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CLINICAL PRACTICE UPDATE

AGA Clinical Practice Update on Evaluation and Management of Belching, Abdominal Bloating, and Distention: Expert Review

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DESCRIPTION: Belching, bloating, and abdominal distention are all highly prevalent gastrointestinal symptoms and account for some of the most common reasons for patient visits to outpatient gastroenterology practices. These symptoms are often debilitating, affecting patients' quality of life, and contributing to work absenteeism. Belching and bloating differ in their pathophysiology, diagnosis, and management, and there is limited evidence available for their various treatments. Therefore, the purpose of this American Gastroenterological Association (AGA) Clinical Practice Update is to provide best practice advice based on both controlled trials and observational data for clinicians covering clinical features, diagnostics, and management considerations that include dietary, gut-directed behavioral, and drug therapies. **METHODS:** This Expert Review was commissioned and approved by the AGA Institute Clinical Practice Updates Committee and the AGA Governing Board to provide timely guidance on a topic of high clinical importance to the AGA membership, and underwent internal peer review by the Clinical Practice Updates Committee and external peer review through standard procedures of *Gastroenterology*. These best practice advice statements were drawn from a review of the published literature based on clinical trials, the more robust observational studies, and from expert opinion. Because systematic reviews were not performed, these best practice advice statements do not carry formal ratings regarding the quality of evidence or strength of the presented considerations.

BEST PRACTICE ADVICE STATEMENTS

BEST PRACTICE ADVICE 1: Clinical history and physical examination findings and impedance pH monitoring can help to differentiate between gastric and supragastric belching. **BEST PRACTICE ADVICE 2:** Treatment options for supragastric belching may include brain–gut behavioral therapies, either separately or in combination, such as cognitive behavioral therapy, diaphragmatic breathing, speech therapy, and central neuromodulators. **BEST PRACTICE ADVICE 3:** Rome IV criteria should be used to diagnose primary abdominal bloating and distention. **BEST PRACTICE ADVICE 4:** Carbohydrate enzyme deficiencies may be ruled out with dietary restriction and/or breath testing. In a small subset of at-risk patients, small bowel aspiration and glucose- or lactulose-based hydrogen breath testing may be used to evaluate for small intestinal bacterial overgrowth. **BEST PRACTICE ADVICE 5:** Serologic testing may rule out celiac disease in patients with bloating and, if serologies are positive, a small bowel biopsy should be done to confirm the diagnosis. A gastroenterology dietitian should be part of the multidisciplinary approach to care for patients with celiac disease and nonceliac gluten sensitivity. **BEST PRACTICE ADVICE 6:** Abdominal imaging and upper endoscopy should be ordered in patients with alarm features, recent worsening symptoms, or an abnormal physical examination only. **BEST PRACTICE ADVICE 7:** Gastric emptying studies should not be ordered routinely for bloating and distention, but may be considered if nausea and vomiting are present. Whole gut motility and radiopaque transit studies should not be ordered unless other additional and treatment-refractory lower gastrointestinal symptoms exist to warrant testing for neuromyopathic disorders. **BEST PRACTICE ADVICE 8:** In patients with abdominal bloating and distention thought to be related to constipation or difficult evacuation, anorectal physiology testing is suggested to rule out a pelvic floor disorder. **BEST PRACTICE ADVICE 9:** When dietary modifications are needed (eg, low–fermentable oligosaccharides, disaccharides, monosaccharides and polyols diet), a gastroenterology dietitian should preferably monitor treatment. **BEST PRACTICE ADVICE 10:** Probiotics should not be used to treat abdominal bloating and distention. **BEST PRACTICE ADVICE 11:** Biofeedback therapy may be effective for bloating and distention when a pelvic floor disorder is identified. **BEST PRACTICE ADVICE 12:** Central neuromodulators (eg, antidepressants) are used to treat bloating and abdominal distention by reducing visceral hypersensitivity, raising sensation threshold, and improving psychological comorbidities. **BEST PRACTICE ADVICE 13:** Medications used to treat constipation should be considered for treating bloating if constipation symptoms are present. **BEST PRACTICE ADVICE 14:** Psychological therapies, such as hypnotherapy, cognitive behavioral therapy, and other brain–gut behavior therapies may be used to treat patients with bloating and distention. **BEST PRACTICE 15:** Diaphragmatic breathing and central neuromodulators are used to treat abdominophrenic dyssynergia.

Keywords: Belching; Bloating; Distention; Gas; Hiccups.

Abbreviations used in this paper: **APD, abdominophrenic dyssynergia; BGBT, brain–gut behavioral therapy; CBT, cognitive behavioral therapy; CD, celiac disease; DGBI, disorder of gut–brain interaction; FD, functional dyspepsia; FODMAP, fermentable oligosaccharides, disaccharides, monosaccharides, and polyols; GERD, gastroesophageal reflux disease; GI, gastrointestinal; GP, gastroparesis; IBS, irritable bowel syndrome; IBS-C, irritable bowel syndrome with constipation; NCGS, nonceliac gluten sensitivity; QOL, quality of life; SIBO, small intestinal bacterial overgrowth; UES, upper esophageal sphincter.**

Most current article

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This American Gastroenterological Association Clinical Practice Update and best practice advice statements describe the definition, clinical features, and treatment for the 3 common symptoms of belching, abdominal bloating, and abdominal distention. When these symptoms are frequent or severe enough to impair daily activities, they are categorized as disorders of gut–brain interaction (DGBIs).[1] The clinical advice herein is evidence-based when data were available, but when insufficient data were available, level 5 evidence is provided on the basis of expert opinion and is empirically based on observational data and expert consensus of the authors.

Why Is This Question Important in Clinical Practice?

These symptoms are highly prevalent, possibly affecting patient quality of life (QOL), work productivity, and visits to emergency and outpatient services.[2-4] Limited information is available for gastroenterologists to find expert advice on diagnosing and managing these DGBI symptoms, as we lack robust evidence because much of the existing data are single-centered and, at times, controversial.

How Much Is Known About This Topic?

Few studies address the pathophysiology or risk factors of belching and bloating, and their treatment options remain suboptimal. Furthermore, these disorders overlap with other common DGBIs, and their mechanisms involve both centrally mediated and peripheral processes. In this Expert Review, we separate belching from bloating and distention, given their differing locations, pathophysiology, and pathways for diagnosis and treatment.

Belching Disorders

Definition

Rome IV defines *belching* as an audible escape of air from the esophagus or the stomach into the pharynx. It is considered a disorder and is referred to as "excessive belching" when it is bothersome enough to disrupt the patient's usual activities and occurs more than 3 days per week.[5] Belching can occur in otherwise healthy individuals. It also may occur with other disorders, including gastroesophageal reflux disease (GERD), functional dyspepsia (FD), gastroparesis (GP), pregnancy, and psychological symptoms, such as anxiety.[6–8] It has been reported in up to 50% of patients with GERD.[9,10] Structural causes of belching include hiatal and paraesophageal hernias and, in patients post Nissen fundoplication, an impaired gastric accommodation can lead to symptoms of belching and dyspepsia.[11]

Belching is subdivided into supragastric belching from the esophagus and gastric belching from the stomach. Supragastric belching occurs in up to 3.4% of patients with upper gastrointestinal (GI) symptoms and is more commonly associated with anxiety.[8] In a global population-based study of more than 73,000 adults, the overall prevalence of Rome IV belching disorders was 1%.[2] Belching is different from aerophagia. With aerophagia, excessive swallowing of air increases intragastric and intestinal gas. This leads to symptoms of bloating, distention, and, less often, belching.[12] Note that in aerophagia, excess air moves to the intestines and colon, therefore, the symptom of flatulence is reported commonly, with bloating as a main manifestation rather than excessive belching alone.[6]

Diagnosis of Belching: Esophageal Physiology Testing Differentiates Belching Syndromes

High-resolution esophageal manometry, if combined with impedance monitoring and impedance pH monitoring, differentiates gastric and supragastric belching and aerophagia. In gastric belching, spontaneous transient relaxation of the lower esophageal sphincter is followed by air transport from the stomach through the esophagus. Gastric belching may be clinically associated with GERD.[9] Then, the upper esophageal sphincter (UES) relaxes and the air is expelled orally.[13] Conversely, in aerophagia, air enters into the esophagus through swallowing, leading to the opening of the UES. Then, as the air clears the esophagus via peristalsis, the lower esophageal sphincter relaxes and the air enters the stomach[6] (Figure 1*A* and *B*).

