

# NARRATIVE REVIEW

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## Management of Chronic Abdominal Distension and Bloating

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Abdominal bloating and distension are 2 of the most commonly reported gastrointestinal symptoms. Abdominal bloating is characterized by symptoms of trapped gas, abdominal pressure, and fullness. Abdominal distension is defined as a measurable increase in abdominal girth. These symptoms frequently co-exist, although they can occur separately. Defined by Rome IV criteria, functional abdominal bloating and distension commonly coincide with other functional gastrointestinal disorders, such as functional dyspepsia, irritable bowel syndrome, and functional constipation. Abdominal bloating and distension can develop for multiple reasons, including food intolerances, a previous infection that perturbed the intestinal microbiota, disordered visceral sensation, delayed intestinal transit, or an abnormal viscerosomatic reflex. Treatment can be challenging to patients and providers—no regimen has been consistently successful. Successful treatment involves identifying the etiology, assessing severity, educating and reassuring patients, and setting expectations. Therapeutic options include dietary changes, probiotics, antibiotics, prokinetic agents, antispasmodics, neuromodulators, and biofeedback. We review the epidemiology and effects of chronic bloating and distension and pathophysiology, discuss appropriate diagnostic strategies, and assess available treatment options.

**Keywords:** IBS; Pain; FGID; Therapy.

Nearly all of the population has felt, at one time or another, gassy, bloated, or distended. For many, these are transient sensations that occur after eating, resolve spontaneously, and do not lead to medical consultation. For others, however, abdominal bloating and distension are chronic, bothersome, and negatively affect daily life. The prevalence of bloating and distension is substantial, ranging from 16% to 31% in the general population, and as high as 66%–90% in patients with irritable bowel syndrome (IBS).<sup>1–6</sup> Women generally report higher rates of bloating than men, while patients with IBS with constipation (IBS-C) have higher rates than those with IBS with diarrhea.<sup>5,7–10</sup>

The impact of chronic abdominal bloating and distension on quality of life is substantial.<sup>11</sup> Seventy-five percent of patients with bloating (without IBS) characterize their symptoms as moderate to severe in nature, while 50% report that symptoms cause a reduction in daily activities.<sup>2</sup> The economic impact of chronic bloating and distension has not been well studied.

### Abdominal Bloating and Distension: Definition and Symptoms

Abdominal bloating is the subjective sensation of gassiness, trapped gas, or a feeling of pressure or being distended without obvious visible distension. Patients also describe a sense of fullness or pressure, which can occur anywhere in the abdomen (epigastric, mid, lower, or throughout). Abdominal distension is the objective physical manifestation of an increase in abdominal girth. Patients commonly describe how they look “like a balloon” or “like I’m pregnant.” Abdominal impedance plethysmography has shown that abdominal girth increases during the course of the day in healthy volunteers, and then returns to baseline levels overnight.<sup>12</sup> Abdominal bloating and distension can occur independently, although they frequently co-exist. One study found that only 50%–60% of patients with bloating report abdominal distension, thus highlighting the distinct nature of these disorders.<sup>13</sup> Patients with chronic functional bloating and distension may be diagnosed using the Rome criteria (see Table 1).<sup>7</sup> Of note, neither symptom is required to be present for a patient to meet Rome criteria for IBS or functional constipation although bloating and distension are frequently present in both disorders and are noted as supporting criteria.

### Pathophysiology

The etiology for chronic abdominal bloating and distension is complex, often multifactorial in nature, and incompletely understood. The differential diagnosis includes both organic and functional disorders (see Table 2). Most patients believe that their symptoms are due to an increased amount of “gas” within the gastrointestinal (GI) tract, although this accounts for symptoms in only a minority of patients. Normal gas production, absorption, and excretion are illustrated in Figure 1.

**Abbreviations used in this paper:** BT, breath test; CIC, chronic idiopathic constipation; CT, computed tomography; FD, functional dyspepsia; FGID, functional gastrointestinal disorder; GI, gastrointestinal; IBS, irritable bowel syndrome; IBS-C, irritable bowel syndrome with constipation; SIBO, small intestinal bacterial overgrowth.

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**Table 1.** Functional Abdominal Bloating and Distension<sup>7</sup>

Diagnostic criteria for functional abdominal bloating and/or distension include:

- Recurrent bloating and/or distention occurring at least 1 d/wk on average;
- Bloating and distension should be the predominant gastrointestinal symptom;
- Patients should not meet criteria for irritable bowel syndrome, functional constipation, functional diarrhea, or postprandial distress syndrome;
- Symptom onset should have occurred at least 6 months prior to diagnosis;
- Symptoms should be active within the preceding 3 months.

Computed tomography (CT) imaging has shown that luminal gas increases in only 25% of patients with functional gastrointestinal disorders (FGIDs) during a spontaneous episode of abdominal distension or following consumption of a “high-flatulence” diet.<sup>14</sup> The following sections highlight major pathophysiologic causes of bloating and distension (see [Figure 2](#)).

