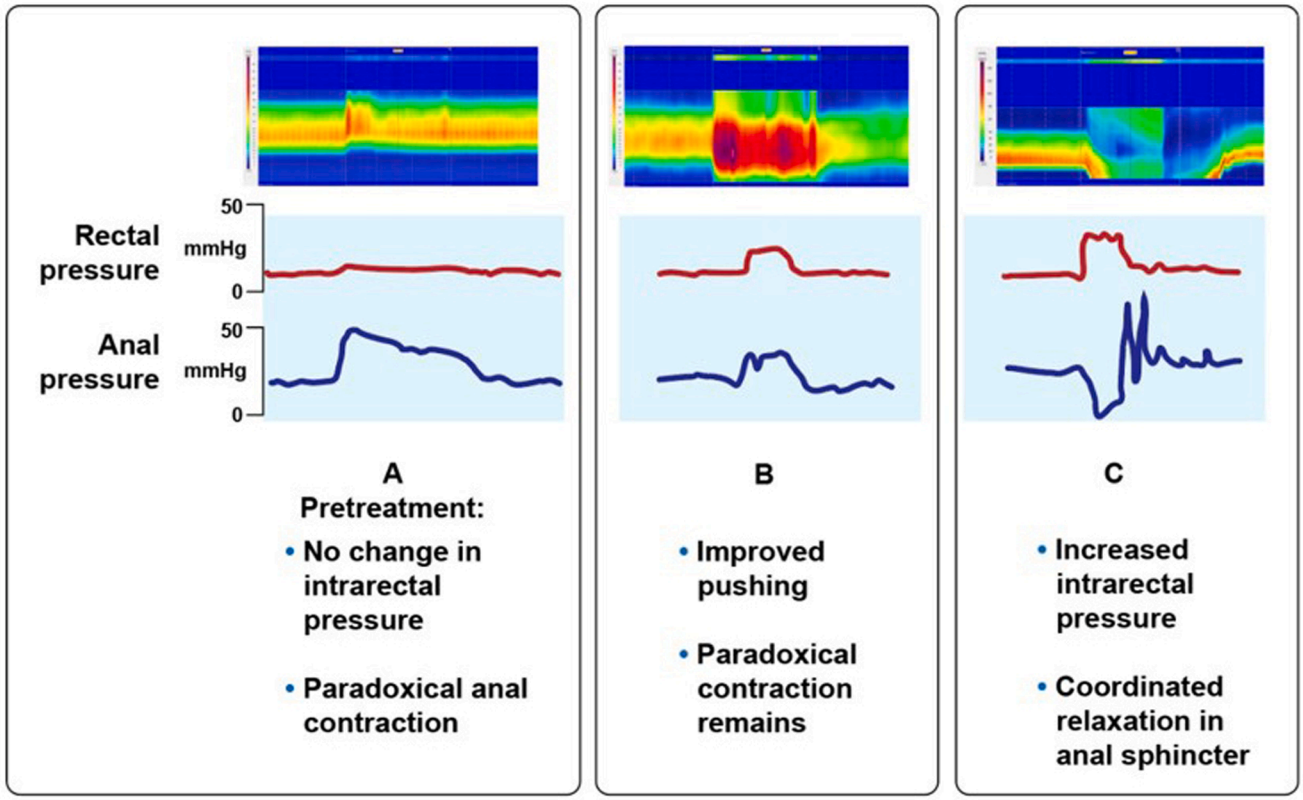

Figure 3. Effects of BT on anorectal pressures and topography in a patient with DD during push effort. (*A*) A patient with type II DD with an inadequate push effort (propulsion) reflected as a flat rectal pressure together with a paradoxical anal contraction (abnormal increase in anal pressure). (*B*) After 2 weeks of BT there is improvement in the rectoanal coordination during the push effort with an increase in rectal pressure activity, but paradoxical anal contraction remains. (*C*) After 6 weeks of BT the dyssynergic pattern has been fully corrected with a significant increase in rectal pressure activity and simultaneous relaxation of anal pressure activity and normal rectoanal coordination.

thresholds to colonic balloon distention in IBS.[241] However, whether psychological dysfunctions are a cause or consequence of hypersensitivity is unclear.

Clinical Evaluation

Patients often present with FI, DD, or IBS accompanied by urgency, lower abdominal pain, or cramps during or after defecation.