In contrast, supragastric belching involves 2 separate mechanisms—the air-suction method and the air-injection method.[14] The air-suction method differs from aerophagia and gastric belching, as the air flows through a pressure gradient resulting from UES relaxation. The UES relaxation occurs before the influx of air into the esophagus, in contrast with gastric belching, where the relaxation is a late event. Unlike aerophagia, the supragastric air flow occurs more quickly and is independent of esophageal peristalsis (Figures 2*A* and *B* and 3*A* and *B*). The air-injection method initiates the influx of air into the upper esophagus by means of elevated pharyngeal pressure. This may occur by means of contraction of the base of the tongue rather than a peristaltic contraction of the pharynx, and is not followed by an esophageal peristaltic wave. This latter mechanism is more akin to deliberate belching or burping in healthy individuals and is a learned behavior.

Psychosocial Factors and Other Conditions Influence Belching

The biopsychosocial history should attend to psychosocial triggering factors, including anxiety, life events, and conditioned responses to stressors of physical symptoms. Notably, supragastric belching stops during sleep, distraction, or when the patient speaks.[15,16] This provides evidence that psychological factors modulate the occurrence and frequency of supragastric belching, which may be responsive to brain–gut behavioral therapies (BGBTs), such as cognitive behavioral therapy (CBT). Belching may be conditioned to reduce the bloating sensation via air release, thereby reducing gastric wall tension. Surprisingly, supragastric belching is less common in children than gastric belching when a GERD association is present.[17] Therefore,

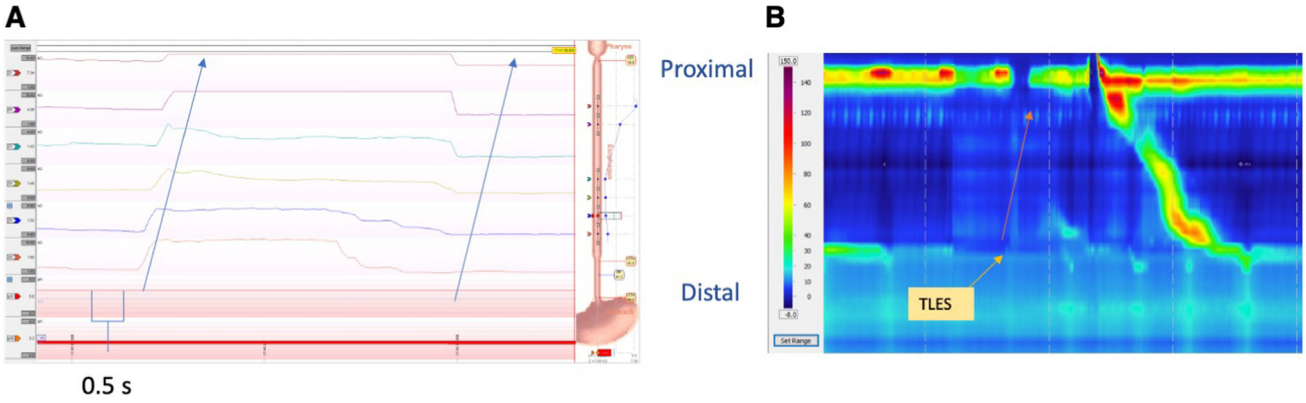

Figure 1. (*A*) Gastric belching demonstrated in contrast to (*B*) with impedance pH study tracing showing instead a distal to proximal increase in impedance with air clearing from the esophagus. *Arrows* indicate direction of air flow (image courtesy of Marcelo F. Vela, MD, FACG, Mayo Clinic, Phoenix, AZ). (*B*) A high-resolution manometric view of gastric belching is shown with direction of air flow from stomach to upper esophagus seen (*orange arrow*), which follows a transient relaxation of lower esophageal sphincter (TLES) shown by *yellow arrow*. The upper esophageal sphincter opens temporarily to allow the air to expel from the esophagus (image courtesy of C. Prakash Gyawali, MD, MRCP, Washington University, St Louis, MO).

behavioral conditioning occurs later in life, as seen with supragastric belching.

Treatment of Belching

Clinicians should first communicate the definition and pathophysiology of gastric and supragastric belching to the patient to establish an understanding and to implement collaborative treatment. Impedance monitoring has helped educate patients, similar to biofeedback therapy for pelvic floor disorders, by objectively demonstrating their physical symptoms as the first step toward treatment when belching is a behavioral disorder and not a consequence of reflux. In belching disorder due to supragastric belching, the reflux episodes are typically nonacidic, which may explain the lack of response to proton pump inhibitors. Recent studies suggested that supragastric belching before reflux activity does not respond to proton pump inhibitor therapy, but supragastric belching after the reflux episodes does.[9]

The most effective suggested treatment for supragastric belching has been behavioral strategies, which include helping the patient become aware of the reasons for their symptoms.[18] Diaphragmatic breathing (see video: https://romedross.video/3azBfEE) increases vagal tone, inducing relaxation and reducing stress response, and is a treatment option for supragastric belching. In addition, belching associated with GERD symptoms improves when diaphragmatic breathing is combined with proton pump inhibitor therapy.[19] Similarly, CBT reduces supragastric belching episodes and esophageal acid exposure, improving QOL.[20]

BGBTs, such as relaxation training and gut-directed hypnotherapy, combined with central neuromodulators

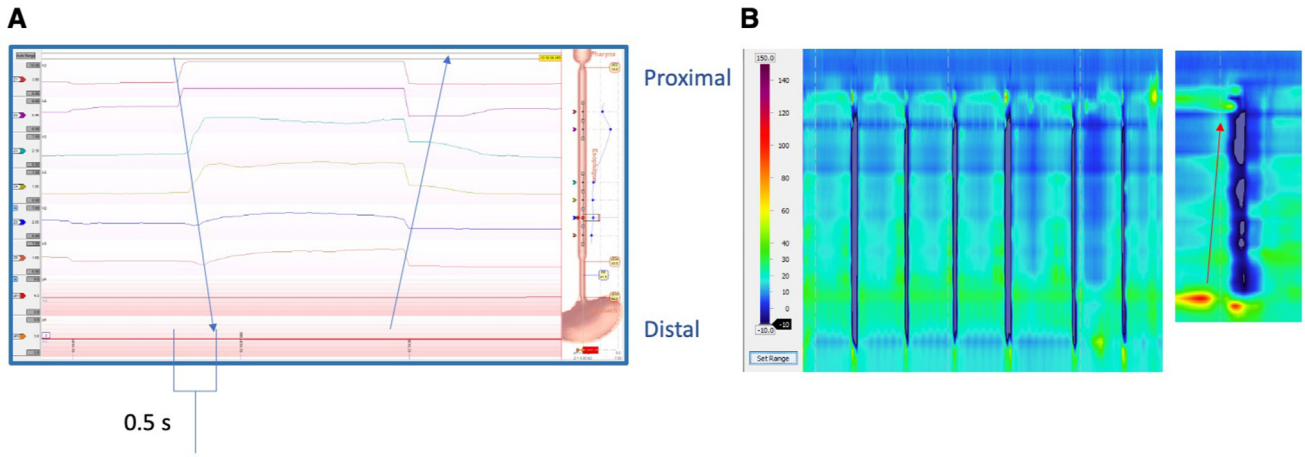

Figure 2. (*A*) Supragastric belching demonstrated in this impedance pH study tracing showing a proximal then distal increase in impedance with air clearing from the esophagus orally. *Arrows* indicate direction of air flow (image courtesy of Marcelo F. Vela, MD, Mayo Clinic, Phoenix, AZ). (*B*) High-resolution manometry showing repetitive supragastic belching with upper esophageal sphincter opening then air propagating through the esophagus, contraction of diaphragm with aborad movement of lower esophageal sphincter. An expanded view is on the *right* (*red arrow* shows direction of air flow) (image courtesy of C. Prakash Gyawali, Washington University, St Louis, MO).