### *Small Intestinal Bacterial Overgrowth and Carbohydrate Intolerance*

Small intestinal bacterial overgrowth (SIBO) and carbohydrate (eg, lactose and fructose) intolerance are common causes of chronic bloating and distension. Excess small intestine bacteria can cause symptoms due to carbohydrate fermentation with subsequent gas production and stretch and distension of the small intestine. Altered sensation and an abnormal viscerosomatic reflex may also play a role although these mechanisms have not been well studied in patients with SIBO. Carbohydrate intolerance may cause symptoms of bloating and distension due to an increased osmotic load, excess fluid retention, and excess fermentation in the colon. The lack of consensus regarding an ideal test to diagnose SIBO makes it difficult to ascertain its true prevalence. In addition, no prospective trial has evaluated patients diagnosed solely with chronic bloating and distension to determine the prevalence of SIBO or food intolerances, and thus most data come from the best-studied FGID, IBS. A meta-analysis reported the prevalence of SIBO to be 0%–20% in healthy control subjects vs 4%–78% in patients with IBS.<sup>15</sup> The prevalence of food intolerance, which has similar symptoms, in the general population approaches 20%.<sup>16</sup> The true prevalence of carbohydrate intolerance is unclear as carbohydrate intolerance does not necessarily correspond with carbohydrate malabsorption by breath test. One prospective study of symptomatic patients with various FGIDs (n = 1372) identified a prevalence of lactose intolerance and malabsorption of 51% and 32%, respectively, and a prevalence of fructose intolerance and malabsorption of 60% and 45%, respectively.<sup>17</sup> Lactase deficiency by itself may not cause malabsorption, as not all individuals who

**Table 2.** Common Causes of Chronic Bloating and Distension

#### **Organic/pathologic etiologies**

- Small intestinal bacterial overgrowth
- Lactose, fructose, and other carbohydrate intolerances
- Celiac disease
- Pancreatic insufficiency
- Prior gastroesophageal surgery (eg, fundoplication, bariatric surgery)
- Gastric outlet obstruction
- Gastroparesis
- Ascites
- Gastrointestinal or gynecologic malignancy
- Hypothyroidism
- Adiposity
- Small intestine diverticulosis
- Chronic intestinal pseudo-obstruction

#### **Disorders of gut-brain interaction**

- Irritable bowel syndrome
- Chronic idiopathic constipation
- Pelvic floor dysfunction
- Functional dyspepsia
- Functional bloating

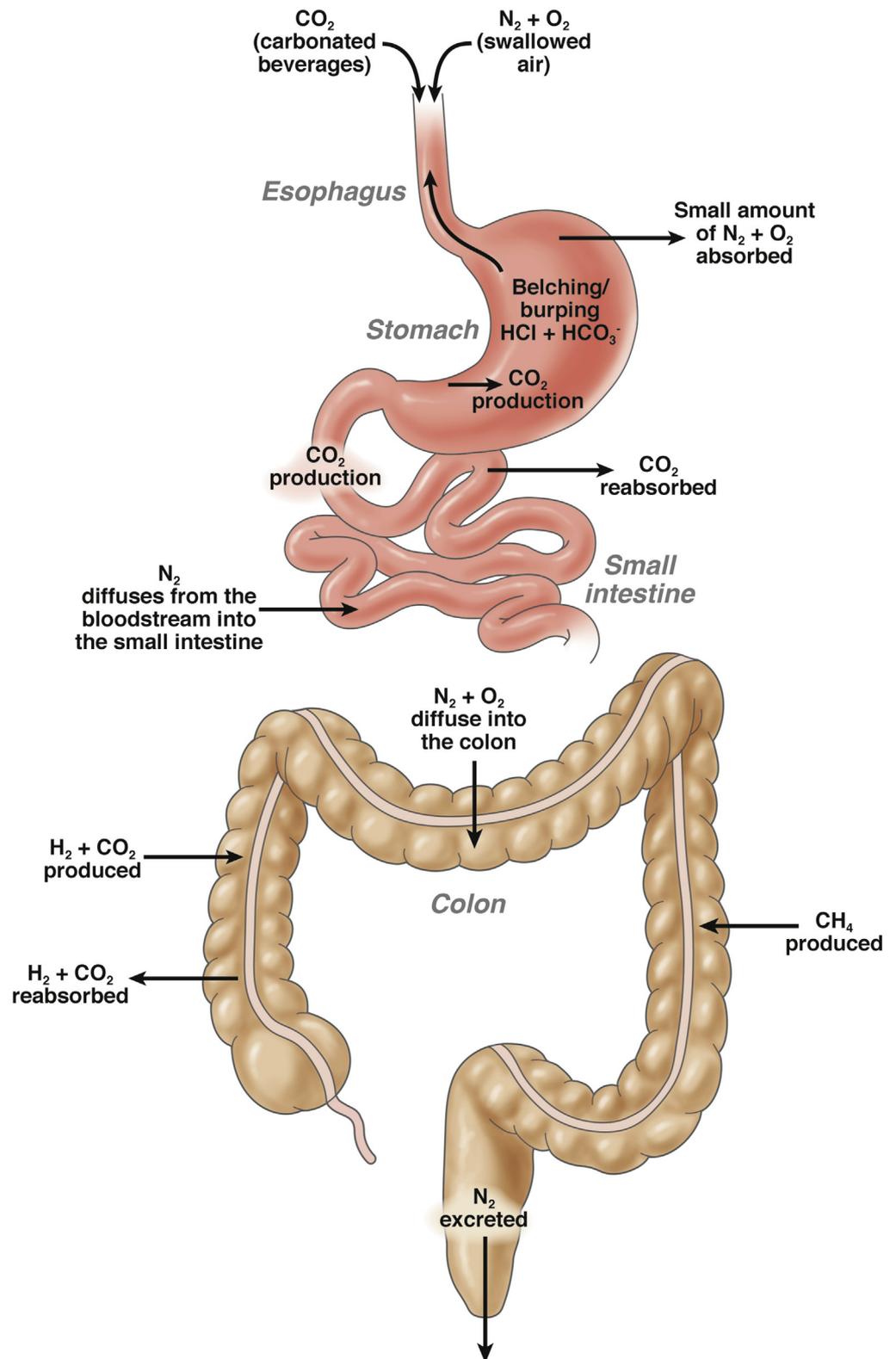
are lactase-deficient become symptomatic after ingesting lactose. This indicates that other factors (eg, genetic predisposition, visceral hypersensitivity) may be required for symptom generation in some patients.