Diagnostic testing. Rectal sensation is assessed by rectal balloon distention (see Rectal Hyposensitivity section). Thresholds for first sensation, desire, urgency, discomfort, and/or pain, are recorded. Hypersensitivity is diagnosed when the threshold volumes/pressures are significantly lower than healthy values.[2,186,191,225,226] One study defined hypersensitivity as ≥2 thresholds below 2 SDs of healthy subjects (ie, first [≤20 mL], desire to defecate [≤80 mL], and urgency to defecate or maximum tolerable volume [≤150 mL]).[231] The first sensation threshold is less reliable.[186]

Both ramp and phasic distention protocols are used. An alternative method is to rate perception on a visual analog scale during phasic barostat balloon distentions between 8

F4b. Diagnostic Criteria[a] for Rectal Hypersensitivity

Must include *all* of the following:

1. Patients fulfill Rome V symptom criteria for fecal incontinence or dyssynergic defecation
2. Demonstration of rectal hypersensitivity with rectal balloon distention sensory testing. Hypersensitivity is defined as 1 or more threshold values (volume/pressure) of rectal sensation[b] (desire to defecate, urgency to defecate, maximum tolerable sensation, or pain) that are less than 2 SDs of the lower limit of the normal range using either a simple balloon (anorectal manometry)[c] or rectal barostat.
3. No history of rectal excision or evidence of structural disease, including full thickness rectal prolapse on endoscopy or imaging studies.

[a]Criteria fulfilled for the last 3 months with symptom onset at least 6 months before diagnosis.
[b]Thresholds for first or constant sensation should not be used.
[c]When using anorectal manometry balloon system, such as a latex balloon, rectal sensory thresholds can be affected by rectal compliance, and this factor should be considered when interpreting the results. Patients with chronic constipation and IBS may have rectal sensory dysfunction.

and 32 mm Hg.[242] This allows stimulus intensity to be graded.[220] The association between symptom severity and hypersensitivity was stronger with phasic distentions than ramp distentions.[243] Distention rate and pattern may influence sensory thresholds.[242,243]

Treatment. Possible therapies include hypnotherapy, psychotherapy, secretagogues, antispasmodics, tricyclic antidepressants, selective serotonin reuptake inhibitors, $\alpha 2$ agonists (eg, clonidine), nitrates, and pregabalin.[218,244–246] Sensory adaptation therapy (SAT) consists of intermittent barostat-mediated phasic distentions in 1- to 2-mm Hg increments, starting at the desire to defecate threshold and continuing until pain is reported on 2 consecutive inflations. Sessions are repeated at 1–2 week intervals for 6 sessions over 3 months.[231] In an RCT, SAT but not escitalopram significantly improved hypersensitivity and bowel symptoms.[231] Both treatments improved QoL and psychological profiles.[231] Most pain responders with SAT (80%) were also hypersensitivity responders, and SAT was better tolerated.

Advances in underlying mechanisms, phenotypes, and symptom clusters, together with novel treatments could substantially improve management of rectal sensory disorders.

Recommendations for Future Research

RCTs comparing BT with SNS, home BT, TNT, and perianal injections for FI; development of standardized instruments and protocols of BT for DD; validations of International Anorectal Physiology Working Group protocol; and RCTs of sensory BT are recommended (Supplementary Table 2).

Supplementary Material

Note: To access the supplementary material accompanying this article, visit the online version of *Gastroenterology* at www.gastrojournal.org, and at https://doi.org/10.1053/j.gastro.2026.01.037.

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Correspondence
Address correspondence to: Satish S. C. Rao, MD, PhD, FRCP (LON), Division of Gastroenterology and Hepatology, Augusta University, 1120 15th Street, AD 2226, Augusta, Georgia 30912. e-mail: srao@augusta.edu.

Conflicts of interest
These authors disclose the following: Satish S. C. Rao: advisory board for Neurogut and Biocap and patent for translumbosacral neuromodulation therapy. Adil E. Bharucha: royalties from Minnesota Medical Technologies; joint patents with Medspira and Medtronic; and consulting for Becapio. Leila Neshatian: data safety monitoring board of Cook MyoSite. Jose M. Remes Troche: advisory board for Adium, Biocodex, Carnot, and Prometheus Pharma; and speaker for Adium, Carnot, Menarini, M8, and MD Pharma. The remaining authors disclose no conflicts.

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