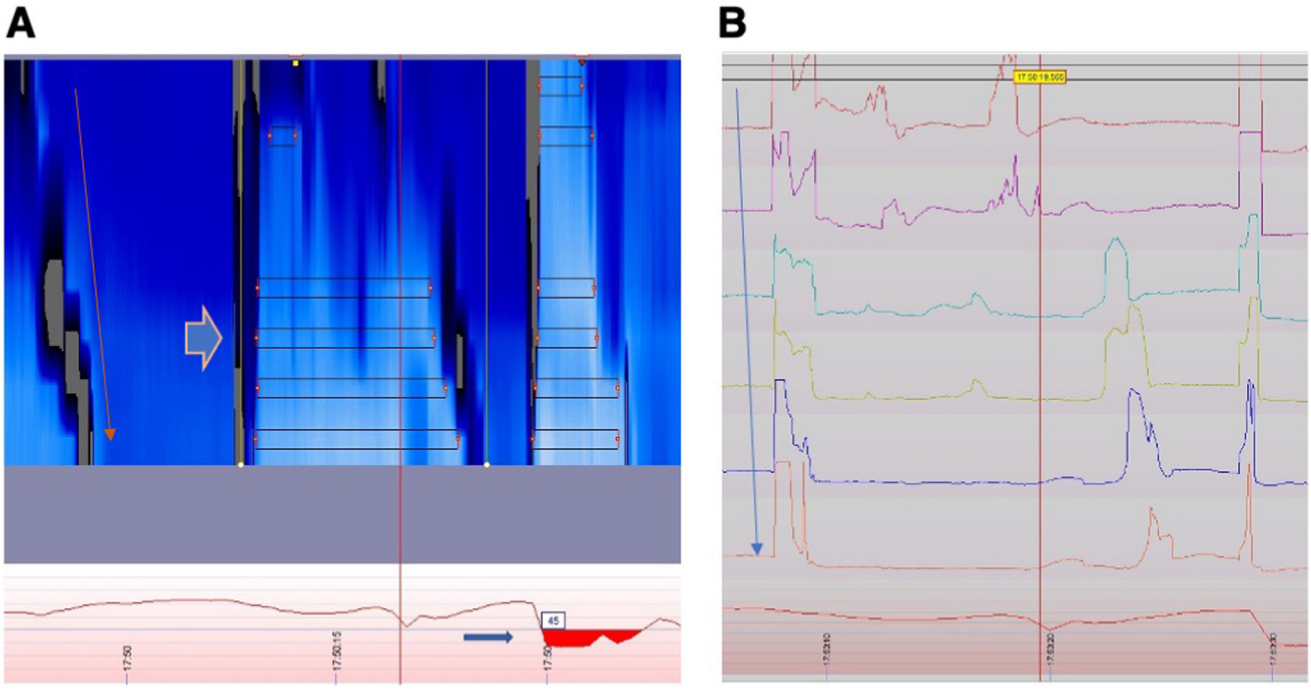

Figure 3. (*A*) The impedance pH image on *left* shows swallowing of air into the esophagus then stomach (see *orange arrow* on *left image*) followed by a supragastric belch (*thick arrow*) then an acid reflux episode follows *(blue arrow* shows pH < 4.0). (*B*) *Line tracing* on *right image* shows the swallow episode (*blue arrow*) seen with swallowing of air.

can improve symptom burden and QOL in patients with belching and other functional esophageal symptoms.[21] In addition, a dedicated speech therapist can treat supragastric belching effectively,[22] as confirmed by our clinical experience. We do not advocate baclofen for use in supragastric belching alone, but it may be considered to prevent lower esophageal sphincter relaxation in those with gastric belching due to GERD.[23,24] Finally, central neuromodulators may be considered to help reduce psychological distress and raise symptom threshold (eg, bloating) that can trigger belching.[25] Because of the different mechanisms of treatment, BGBTs and neuromodulators may be applied in combination (Figure 4).

Abdominal Bloating and Distention

Definition

Abdominal bloating is a subjective sensation in any abdominal region experienced by patients as fullness, swelling, trapped gas or gaseousness, or tightness, and is described as "inflamed" in some cultures. In contrast, abdominal distention is a visible increase in abdominal girth, often described as "like a balloon" or "like being pregnant." These conditions have interrelated pathophysiologies, and usually coexisting treatment strategies are hard to separate. The Rome IV criteria define functional bloating and distention as DGBIs with recurrent symptoms of abdominal fullness or pressure or a visible increase in abdominal girth with symptoms at least 1 day per week and active for 3 months, with onset of 6 months, and without a predominance of pain and alteration in bowel habits.[5] Rome IV has an abdominal bloating and distention category that is separate from other DGBIs, acknowledging that this can be a primary disorder in some patients. A large global population-based study found a prevalence of functional bloating and distention as high as 3.5% (4.6% in women and 2.4% in men).[2] However, bloating and distention are much more prevalent (>50%) when associated with other DGBIs, including irritable bowel syndrome (IBS), constipation, and FD.[26] We will address bloating and abdominal distention as isolated diagnoses and in association with other DGBIs.[27]

Diagnosis

When the Rome IV diagnostic criteria, as defined above for functional abdominal bloating and distention, are met, the patient should not fulfill criteria for a diagnosis of IBS, functional constipation, functional diarrhea, or FD.[5] Because bloating and distention are so prevalent, the Rome IV criteria separate the clinical syndromes from occasional symptoms of bloating and distention. This allows for standardized systematic research and guides the provider to identify which patients should undergo diagnostic testing and treatment.[1,28] Given the multiple etiologies for bloating and distention, diagnostic testing will depend on an algorithmic approach based on presumptive causes, as discussed below (Figure 5).

Common Causes of Bloating and Distention

Food intolerance and hypersensitivity. Carbohydrate enzyme deficiencies (eg, lactase and sucrase), many artificial sweeteners (eg, sugar alcohols and sorbitol),

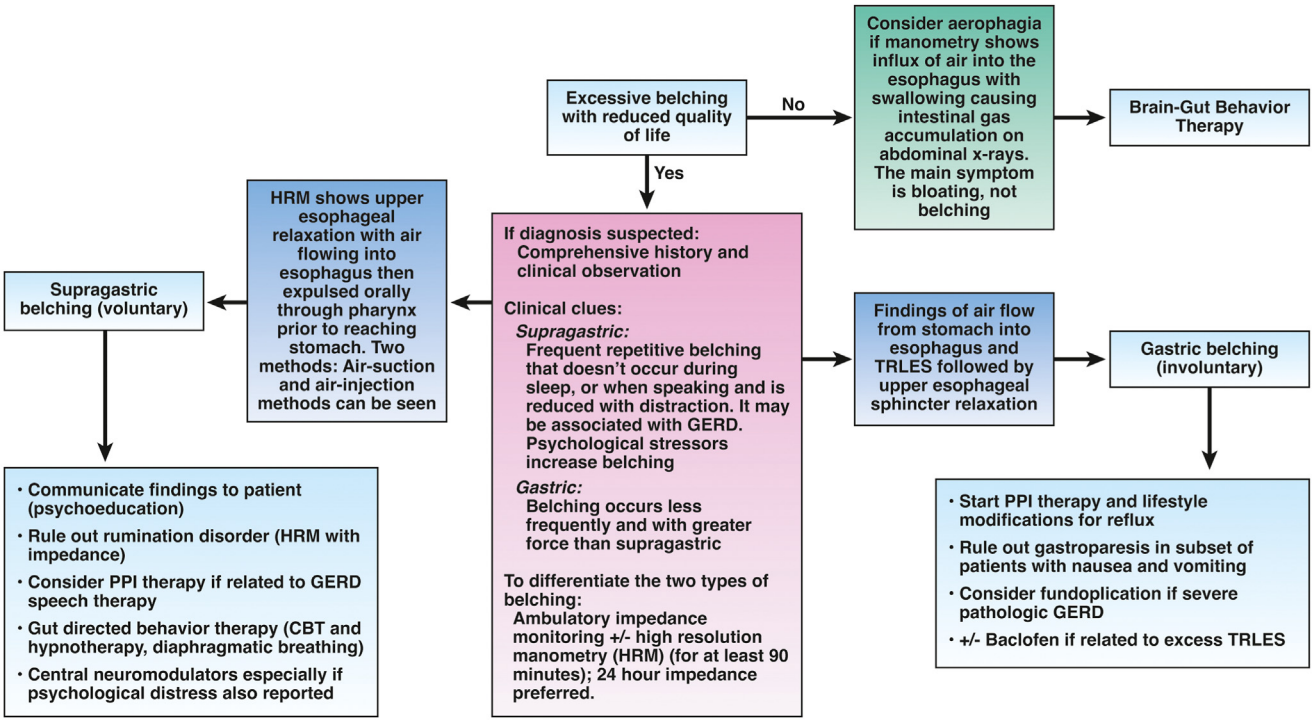

Figure 4. Belching diagnosis and management plan is provided in the algorithm for gastric belching, supragastric belching, and aerophagia. CBT, cognitive behavioral therapy; GERD, gastroesophageal reflux disease; HRM, high resolution manometry; PPI, proton pump inhibitor; TRLES, transient relaxation of lower esophageal sphincter.