### *Altered Microbiota*

No studies have focused solely on the implications of the gut microbiome in the pathogenesis of symptoms of abdominal bloating or distension. In contrast, numerous studies have described the role of the gut microbiota in disorders of gastrointestinal motility, sensation, and intestinal permeability.<sup>18</sup> Quantitative and qualitative differences in the intestinal microbiota have been identified comparing patients with IBS and healthy control subjects.<sup>19,20</sup> One study noted significant reductions in specific taxa from members of the Ruminococcaceae and Eubacteriaceae families among IBS patients without bloating compared with IBS patients with bloating and healthy control subjects.<sup>21</sup>

### *Abnormal Gastrointestinal Motility*

Bloating is common in patients with gastroparesis (over 50%)<sup>22</sup> and those with small bowel dysmotility (eg, chronic intestinal pseudo-obstruction and scleroderma). A prospective study of more than 2000 patients with functional constipation and IBS-C demonstrated that over 90% reported symptoms of bloating.<sup>23</sup> In IBS-C patients, those with prolonged colonic transit were shown to have greater abdominal distension compared with patients with normal transit.<sup>24</sup> Patients with functional bloating and IBS have impaired gas clearance from the proximal colon but normal colonic accommodation to gas infusion.<sup>25</sup>



**Figure 1.** Normal gas production, absorption, and excretion.

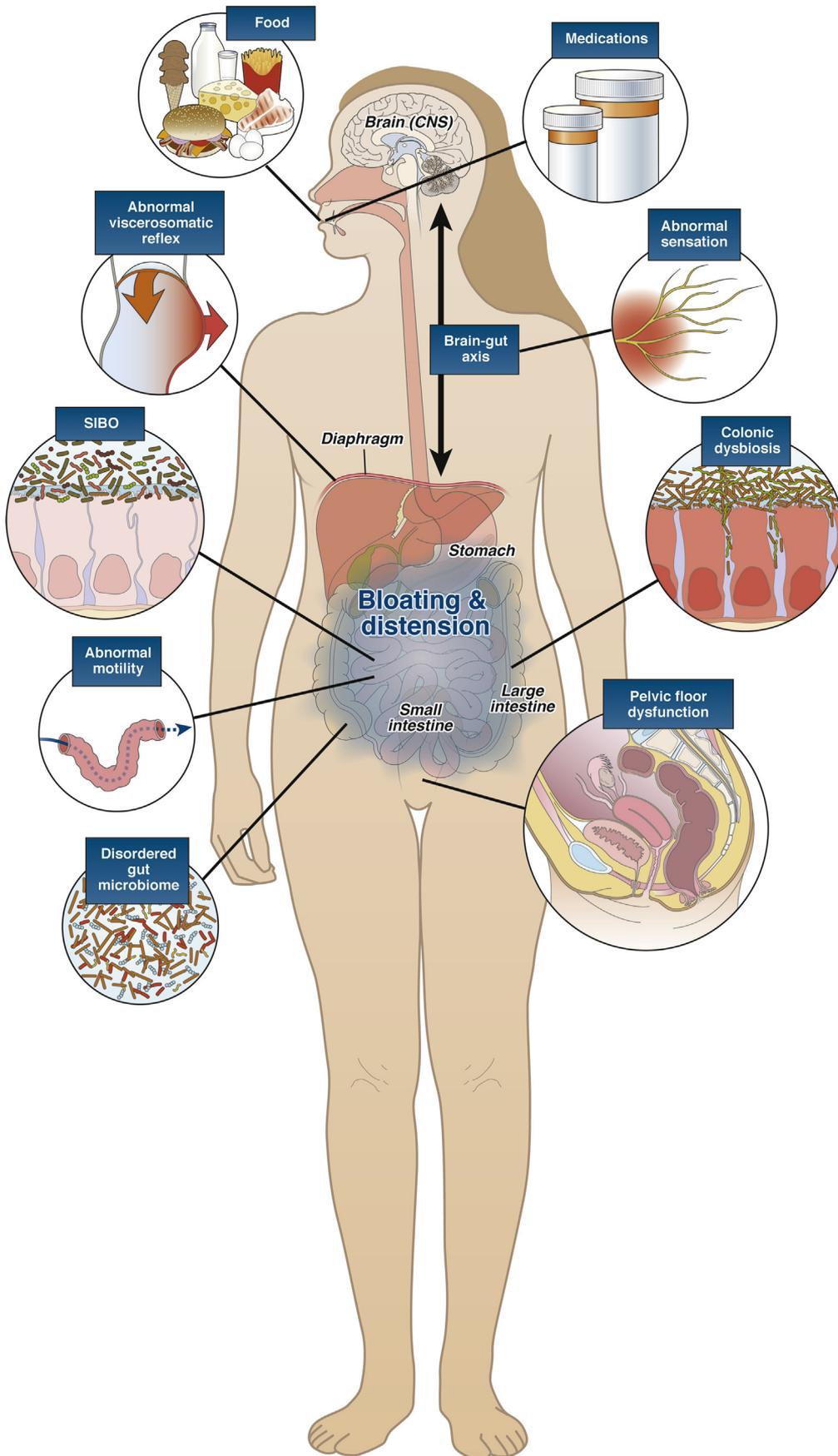
### *Pelvic Floor Dysfunction*

Patients with anorectal motor dysfunction may experience bloating and distension owing to an impaired ability to effectively evacuate both flatus and stool. Prolonged balloon expulsion correlates with symptoms of distension among patients with constipation.<sup>26</sup> Pelvic

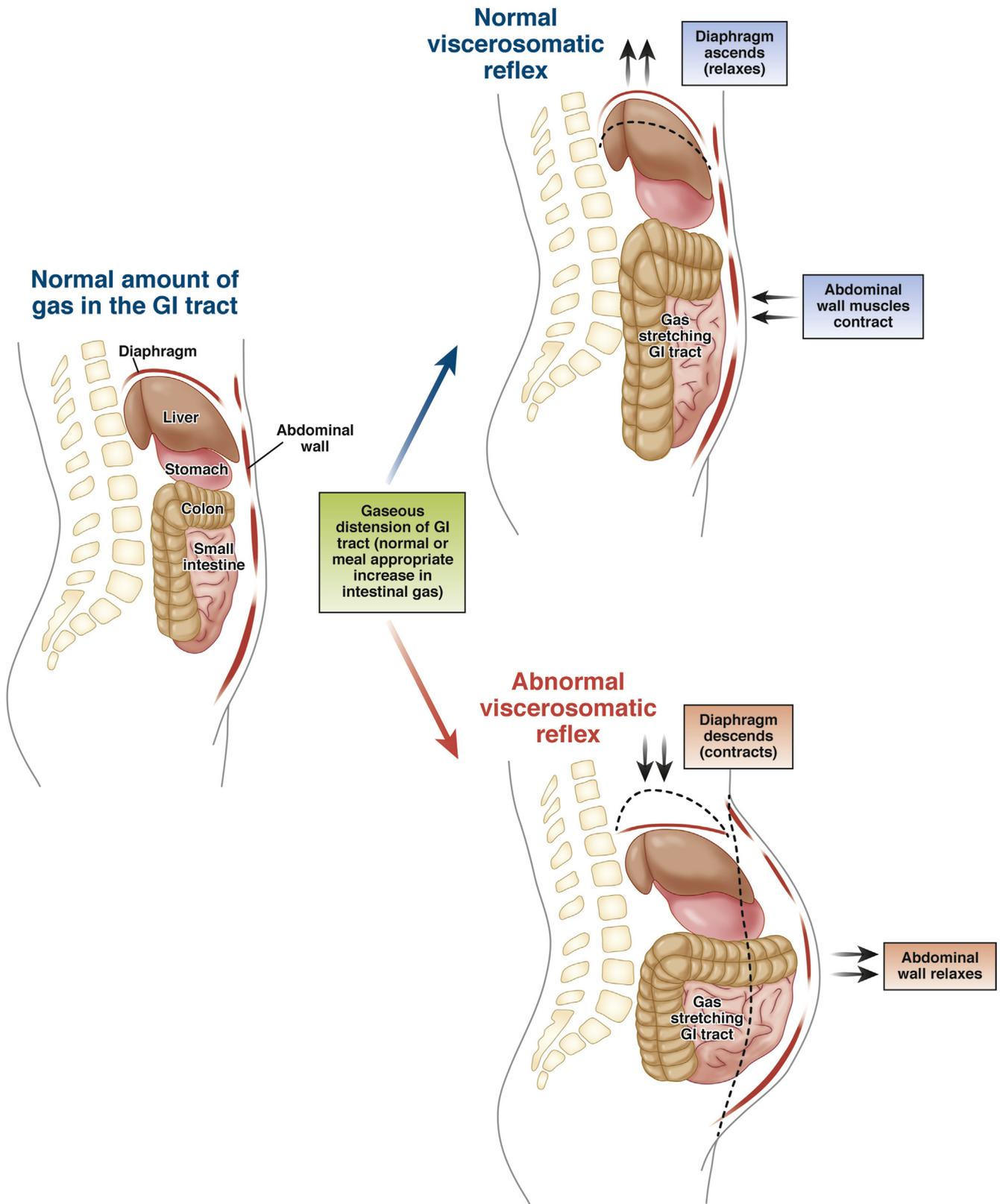
outlet obstruction has been shown to delay colonic transit.<sup>27</sup>

### *Abdominophrenic Dyssynergia*

A paradoxical abdominophrenic response, called abdominophrenic dyssynergia, develops in some patients



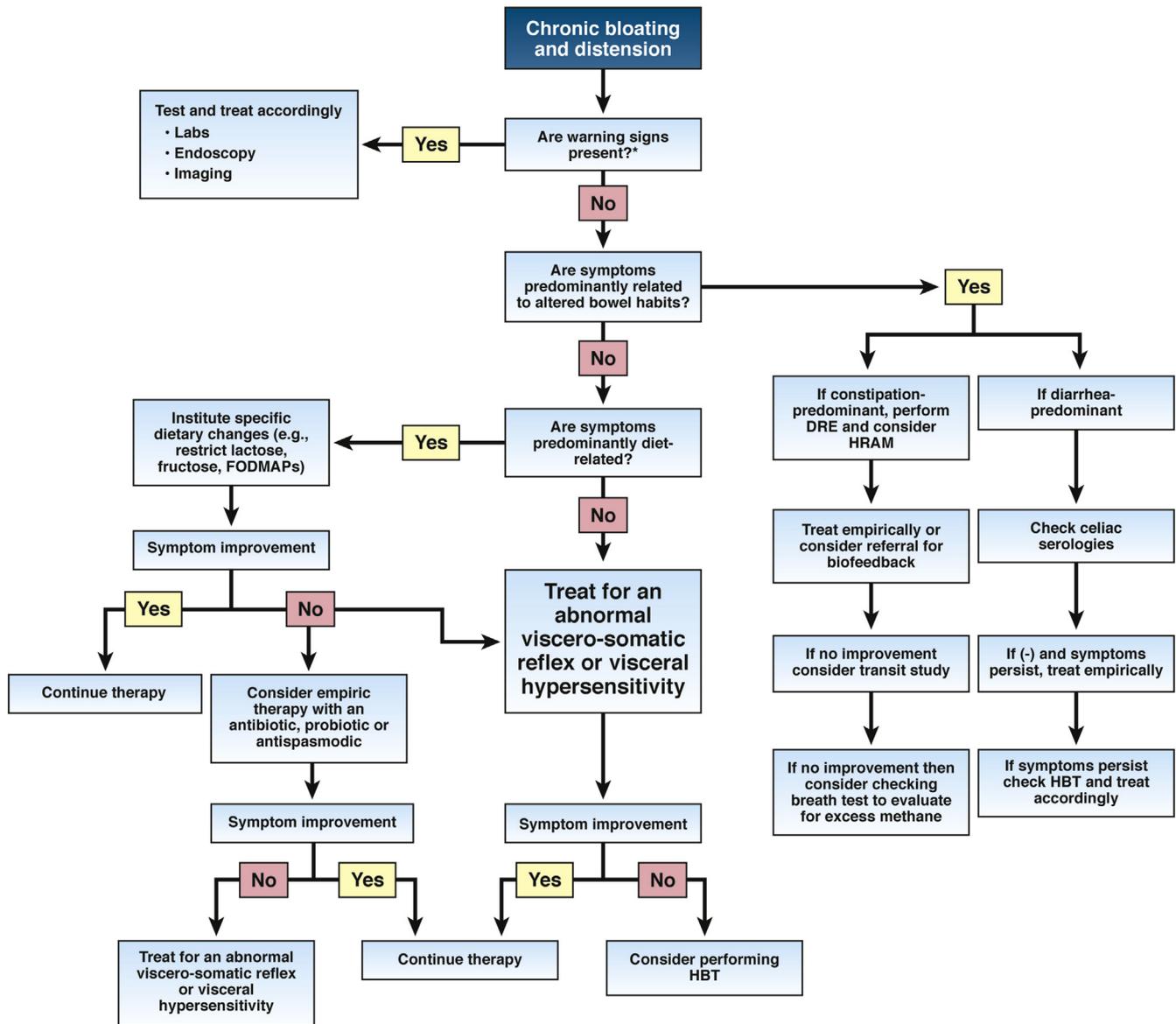
**Figure 2.** Pathophysiology of gas and bloating.



**Figure 3.** Normal and abnormal viscerosomatic reflexes.

with chronic bloating and distension. During this process the diaphragm contracts (descends) and the anterior abdominal wall muscles relax.<sup>28</sup> This response is in contrast to the normal physiologic response to increased

intraluminal gas, whereby the diaphragm relaxes and the anterior abdominal muscles contract in order to increase the craniocaudal capacity of the abdominal cavity without abdominal protrusion (see Figure 3). An elegant



**Figure 4.** Treatment algorithm for bloating and distension. The asterisk (\*) refers to anemia, gastrointestinal bleeding, weight loss >10% of body weight, and family history of GI malignancy. DRE, digital rectal examination; HBT, hydrogen breath test; HRAM, high-resolution anorectal manometry.