and fructans may lead to symptoms of bloating. They are common in the general population, as the undigested sugars have osmotic effects in the colon due to malabsorption from the failure to digest or absorb lactose or sucrose.[29,30] However, not all individuals who malabsorb carbohydrates get symptoms. Those with visceral hypersensitivity (eg, with IBS) are more likely to experience symptoms due to lower sensation thresholds in response to bowel distention.[31] In the largest cohort of patients with DGBIs, and specifically IBS of all subtypes, evaluated to date, fructose intolerance was more common—seen in 60% of patients—and was higher than lactose intolerance (51%), and its prevalence was similar across all major types of DGBIs, except IBS with constipation (IBS-C).[31] The simplest and most economically sound way to diagnose any food intolerance is usually a dietary restriction of short duration (2 weeks), with resolution of symptoms as a positive predictor. Although endoscopic biopsies with enzyme assays are available, use of breath testing, which measures hydrogen, methane, and CO_2, is a better low-cost option, albeit reserved for patients refractory to dietary restrictions first and suspected lactose, fructose, or sucrose intolerances.[32]

When and how to test for small intestinal bacterial overgrowth. Small intestinal bacterial overgrowth (SIBO) is a clinical syndrome seen with many diseases (eg, cystic fibrosis, Parkinson disease, scleroderma, diabetes, and connective tissue diseases) and physiological, DGBI, or nonstructural conditions (ie, IBS, FD, post-surgical motility disorders, and opioid and steroid use).[33,34] Patients with SIBO commonly report bloating[33] and the reference standard for diagnosis is small bowel aspiration and bacterial culture. However, given the cost, invasiveness, and technical difficulties in obtaining these samples, clinicians treat empirically with antibiotics or order lactulose or glucose breath testing.[35] Some consensus guidelines recommend breath testing and establish cutoff criteria within 90 minutes of glucose or lactulose ingestion, but not all experts agree that testing for SIBO is necessary in patients with bloating.[33,35] Increased methane levels represent intestinal methanogen overgrowth, with a primary causative agent being the archaea *Methanobrevibacter smithii* possibly responsible for symptom generation of bloating and distention, especially in the IBS-C population.[36] The designated cutoff times, values, and doses for sugar substrates for the small bowel aspirates and the breath tests are controversial, and the scientific community has not agreed on these parameters, therefore, we do not advocate testing for SIBO in patients with bloating and distention unless clear risk factors or severe symptoms dictate this test-and-treat decision.[33] Although rifaximin is the most studied antibiotic and is a nonabsorbable antibiotic choice, it is also the most expensive. Several systemically absorbed antibiotics have also been studied, including amoxicillin, fluoroquinolones, and metronidazole.[33] Therefore, careful patient selection is needed when treating with this or any other antibiotic medication, as they are not US Food and Drug Administration–approved for the indication of SIBO or

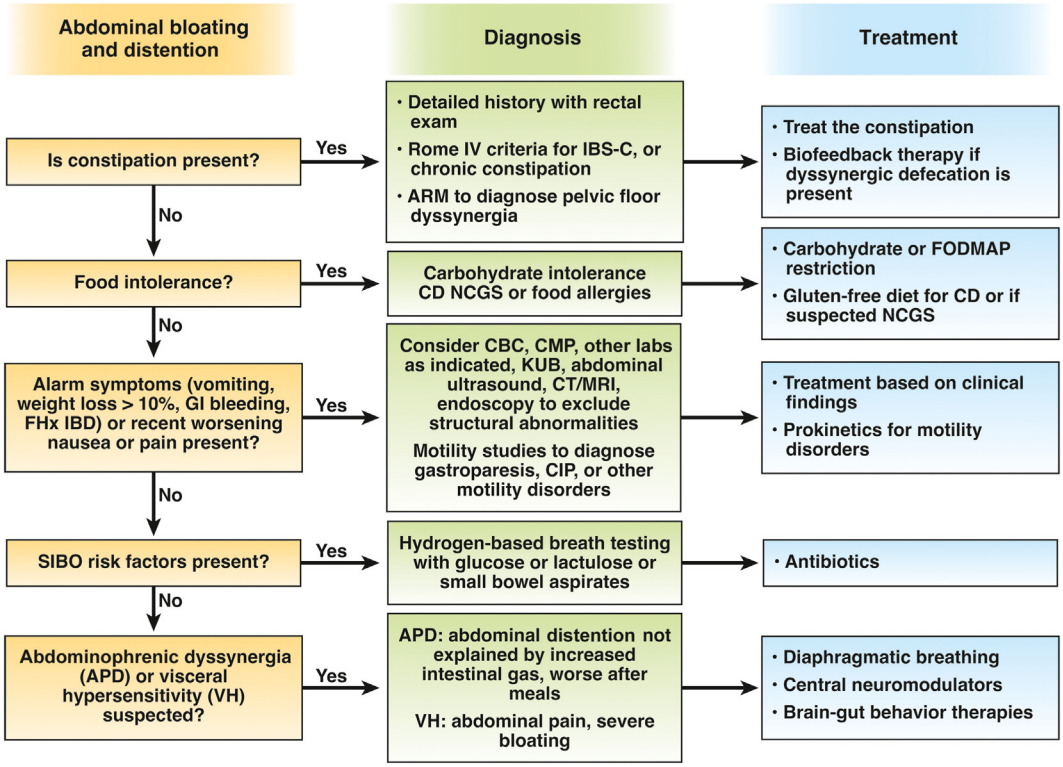

Figure 5. Diagnostic and treatment algorithm for abdominal bloating and distention. NOTE. Diaphragmatic breathing, central neuromodulators, and brain–gut behavioral therapies may be considered for treatment of abdominal bloating and distention regardless of diagnostic correlates. Please refer to manuscript for diagnostic testing warranted based on specific symptoms. APD, abdominophrenic dyssynergia; ARM, anorectal manometry; CBC, complete blood count; CD, celiac disease; CIP, chronic idiopathic intestinal pseudoobstruction; CMP, comprehensive metabolic profile; CT, computed tomography; FHx, family history; FODMAP, fermentable oligosaccharides, disaccharides, monosaccharides, and polyols; GI, gastrointestinal; IBD, inflammatory bowel disease; IBS-C, irritable bowel syndrome with constipation; KUB, kidney, ureter, and bladder X-ray; MRI, magnetic resonance imaging; NCGS, nonceliac gluten sensitivity; SIBO, small intestinal bacterial overgrowth; VH, visceral hypersensitivity.

bloating.[33,36,37] Guidelines for patients with symptom recurrence do not exist. Patients with chronic watery diarrhea, signs of malnutrition and weight loss, and systemic or structural diseases that cause small bowel dysmotility or GI transit delay (eg, cystic fibrosis or Parkinson disease) are at high risk of SIBO and may need diagnostic testing or empiric treatment with antibiotics.

Celiac disease, gluten, and fructans as causes. Patients with celiac disease (CD), nonceliac gluten sensitivity (NCGS), and gluten intolerance experience bloating and distention with or without changes in bowel habits. NCGS is an immune-mediated reaction to gluten or components of fructans. The cornerstone of treatment is the dietary restriction of gluten-containing foods, especially in patients with alarm symptoms, such as weight loss, iron-deficiency anemia, or direct association of ingestion with GI symptoms. Tissue transglutaminase IgA and IgA levels to preclude IgA deficiency is the recommended serologic testing for CD in patients with IBS with diarrhea.[38] The reference standard for diagnosis of CD is a small bowel biopsy confirming the diagnosis if serology is positive before treatment. Alarm symptoms should trigger a small bowel biopsy. In some patients with self-reported NCGS, the fructans in gluten-rich foods rather than gluten cause the symptoms. Thus, the elimination of fructans only is recommended.[39] The availability of a capable gastroenterology dietitian is vital for all of these diseases and syndromes once a diagnosis is confirmed.[40]

Diagnostic testing and who to image or endoscope. Symptoms of bloating and distention do not routinely require laboratory testing or obtaining radiologic imaging or endoscopy unless the history discloses recent worsening symptoms, an abnormal physical examination, or alarm features. We advise ordering tests with worsening dyspepsia or abdominal pain, particularly of recent onset; vomiting; GI bleeding; unintentional weight loss >10% of body weight; chronic diarrhea; or a family history of GI malignancy, CD, or inflammatory bowel disease.

If visible abdominal distention is present, an abdominal examination will help to evaluate for an abdominal mass. The presence of tympany to percussion suggests bowel dilatation. On auscultation, abnormal bowel sounds may suggest obstruction or ileus, and a succussion splash may identify intra-abdominal fluid. Any abnormalities would lead to a computed tomography scan or ultrasound of the abdomen to evaluate for ascites or a mass or to identify increased bowel gas due to ileus, obstruction, or pseudo-obstruction. Bloating and abdominal fullness are often presenting symptoms in patients with ovarian cancer; the highest risk is in women 50 years or older.[41] However, in

the absence of alarm symptoms, the yield of clinically meaningful findings from these tests is low.