CT scan study demonstrated that patients with functional bloating have significant abdominal wall protrusion and diaphragmatic descent with relatively small increases in intraluminal gas. In comparison, patients with bloating and intestinal dysmotility were found to have marked increases in intraluminal gas content with resulting diaphragmatic ascent.<sup>29</sup> Abdominophrenic dyssynergia has also been identified in patients with functional dyspepsia (FD) and symptoms of postprandial bloating.<sup>30</sup>

### Visceral Hypersensitivity

IBS patients with symptoms of bloating alone have heightened visceral hypersensitivity compared with those with symptoms of bloating and distension.<sup>31,32</sup> Postprandial sensitivity to gastric balloon distension

was found strongly correlated with postprandial symptoms, such as bloating, in FD patients.<sup>33</sup> Conscious perception of intraluminal content and abdominal distension may contribute to symptomatic bloating and this can be amplified by complex brain-gut neural pathways, further influenced by factors such as anxiety, depression, somatization, and hypervigilance.<sup>34</sup>

### Diagnosis and The Role of Diagnostic Testing

A detailed clinical history and physical examination is critical to understand the underlying cause of bloating and distention. Details regarding the onset and timing of bloating and distention, the relationship to food or bowel movements, a surgical history (ie, Nissen fundoplication),<sup>35</sup>

and a careful review of medications (ie, narcotics), supplements, and dietary habits should be obtained.<sup>36,37</sup> A physical examination should include a rectal examination to identify an evacuation disorder in patients with constipation.<sup>38</sup> Information obtained will guide specific diagnostic testing (see [Supplementary Table 1](#)).

## Breath Tests

Breath tests (BTs) are a safe, noninvasive test to measure carbohydrate maldigestion based on the carbohydrate of interest. Test substances include glucose, lactulose, fructose, sorbitol, sucrose, and inulin.<sup>39</sup> Gas produced during colonic fermentation from nonabsorbed carbohydrates diffuses into the systemic circulation and is excreted in the breath, where it can be quantified.<sup>40</sup> Hydrogen and methane are the gases that are exclusively produced in the GI tract from microbial fermentation.

### Lactose BT

The absorption of lactose, a disaccharide composed of glucose and galactose, is dependent on the activity of the brush border enzyme lactase-phlorizin hydrolase.<sup>41</sup> Lactose maldigestion produces symptoms of bloating, abdominal cramping, flatulence, and diarrhea. Twenty-five grams of lactose is a standard test dose. An increase of  $\geq 20$  ppm of hydrogen or  $>10$  ppm of methane above baseline with associated GI symptoms is a positive test. Lactose BT has good sensitivity (mean value of 77.5%) and excellent specificity (mean value of 97.6%).<sup>40,42,43</sup>

### Fructose BT

Fructose is a naturally occurring sugar in fruits, different foods, and sweeteners. The absorptive capacity for fructose in the small intestine is minimal; unabsorbed fructose leads to symptoms of bloating and diarrhea. Controversies exist regarding the amount of fructose to use during BT,<sup>44</sup> although the most widely accepted dose is 25 g. A positive test is an increase  $\geq 20$  ppm of hydrogen or  $>10$  ppm of methane above baseline with associated GI symptoms.

### Small Intestine Bacterial Overgrowth

SIBO can cause symptoms of bloating, abdominal pain, gas, and diarrhea; vitamin deficiencies are less common.<sup>45</sup> The gold standard for the diagnosis of SIBO is aspiration and culture of jejunal fluid, but this is rarely performed, as it is costly, cumbersome, and invasive.<sup>46</sup> BT is widely available, inexpensive, and noninvasive, although there are limitations regarding standardization, test performance and interpretation.<sup>40,46</sup> Glucose and lactulose are the most accepted substrates.<sup>40,46</sup> A lactulose breath test is considered positive with a baseline

level  $>20$  ppm of hydrogen or  $>10$  ppm of methane, an early peak within 90 minutes, or a sustained increase by  $>10$  ppm more than baseline level.<sup>39</sup> A glucose BT is considered positive with an increase of 12 ppm of hydrogen or more over baseline or a baseline  $>20$  ppm of hydrogen or  $>10$  ppm of methane.

## Celiac Serologies

Malabsorption of wheat and gluten may cause symptoms of bloating, distension and accelerated GI transit in untreated celiac disease.<sup>47</sup> Serologic testing using tissue transglutaminase and IgA is recommended for patients with a high pretest probability for celiac disease.<sup>48</sup> Upper endoscopy with small bowel biopsies should be performed to confirm celiac disease in those who test positive.

## Upper Endoscopy

Upper endoscopy is necessary in patients when alarm symptoms are identified (recurrent nausea and vomiting, unexplained anemia, hematemesis, weight loss  $>10\%$  of body weight, a family history of gastroesophageal malignancy) or when gastric outlet obstruction, gastroparesis or FD is suspected. Upper endoscopy also provides an opportunity to biopsy the small intestine and stomach to exclude organic disorders as causes of bloating (see [Table 2](#)).

## Abdominal Imaging

An abdominal radiograph can establish stool burden in a patient with coexisting constipation. In patients with prior abdominal surgery, Crohn's disease, or known or suspected small bowel dysmotility, a CT scan of the abdomen, CT or magnetic resonance imaging enterography, or a careful fluoroscopic examination may be warranted.

## Gastrointestinal Function

Bloating is prevalent in patients with gastroparesis.<sup>49</sup> A 4-hour scintigraphic gastric emptying study is the standard test to diagnose gastroparesis or rapid gastric emptying.<sup>50</sup> Complete gastrointestinal transit assessment with either scintigraphy<sup>51</sup> or a wireless motility capsule<sup>52</sup> may be useful in patients with dysmotility or constipation thought secondary to slow transit. A gastric barostat or single-photon-emission CT imaging of the stomach can identify impaired gastric accommodation, although these tests are not widely available.<sup>53</sup>

## Anorectal Function Testing

Patients with severe constipation and bloating and with characteristic rectal examination findings

suggesting a pelvic floor disorder should be evaluated objectively.<sup>26,54</sup> Anorectal manometry with balloon expulsion is the most widely used test for evaluation of anorectal disorders.<sup>55,56</sup> Defecography, either barium or magnetic resonance imaging, provides an additional anatomical and functional assessment of the pelvic floor.<sup>57</sup>

## Treatment

The following sections highlight therapeutic approaches for the treatment of chronic bloating and distension (Figure 4). Treatments are listed in a sequence commonly followed in clinical practice, recognizing that treatment needs to be individualized. When available, data are presented from studies involving patients with functional bloating and distension (Supplementary Table 2).

### Diet

Artificial sweeteners that contain poorly absorbed sugar alcohols such as sorbitol, mannitol, xylitol, and glycerol promote gas production.<sup>58</sup> Restricting nonabsorbable sugars led to symptomatic improvement in 81% patients with functional abdominal bloating who had documented sugar malabsorption.<sup>59,60</sup>

Approximately 70% of patients considered to have nonceliac gluten sensitivity report bloating.<sup>60,61</sup> IBS patients, previously symptomatically controlled on a gluten-free diet, when challenged with gluten for 1 week had significantly worse bloating compared with placebo.<sup>62</sup> However, the role of gluten as a dietary source of bloating and other GI symptoms is controversial. Evidence suggests that fructans and FODMAPs (fermentable oligo-, di-, and monosaccharides) are the true culprits.<sup>61,63–65</sup> Two studies have shown that IBS patients treated with a low-FODMAP diet noted improvement in bloating and distension.<sup>66,67</sup>

Referral to a dietician can be useful if empiric treatment and/or therapeutic testing do not resolve symptoms.

### Probiotics

Probiotics, live microorganisms that confer a benefit to the host, can modify the gut microbiome and thus potentially improve bloating. Probiotics are available in a dizzying array of combinations and formulations. However, well-designed studies to evaluate improvements in bloating with probiotics are few in number. Studies with *Lactobacillus casei* strain GG, *L. plantarum*, and *L. reuteri* have shown no benefit in IBS patients with abdominal bloating and distention.<sup>68–70</sup> IBS patients randomized to *L. sporogens/Bacillus coagulans* had significant improvements in their bloating severity scores,<sup>71</sup> while studies with *Bifidobacterium* species demonstrated efficacy as

well.<sup>72,73</sup> However, in a recent multicenter trial, the encapsulated *B. infantis* 35624 failed to show any differences in bloating scores in subjects recruited from the general population.<sup>74</sup> A study of VSL#3 in IBS patients found a significant reduction in flatulence but no differences in bloating.<sup>51</sup>

### Antibiotics

The most commonly studied antibiotic for the treatment of bloating is rifaximin.<sup>75–77</sup> Global symptomatic improvement in IBS patients treated with antibiotics has correlated with normalization of hydrogen levels in lactulose hydrogen BT.<sup>75,78</sup> This suggests that the rationale for using antibiotics is to modify the gut microbiome and reduce bacterial production of gas. Patients with nonconstipation IBS randomized to rifaximin 550-mg three times daily for 14 days had a greater proportion of relief of IBS-related bloating compared with placebo<sup>77</sup> for at least 2 of the first 4 weeks after treatment (40.2% vs 30.3%;  $P < .001$ ). In a separate study mean cumulative and bloating-specific scores improved significantly in IBS patients treated with rifaximin 400 mg twice daily  $\times$  10 days compared with placebo ( $P < .05$ ).<sup>75</sup>

### Antispasmodics

Smooth muscle antispasmodics may improve symptoms if they arise due to gaseous distension of the GI tract.<sup>7,79</sup> Two meta-analyses of antispasmodics have focused on symptoms of bloating or distension. The first evaluated 6 studies involving 885 patients and 5 different agents.<sup>80</sup> Smooth muscles relaxants were more likely to improve symptoms of abdominal distension in IBS patients compared with placebo (odds ratio, 1.46; 95% confidence interval, 1.10–1.94). The second meta-analysis of 7 studies involving 1419 patients and 4 different agents were evaluated, and it was found that antispasmodics were better than placebo, although the odds ratio for both studies was borderline (odds ratio, 1.455; 95% confidence interval, 1.17–1.81).<sup>81</sup> A prospective, randomized, placebo controlled trial in patients with IBS (Rome III criteria;  $n = 285$ ) found that the combination of pinaverium bromide and simethicone was more effective at relieving symptoms of abdominal pain and bloating than placebo.<sup>82</sup>