Occasionally, an abdominal x-ray reveals increased stool burden and suggests further evaluation for slow transit constipation or a pelvic floor disorder for patients with functional constipation, IBS-mixed (diarrhea and constipation), or IBS-C with severe constipation.[38] In addition, an upper endoscopy may be considered in patients older than 40 years with dyspeptic symptoms and abdominal bloating or distention, mainly if occurring in a geographic region with a high prevalence of *Helicobacter pylori*.[42] Another etiology of bloating usually accompanied by pain may be chronic pancreatitis, despite adequate pancreatic enzyme replacement warranting fecal elastase testing first.[43]

Motility disorders. Patients with FD or GP have symptoms of bloating and fullness, and scintigraphy often cannot differentiate the 2 disorders.[44,45] Approximately 40% of patients with GP in the National Institutes of Health GP registry reported bloating that correlated with nausea, abdominal fullness, and abdominal pain ($P < .05$), and none of these symptoms correlate with the degree of gastric emptying delay per scintigraphy.[46] The National Institute of Diabetes and Digestive and Kidney Disease Gastroparesis Clinical Consortium authors stated, "FD and GP may be part of the same clinicopathological spectrum of gastric neuromuscular dysfunction."[44,45] Hence, we cannot advocate gastric scintigraphy or wireless motility capsule studies for evaluation of bloating or distention alone, but these tests should be considered in patients with severe nausea or vomiting presumed to be due to delayed gastric emptying or in patients with the postprandial FD subtype based on the National Institute of Diabetes and Digestive and Kidney Disease Consortium and European consensus on FD and GP.[42,45,47] In addition, severe constipation is present in >30% of patients with symptoms of severe GP and is associated with delayed small bowel and colonic transit but not with gastric emptying delay.[48] Therefore, in a small subset of patients with refractory upper GI symptoms, including severe bloating and distention, especially combined with weight loss, and those suspected of having intestinal neuromyopathic disorders based on other supporting history, small bowel motility evaluation with antroduodenal manometry; wireless motility capsule; whole gut scintigraphy; and/or radiopaque markers may elucidate extragastric dysmotility that could respond to prokinetics.[49] Importantly, many of these specialized tests, such as antroduodenal manometry, are not standardized and require referral to tertiary care centers in patients with severe symptoms refractory to standard treatments and malnutrition.

Evaluating anorectal disorders. Patients with functional defecation disorders, such as dyssynergic defecation, frequently experience constipation and bloating. This may relate to visceral hypersensitivity (as with IBS) and a retained stool load with colon distention. Evaluation begins with a complete history emphasizing bowel habits, such as straining even with soft stool or throughout the evacuation, digital disimpaction, or splinting. Fecal incontinence may even occur secondary to a large fecal load in the rectum relaxing the sphincter muscle. Other structural causes may lead to anorectal symptoms and obstructive defecation.

A digital rectal examination helps to identify increased or decreased sphincter muscle tone, pelvic floor dyssynergia, rectal prolapse, anal stricture or a rectocele. A diagnosis of pelvic floor dyssynergia should be confirmed by means of anorectal physiology testing combined with balloon expulsion. Defecography with barium or magnetic resonance imaging can be done if there is concern for a structural etiology contributing to the symptoms, such as pelvic organ prolapse or rectal intussusception in patients with rectal pain or a large rectocele or cystocele. Both the American College of Gastroenterology and British Society of Gastroenterology advocate anorectal physiology testing, especially for women with IBS-C not responding to standard therapies or who have suspected pelvic floor disorders based on history or examination findings.[38,50]

Treatments for Bloating and Abdominal Distention

Given the lack of scientific evidence for treatments for abdominal bloating and distention and their limited success, patients may resort to possibly detrimental and unscrupulous resources propagated through social media. They may also identify providers who promote ineffective fad diets and herbal therapies, which can lead to malnutrition or potential toxicity. Especially for patients with DGBI, effective communication improves the patient–provider relationship, leading to better health outcomes, less strain on the health care system by avoiding unnecessary urgent care visits, and improved patient satisfaction.[28,51] Once the provider understands the biopsychosocial model and can educate patients on brain–gut interactions, treatments such as diet, biofeedback therapy, central neuromodulators, and psychotherapies can be incorporated.

Dietary interventions for bloating and distention. Foods may trigger bloating and abdominal distention, especially in patients with overlapping DGBIs.[52,53] However, few studies have evaluated dietary restriction for primary abdominal bloating and distention. The most investigated dietary recommendations have been the exclusion of gluten in patients with NCGS and CD, fructan avoidance, and initiation of the low–fermentable oligosaccharides, disaccharides, monosaccharides and polyols (FODMAP) diet in those sensitive to them.[54–56] A small study of patients who met Rome II criteria for functional bloating and gas-related symptoms, fructose intolerance was the most common finding based on breath testing; 65% of patients had carbohydrate malabsorption and dietary restriction led to improvement of symptoms in >80% at 1 month and complete improvement in 50% at 1 year.[54] Recent studies have suggested that fructans, rather than gluten, may cause symptoms in those with NCGS.[39] Although the low-FODMAP diet has not been evaluated for treating functional bloating and distention explicitly, bloating and QOL improvements have been reported in randomized controlled trials when comparing the low-FODMAP diet

with traditional dietary advice in FD and IBS.[56–58] Because the low-FODMAP diet may have potential negative impacts on the gut microbiome, with a decrease in *Bifidobacterium* species and malnutrition, its implementation with plans for a reintroduction should be done in the hands of a trained gastroenterology dietitian or a trained gastroenterology provider exclusively.[59] In synergy with dietary restrictions, a careful recognition of risk factors for eating disorders and avoidant or restrictive food intake disorder should be made, preferably with the help of a gastroenterology psychologist or well-informed clinician.[60] If an eating disorder is identified, dietary restrictions should be tailored to avoid malnutrition. In general, if an elimination diet is not beneficial, it should be discontinued.

Treatment with probiotics and medical foods is not recommended for bloating or distention. No studies have examined the efficacy of probiotics in specifically treating bloating and distention. One double-blind placebo-controlled trial of 2 separate probiotics with *Bifidobacterium lantis* and *Lactobacillus acidophilus* showed improvements in global GI symptoms of patients with DGBI at 8 weeks vs placebo, with improvements in bloating symptoms ($P < .01$) achieved.[61] However, the newest British, European, and American guidelines for IBS and FD have not endorsed the use of probiotics to treat global symptoms in these conditions.[38,50,62] Probiotics may be associated with developing new onset of brain fogginess, bloating, and lactic acidosis. There is currently insufficient data supporting their use for any DGBI, including bloating.[63] In the United States, peppermint oil is the most studied herbal remedy for global IBS symptoms. A recent placebo-controlled randomized controlled trial in IBS found no improvement in bloating symptoms with peppermint oil at the 6-week end point.[64] Although peppermint oil is commonly used because of minimal adverse effects, further studies are needed to document its benefit in bloating and distention.

Anorectal biofeedback therapy may help bloating and distention. Bloating and abdominal distention are key symptoms in patients with IBS-C and chronic constipation. Because these conditions overlap with dyssynergic defecation, anorectal biofeedback therapy is often recommended, with the expectation that improving pelvic floor function will reduce bloating symptoms.[38] Anorectal biofeedback uses an instrument-based "operant-conditioning" technique, when evacuation coordination occurs via a visual monitor that demonstrates the results of anorectal push and relaxation, which promotes normal defecation.[65] One study in Italy reported a high prevalence of disordered defecation in patients with diet-refractory bloating. Treatment led to a responder rate of 54% for bloating scores decreased by 50%.[66] The response rates to biofeedback therapy in those with IBS-C and chronic constipation are similarly favorable and long-lasting based on RCTs, with improvements in abdominal distention, rectal hypersensitivity, and bloating.[67,68] Because these tests and their therapists are not widely available, home-based biofeedback therapy alternatives and point-of-care anorectal function testing may aid easy application in an office setting without access to a motility specialist.[68,69] We propose that biofeedback therapy is effective for bloating disorder when an evacuation disorder is identified.

Central neuromodulators for bloating and distention. Bloating is an uncomfortable sensation that results from multiple disturbed mechanisms along the gut–brain axis. The symptom may result from visceral hypersensitivity to impaired central down-regulation of incoming visceral signals.[70] These sensations may also be amplified by the psychological state when visceral anxiety, depression, or somatization coexist.[71] Central neuromodulators (eg, antidepressants) reduce the perception of incoming visceral signals, re-regulate brain–gut dysregulated control mechanisms, and improve psychological comorbidities.[25] Antidepressants that activate noradrenergic and serotonergic pathways, including tricyclic antidepressants (eg, amitriptyline) and the serotonin–norepinephrine reuptake inhibitors (eg, duloxetine and venlafaxine) show the greatest benefit in reducing visceral sensations. Many studies have shown improvement for bloating, pain, and global symptoms for various DGBIs, including IBS, FD, functional heartburn, and nausea and vomiting.[72–74] Central neuromodulators, such as pregabalin, have also shown improvements in bloating in patients with IBS.[75] A few other studies have evaluated the effect of central neuromodulators specifically on bloating in the context of associated DGBIs, such as IBS, centrally mediated abdominal pain, or GP, and have shown symptom benefits.[18,25,49] Based on our clinical experience, abdominal distention improves with central neuromodulators by reducing the bloating sensation that triggers the distention via that abnormal viscerosomatic reflex. The benefit seems to work best when the distention occurs during or after a meal.[25,28,76]

Gut-related medications that treat constipation may help bloating symptoms. Several studies have used bloating as a secondary outcome measure and reported their benefit over placebo. These include secretagogues (eg, lubiprostone, linaclotide, and plecanatide), a 5-hydroxytryptamine 4 receptor agonist (eg, tegaserod, recently removed from the market), and a sodium-hydrogen exchanger-3 agent (eg, tenapanor) specifically for IBS-C.[77–83] A recent meta-analysis using 13 trials found all medications superior to placebo for treating abdominal bloating in patients with IBS-C.[84] Results of an indirect comparison of all the drugs showed no differences among these medications. A selective 5-hydroxytryptamine 4 receptor agonist, prucalopride, is used to treat GP and constipation.[85,86] Results of pooled analyses from constipation trials showed a number needed to treat of 8, with moderate to severe bloating improvements.

Brain–gut behavioral therapies for bloating. A recently published multidisciplinary consensus report provides evidence to support various BGBTs to treat DGBI.[18] These therapies, including hypnotherapy, CBT, and other modalities, may be combined with central neuromodulators and other GI treatments in a safe and complementary fashion. In addition, these treatments do not need to be symptom-specific, as they improve the overall QOL parameters, anxiety, stress, and burden associated with DGBIs. A

meta-analysis evaluating the efficacy of psychological therapies in IBS found the most robust evidence with CBT and gut-directed hypnotherapy, however, all therapies were effective in IBS. A risk of bias was noted in many of the trials.[87] To date, none of the BGBTs have focused on functional bloating only; however, empiric evidence based on the consensus of experts supports their use, in the least, to reduce psychological distress and improve QOL. In addition, prescription-based psychological therapies are now US Food and Drug Administration–approved for use on smart apps. Furthermore, because these therapies improve global symptoms that include bloating in IBS and FD, and they are safe and relatively inexpensive, we suggest BGBT for treating symptoms of bloating and distention.[18]

Abdominophrenic Dyssynergia as a Basis for Bloating and Distention

Abdominophrenic dyssynergia (APD) describes a paradoxical viscerosomatic reflex response to minimal gaseous distention in subjects with symptoms of bloating and marked abdominal distention. Healthy individuals react to increased intestinal gas by contracting their anterior abdominal muscles and relaxing the diaphragm, thereby preventing abdominal distention. With APD, the diaphragm paradoxically contracts (goes down), and the anterior abdominal wall muscles relax, leading to abdominal distention.[88] In patients with functional bloating or IBS, computed tomography scans demonstrate the APD response with even small increases in intraluminal gas (approximately 10%, insufficient to explain the degree of abdominal distention) and visceral hypersensitivity.[89] In contrast, patients with severe intestinal dysmotility with large amounts of intestinal gaseous distention (eg, intestinal pseudo-obstruction) did not exhibit an APD pattern with similar symptoms of distention and bloating. For many patients, the APD distention occurs during or immediately after a meal.[76] This suggests that gastric and intestinal distention leads to bloating, which triggers the viscerosomatic reflex and the APD response. In this context, our experience indicates that central neuromodulators reduce abdominal distention by reducing the bloating sensation, thereby reducing the triggering mechanism for the APD. This does not seem to work well when the bloating is constant or unrelated to meals.[28]

Biofeedback therapy for APD using electromyography of muscle activity (diaphragm and intercostal muscles) has been proposed, with a benefit reported in 2 studies by 1 group.[90] Additional studies by other investigators are needed to confirm this benefit and determine its applicability.

Diaphragmatic breathing, which reduces vagal tone and sympathetic activity, may be used to treat APD. For example, in patients with IBS, slow deep breathing intervention leads to improvements in autonomic response assessed by exercise heart rate recovery and heart rate variability.[91] Still, more data are needed, although expert consensus from brain–gut behavioral therapists and neurogastroenterologists report improvement in patient symptoms, and this method is inexpensive and safe (for further information on APD please see the video at: https://romedross.video/Q_A_AbdomDyssyn).

Conclusions

Symptoms of belching, abdominal bloating, and distention are common and, when experienced frequently and bothersome, leading to impairment of patients' daily activities, they are categorized as a DGBI. Although many gaps exist in understanding these symptoms, their pathophysiology seems to be converging on the importance of dysregulation of the brain–gut axis and the application of the biopsychosocial model for treatment that addresses diet, motility visceral sensitivity, and psychosocial parameters. The limited number of well-designed studies makes evidence-based treatment recommendations difficult. Therefore, we must extrapolate data from other DGBIs where these symptoms may coexist and rely on empiric evidence to identify treatments supported by clinical experience. We believe a multidisciplinary approach and a patient-centered model are keys to managing treatment in patients with belching, abdominal bloating, and distention. Integrated care involving gastroenterologists, gastroenterology dietitians, brain–gut behavioral therapists, and motility providers may not be available in all settings. Careful attention to the patients' primary symptoms, physical examination, and limited diagnostic studies can help to navigate patients toward the proper diagnostic evaluation. Furthermore, education and effective communication skills using a patient-centered care model will optimize treatment with improved outcomes and increased patient and provider satisfaction and reduce unneeded diagnostic testing and health care costs.[28,51,92]

Supplementary References

Note: To access the supplementary references numbered 26 to 92 accompanying this article, visit the online version of *Gastroenterology* at www.gastrojournal.org, and at https://doi.org/10.1053/j.gastro.2023.04.039.

References

1. Drossman DA. Functional gastrointestinal disorders: history, pathophysiology, clinical features and Rome IV. Gastroenterology 2016;150:1282–1279.
2. Sperber AD, Bangdiwala SI, Drossman DA, et al. Worldwide prevalence and burden of functional gastrointestinal disorders, results of Rome Foundation global study. Gastroenterology 2021;160:99–114.
3. Drossman DA, Li Z, Andruzzi E, et al. US householder survey of functional gastrointestinal disorders. Prevalence, sociodemography, and health impact. Dig Dis Sci 1993;38:1569–1580.
4. Peery AF, Crockett SD, Murphy CC, et al. Burden and cost of gastrointestinal, liver, and pancreatic diseases in the United States: update 2018. Gastroenterology 2019;156:254–272.
5. Stanghellini V, Chan FK, Hasler WL, et al. Gastroduodenal disorders. Gastroenterology 2016;150:1380–1392.

6. Bredenoord AJ, Weusten BL, Timmer R, et al. Air swallowing, belching, and reflux in patients with gastroesophageal reflux disease. Am J Gastroenterol 2006; 101:1721–1726.
7. Piessevaux H, De Winter B, Louis E, et al. Dyspeptic symptoms in the general population: a factor and cluster analysis of symptom groupings. Neurogastroenterol Motil 2009;21:378–388.
8. Koukias N, Woodland P, Yazaki E, et al. Supragastric belching: prevalence and association with gastroesophageal reflux disease and esophageal hypomotility. J Neurogastroenterol Motil 2015;21:398–403.
9. Jeong SO, Lee JS, Lee TH, et al. Characteristics of symptomatic belching in patients with belching disorder and patients who exhibit gastroesophageal reflux disease with belching. J Neurogastroenterol Motil 2021; 27:231–239.
10. Hemmink GJ, Bredenoord AJ, Weusten BL, et al. Supragastric belching in patients with reflux symptoms. Am J Gastroenterol 2009;104:1992–1997.
11. Pauwels A, Boecxstaens V, Broers C, et al. Severely impaired gastric accommodation is a hallmark of post-Nissen functional dyspepsia symptoms. Neurogastroenterol Motil 2017;29:e13063.
12. Hemmink GJM, Weusten BLAM, Bredenoord AJ, et al. Aerophagia: excessive air swallowing demonstrated by esophageal impedance monitoring. Clin Gastroenterol Hepatol 2009;7:1127–1129.
13. Dent J, Holloway RH, Toouli J, et al. Mechanisms of lower oesophageal sphincter incompetence in patients with symptomatic gastrooesophageal reflux. Gut 1988; 29:1020–1028.
14. Kessing BF, Bredenox AJ, Smout AJPM. The pathophysiology, diagnosis and treatment of excessive belching symptoms. Am J Gastroenterol 2014; 109:1196–1203.
15. Karamanolis G, Triantafyllou K, Tsiamoulos Z, et al. Effect of sleep on excessive belching: a 24-hour impedance-pH study. J Clin Gastroenterol 2010; 44:332–334.
16. Bredenoord AJ, Weusten BL, Timmer R, et al. Psychological factors affect the frequency of belching in patients with aerophagia. Am J Gastroenterol 2006; 101:2777–2781.
17. Masui D, Nikaki K, Sawada A, et al. Belching in children: prevalence and association with gastroesophageal reflux disease. Neurogastroenterol Motil 2022;34:e14194.
18. Keefer L, Ballou SK, Drossman DA, et al. A Rome working team report on brain-gut behavior therapies for disorders of gut-brain interaction. Gastroenterology 2022;162:300–315.
19. Ong AM, Chua LT, Khor CJ, et al. Diaphragmatic breathing reduces belching and proton pump inhibitor refractory gastroesophageal reflux symptoms. Clin Gastroenterol Hepatol 2018;16:407–416.e2.
20. Glasinovic E, Wynter E, Arguero J, et al. Treatment of supragastric belching with cognitive behavioral therapy improves quality of life and reduces acid gastroesophageal reflux. Am J Gastroenterol 2018;113:539–547.
21. Hurtte E, Rogers BD, Richards C, et al. The clinical value of psycho-gastroenterological interventions for functional esophageal symptoms. Neurogastroenterol Motil 2022;34:e14315.
22. Hemmink GJ, Ten Cate L, Bredenoord AJ, et al. Speech therapy in patients with excessive supragastric belching—a pilot study. Neurogastroenterol Motil 2010; 22:24–28.e2–e3.
23. Blondeau K, Boecxstaens V, Rommel N, et al. Baclofen improves symptoms and reduces postprandial flow events in patients with rumination and supragastric belching. Clin Gastroenterol Hepatol 2012;10:379–384.
24. Curcic J, Schwizer A, Kaufman E, et al. Effects of baclofen on the functional anatomy of the oesophagogastric junction and proximal stomach in healthy volunteers and patients with GERD assessed by magnetic resonance imaging and high-resolution manometry: a randomised controlled double-blind study. Aliment Pharmacol Ther 2014;40:1230–1240.
25. Drossman DA, Tack J, Ford AC, et al. Central neuromodulators for functional gastrointestinal disorders (disorders of gut-brain interaction): a Rome Foundation working team report. Gastroenterology 2018;154:1140–1171.

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Baha Moshiree: Study conception and design, drafting of manuscript, and critical revision of the manuscript. Douglas Drossman: Study conception and design, drafting of manuscript, and critical revision of the manuscript. Aasma Shaukat: Study conception and critical revision of the manuscript.

Conflicts of interest
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Supplementary References

26. Palsson OS, Simren M, Tack J, et al. Bloating and distension: inherent characteristics of irritable bowel syndrome (IBS) and functional dyspepsia (FD)? Poster presented at: Digestive Disease Week 2022; May 21–24, 2022; San Diego, CA.
27. Lacy BE, Cagnemi D, Vazquez-Roque M. Management of chronic abdominal distension and bloating. Clin Gastroenterol Hepatol 2021;19:219–231.
28. Drossman D, Ruddy J. Gut Feelings: Disorders of Gut-Brain Interaction and the Patient-Doctor Relationship. Drossman Care, 2021.
29. Misselwitz B, Butter M, Verbeke K, et al. Update on lactose malabsorption and intolerance: pathogenesis, diagnosis and clinical management. Gut 2019; 68:2080–2091.
30. Storey D, Lee A, Bornet F, et al. Gastrointestinal tolerance of erythritol and xylitol ingested in a liquid. Eur J Clin Nutr 2007;61:349–354.
31. Wilder-Smith CH, Materna A, Wermelinger C, et al. Fructose and lactose intolerance and malabsorption testing: the relationship with symptoms in functional gastrointestinal disorders. Aliment Pharmacol Ther 2013; 37:1074–1083.
32. Amieva-Balmori M, Coss-Adame E, Rao NS, et al. Diagnostic utility of carbohydrate breath tests for SIBO, fructose, and lactose intolerance. Dig Dis Sci 2020; 65:1405–1413.
33. Quigley EMM, Murray JA, Pimentel M. AGA clinical practice update on small intestinal bacterial overgrowth: expert review. Gastroenterology 2020;159:1526–1532.
34. Pimentel M, Saad RJ, Long MD, et al. ACG clinical guideline: small intestinal bacterial overgrowth. Am J Gastroenterol 2020;115:165–178.
35. Rezaie A, Buresi M, Lembo A, et al. Hydrogen and methane-based breath testing in gastrointestinal disorders: the North American Consensus. Am J Gastroenterol 2017;112:775–784.
36. Pimentel M, Lembo A, Chey WD, et al; TARGET Study Group. Rifaximin therapy for patients with irritable bowel syndrome without constipation. N Engl J Med 2011; 364:22–32.
37. Gatta L, Scarpignato C. Systematic review with meta-analysis: rifaximin is effective and safe for the treatment of small intestine bacterial overgrowth. Aliment Pharmacol Ther 2017;45:604–616.
38. Lacy BE, Pimentel M, Brenner DM, et al. ACG clinical guideline: management of irritable bowel syndrome. Am J Gastroenterol 2021;116:17–44.
39. Skodje GI, Sarna VK, Minelle IH, et al. Fructan, rather than gluten, induces symptoms in patients with self-reported non-celiac gluten sensitivity. Gastroenterology 2018;154:529–539.
40. Scarlata K, Eswaran S, Baker JR, et al. Utilization of dietitians in the management of irritable bowel syndrome by members of the American College of Gastroenterology. Am J Gastroenterol 2022;117:923–926.
41. Runowicz CD. Ovarian cancer: not-so-silent. A swollen abdomen, urinary symptoms, and bloating can be warning signs of ovarian cancer, which afflicts 1 in 57 women in the US. Health News 2004;10:12.
42. Moshiree B, Talley NJ. Functional dyspepsia: a critical appraisal of the European consensus from a global perspective. Neurogastroenterol Motil 2021;33:e14216.
43. Moshiree B, Freeman AJ, Vu PT, et al; GALAXY Study Group. Multicenter prospective study showing a high gastrointestinal symptom burden in cystic fibrosis. J Cyst Fibros 2023;22:266–274.
44. Pasricha PJ, Grover M, Yates KP, et al; National Institute of Diabetes and Digestive and Kidney Diseases/National Institutes of Health Gastroparesis Clinical Research Consortium. Functional Dyspepsia and Gastroparesis in Tertiary Care are Interchangeable Syndromes With Common Clinical and Pathologic Features. Gastroenterology 2021;160:2006–2017.
45. Tack J, Schol J, Horowitz M. Gastroparesis: a dead-end street after all? Gastroenterology 2021;160:1931–1933.
46. Hasler WL, Wilson LA, Parkman HP, et al; NIDDK Gastroparesis Clinical Research Consortium (GpCRC). Bloating in gastroparesis: severity, impact, and associated factors. Am J Gastroenterol 2011;106:1492–1502.
47. Camilleri M, Kuo B, Nguyen L, et al. ACG clinical guideline: gastroparesis. Am J Gastroenterol 2022; 117:1197–1220.
48. Parkman HP, Sharkey E, McCallum RW, et al; NIH/NIDDK Gastroparesis Consortium. Constipation in patients with symptoms of gastroparesis: analysis of symptoms and gastrointestinal transit. Clin Gastroenterol Hepatol 2022;20:546–558.
49. Hasler WL, May KP, Wilson LA, et al; NIDDK Gastroparesis Clinical Research Consortium (GpCRC). Relating gastric scintigraphy and symptoms to motility capsule transit and pressure findings in suspected gastroparesis. Neurogastroenterol Motil 2018;30:e13196.
50. Vasant DH, Paine PA, Black CJ, et al. British Society of Gastroenterology guidelines on the management of irritable bowel syndrome. Gut 2021;70:1214–1240.
51. Drossman DA, Chang L, Deutsch JK, et al. A review of the evidence and recommendations on communication skills and the patient-provider relationship: a Rome Foundation Working Team Report. Gastroenterology 2021;161:1670–1688.e7.
52. Chey WD, Tack J. The role of food in disorders of gut-brain interaction: introduction to a Rome Foundation Working Group Series. Am J Gastroenterol 2022; 117:935–936.
53. Tack J, Tornblom H, Tan V, et al. Evidence-based and emerging dietary approaches to upper disorders of gut-brain interaction. Am J Gastroenterol 2022;117:965–972.
54. Fernández-Bañares F, Rosinach M, et al. Sugar malabsorption in functional abdominal bloating: a pilot study on the long-term effect of dietary treatment. Clin Nutr 2006;25:824–831.
55. Biesiekierski JR, Newnham ED, Irving PM, et al. Gluten causes gastrointestinal symptoms in subjects without celiac disease: a double-blind randomized placebo-controlled trial. Am J Gastroenterol 2011;106:508–514.
56. Chey WD, Hashash JG, Manning L, et al. AGA clinical practice update on the role of diet in irritable bowel

syndrome: expert review. Gastroenterology 2022; 162:1737–1745.e5.
57. Goyal O, Nohria S, Batta S, et al. Low fermentable oligosaccharides, disaccharides, monosaccharides, and polyols diet versus traditional dietary advice for functional dyspepsia: a randomized controlled trial. J Gastroenterol Hepatol 2022;37:301–309.
58. Böhn L, Störsrud S, Liljebo T, et al. Diet low in FODMAPs reduces symptoms of irritable bowel syndrome as well as traditional dietary advice: a randomized controlled trial. Gastroenterology 2015;149:1399–1407.e2.
59. Staudacher HM, Scholz M, Lomer MC, et al. Gut microbiota associations with diet in irritable bowel syndrome and the effect of low FODMAP diet and probiotics. Clin Nutr 2021;40:1861–1870.
60. Murray HB, Doerfler B, Harer KN, et al. psychological considerations in the dietary management of patients with DGBI. Am J Gastroenterol 2022;117:985–994.
61. Ringel-Kulka T, Palsson OS, Maier D, et al. Probiotic bacteria *Lactobacillus acidophilus* NCFM and *Bifidobacterium lactis* Bi-07 versus placebo for the symptoms of bloating in patients with functional bowel disorders: a double-blind study. J Clin Gastroenterol 2011; 45:518–525.
62. Wauters L, Dickman R, Drug V, et al; ESNM FD Consensus Group. United European Gastroenterology (UEG) and European Society for Neurogastroenterology and Motility (ESNM) consensus on functional dyspepsia. United European Gastroenterol J 2021;9:307–331.
63. Rao SSC, Rehman A, Yu S, et al. Brain fogginess, gas and bloating: a link between SIBO, probiotics and metabolic acidosis. Clin Transl Gastroenterol 2018; 9:162.
64. Nee J, Ballou S, Kelley JM, et al. Peppermint oil treatment for irritable bowel syndrome: a randomized placebo-controlled trial. Am J Gastroenterol 2021; 116:2279–2285.
65. Sharma A, Herekar A, Yan Y, et al. Dyssynergic defecation and other evacuation disorders. Gastroenterol Clin North Am 2022;51:55–69.
66. Iovino P, Neri MC, D'Alba L, et al. Pelvic floor biofeedback is an effective treatment for severe bloating in disorders of gut-brain interaction with outlet dysfunction. Neurogastroenterol Motil 2022;34:e14264.
67. Patcharatrakul T, Gonlachanvit S. Outcome of biofeedback therapy in dyssynergic defecation patients with and without irritable bowel syndrome. J Clin Gastroenterol 2011;45:593–598.
68. Rao SS, Valestin JA, Xiang X, et al. Home-based versus office-based biofeedback therapy for constipation with dyssynergic defecation: a randomized controlled trial. Lancet Gastroenterol Hepatol 2018;3:768–777.
69. Chey WD, Baker JR, Watts L, et al. Development of a simple, point-of-care device to test anorectal function in patients with constipation: randomized clinical trial. Clin Gastroenterol Hepatol 2023;21:832–834.
70. Malagelada JR, Accarino A, Azpiroz F. Bloating and abdominal distension: old misconceptions and current knowledge. Am J Gastroenterol 2017;112:1221–1231.
71. Keefer L, Drossman DA, Guthrie E, et al. Centrally mediated disorders of gastrointestinal pain. Gastroenterology 2016;150:1408–1419.
72. Tack J, Janssen P, Masaoka T, et al. Efficacy of buspirone, a fundus-relaxing drug, in patients with functional dyspepsia. Clin Gastroenterol Hepatol 2012; 10:1239–1245.
73. Tack J, Ly HG, Carbone F, et al. Efficacy of mirtazapine in patients with functional dyspepsia and weight loss. Clin Gastroenterol Hepatol 2016;14:385–392.e4.
74. Talley NJ, Locke GR, Saito YA, et al. Effect of amitriptyline and escitalopram on functional dyspepsia: a multicenter, randomized controlled study. Gastroenterology 2015;149:340–349.e2.
75. Saito YA, Almazar AE, Tilkes KE, et al. Randomised clinical trial: pregabalin vs placebo for irritable bowel syndrome. Aliment Pharmacol Ther 2019;49:389–397.
76. Livovsky DM, Barber C, Barba E, et al. Abdominothoracic postural tone influences the sensations induced by meal ingestion. Nutrients 2021;13:658.
77. Chang L, Chey WD, Drossman D, et al. Effects of baseline abdominal pain and bloating on response to lubiprostone in patients with irritable bowel syndrome with constipation. Aliment Pharmacol Ther 2016; 44:1114–1122.
78. Chey WD, Lembo AJ, Lavins BJ, et al. Linaclotide for irritable bowel syndrome with constipation: a 26-week, randomized, double-blind, placebo-controlled trial to evaluate efficacy and safety. Am J Gastroenterol 2012; 107:1702–1712.
79. Rao S, Lembo AJ, Shiff SJ, et al. A 12-week, randomized, controlled trial with a 4-week randomized withdrawal period to evaluate the efficacy and safety of linaclotide in irritable bowel syndrome with constipation. Am J Gastroenterol 2012;107:1714–1724.
80. Chey WD, Lembo AJ, Yang Y, et al. Efficacy of tenapanor in treating patients with irritable bowel syndrome with constipation: a 26-week, placebo-controlled phase 3 trial (T3MPO-2). Am J Gastroenterol 2021; 116:1294–1303.
81. Tack J, Müller-Lissner S, Bytzer P, et al. A randomised controlled trial assessing the efficacy and safety of repeated tegaserod therapy in women with irritable bowel syndrome with constipation. Gut 2005; 54:1707–1713.
82. Brenner DM, Fogel R, Dorn SD, et al. Efficacy, safety, and tolerability of plecanatide in patients with irritable bowel syndrome with constipation: results of two phase 3 randomized clinical trials. Am J Gastroenterol 2018; 113:735–745.
83. Chang L, Sultan S, Lembo A, et al. AGA clinical practice guideline on the pharmacological management of irritable bowel syndrome with constipation. Gastroenterology 2022;163:118–136.
84. Nelson AD, Black CJ, Houghton LA, et al. Systematic review and network meta-analysis: efficacy of licensed drugs for abdominal bloating in irritable bowel syndrome with constipation. Aliment Pharmacol Ther 2021; 54:98–108.

85. Carbone F, Van den Houte K, Clevers E, et al. Prucalopride in gastroparesis: a randomized placebo-controlled crossover study. Am J Gastroenterol 2019;114:1265–1274.
86. Staller K, Hinson J, Kerstens R, et al. Efficacy of prucalopride for chronic idiopathic constipation: an analysis of participants with moderate to very severe abdominal bloating. Am J Gastroenterol 2022;117:184–188.
87. Black CJ, Thakur ER, Houghton LA, et al. Efficacy of psychological therapies for irritable bowel syndrome: systematic review and network meta-analysis. Gut 2020;69:1441–1451.
88. Villoria A, Azpiroz F, Burri E, et al. Abdomino-phrenic dyssynergia in patients with abdominal bloating and distension. Am J Gastroenterol 2011;106:815–819.
89. Accarino A, Perez F, Azpiroz F, et al. Abdominal distention results from caudo-ventral redistribution of contents. Gastroenterology 2009;136:1544–1551.
90. Barba E, Accarino A, Azpiroz F. Correction of abdominal distention by biofeedback-guided control of abdominothoracic muscular activity in a randomized, placebo-controlled trial. Clin Gastroenterol Hepatol 2017;15:1922–1929.
91. Jurek MK, Seavey H, Guidry M, et al. The effects of slow deep breathing on microvascular and autonomic function and symptoms in adults with irritable bowel syndrome: a pilot study. Neurogastroenterol Motil 2022;34:e14275.
92. Cangemi DJ, Lacy BE. A practical approach to the diagnosis and treatment of abdominal bloating and distension. Gastroenterol Hepatol (N Y) 2022;18:75–84.