### Secretagogues

In a randomized, placebo-controlled study of 1171 patients with IBS-C (Rome II criteria) twice daily lubiprostone (8  $\mu$ g) improved global IBS symptoms and also the secondary endpoint of bloating ( $P < .05$ ).<sup>83</sup> A 48-week open-label study of lubiprostone (24  $\mu$ g twice daily) in patients with chronic idiopathic constipation (CIC) ( $n = 248$ ) found that abdominal bloating was improved compared with baseline ( $P < .011$ ), although a

placebo group was not included.<sup>84</sup> Two randomized, double-blind, placebo-controlled studies evaluated the efficacy and safety of linaclotide (145 or 290  $\mu\text{g}$ ) in 1276 patients with CIC.<sup>85</sup> Both doses improved symptoms of bloating during the 12-week study ( $P < .001$ ). In a prospective, randomized study of 483 patients with CIC (Rome II criteria) with moderate-to-severe bloating, once-daily linaclotide (both 145 and 290  $\mu\text{g}$ ) significantly improved symptoms of abdominal bloating compared with placebo.<sup>86</sup> In a phase 3, double-blind, placebo-controlled trial of 804 patients, linaclotide improved IBS-C symptoms in addition to the secondary endpoint of bloating ( $P < .001$ ).<sup>87</sup> In a randomized, placebo-controlled study of 1394 patients with CIC (Rome III criteria) both the 3- and 6-mg doses of plecanatide once daily improved symptoms of constipation and bloating ( $P$ s  $< .002$  and  $.045$  for the 3- and 6-mg doses, respectively), compared with placebo.<sup>88</sup> Finally, 2 identically designed, randomized, double-blind placebo-controlled studies evaluated the efficacy and safety of plecanatide for the treatment of IBS-C ( $n = 2189$ ).<sup>89</sup> Both doses (3 and 6 mg once daily) improved symptoms of bloating ( $P < .001$ ).

### Prokinetic Agents

Prokinetic agents are used to treat symptoms of FD, gastroparesis, CIC, and IBS.<sup>7,90,91</sup> Data on the use of prokinetics for the treatment of chronic bloating and distension are limited. Neostigmine, a cholinesterase inhibitor, improved gas clearance in patients who underwent jejunal gas infusion.<sup>92</sup> Pyridostigmine was marginally better than placebo at improving symptoms of bloating in a small randomized, placebo-controlled study of IBS patients ( $n = 20$ ).<sup>93</sup> Acotiamide, a muscarinic antagonist and cholinesterase inhibitor, slightly improved, but did not eliminate, symptoms of meal-related bloating in Japanese patients with FD.<sup>94</sup> Metoclopramide did not improve symptoms of bloating in patients with either dyspepsia or gastroparesis.<sup>95,96</sup> An analysis of 4 randomized, placebo-controlled studies involving 1596 Asian and non-Asian women found that 2 mg of prucalopride, a 5-HT<sub>4</sub> agonist, once daily improved stool frequency and reduced symptoms of bloating.<sup>97</sup> A randomized, placebo-controlled crossover study involving 34 patients with gastroparesis found that prucalopride (2 mg once daily) improved global gastroparesis symptoms, including those of bloating and distension ( $P < .0005$ ).<sup>98</sup> Tegaserod, another 5-HT<sub>4</sub> agonist, was recently approved by the Food and Drug Administration for the treatment of IBS-C in women  $<65$  years of age without cardiovascular risk factors. Three separate, 12-week, randomized, prospective controlled studies demonstrated that tegaserod (6 mg orally twice daily) improved global IBS-C symptoms including bloating.<sup>99,100</sup>

### Neuromodulators

Medications that act in the brain and the gut have been relabeled as “neuromodulators.”<sup>101</sup> This group includes both central and peripherally acting agents. No study to date has focused on bloating as a single symptom. A large study of FD patients demonstrated that both amitriptyline (50 mg daily) and escitalopram (10 mg daily) significantly improved postprandial bloating compared with placebo ( $P$ s =  $.03$  and  $.02$ , respectively).<sup>102,103</sup> A crossover study of 17 FD patients showed that buspirone (10 mg three times daily), a 5-HT<sub>1A</sub> receptor agonist, significantly improved the overall severity of bloating.<sup>104</sup> In a 6-week crossover study of 23 IBS patients citalopram (20 mg and then 40 mg four times daily) significantly improved the frequency and severity of bloating, independent of anxiety and depression.<sup>105</sup>

### Biofeedback

In a study of 45 patients with various FGIDs, episodes of abdominal distension were associated with an abnormal viscerosomatic reflex.<sup>106</sup> Biofeedback treatment in 26 patients, utilizing visual guidance provided by electromyography signal, allowed all patients to effectively control muscular activity and improve distension.<sup>106</sup> These results were confirmed in a subsequent randomized, placebo-controlled trial in 44 patients with FGIDs and symptoms of postprandial bloating.<sup>107</sup> In a study of 52 patients with slow transit constipation, biofeedback therapy was shown to significantly reduce symptoms of bloating in patients with pelvic dyssynergia and pelvic outlet obstruction.<sup>27</sup> Additional studies of patients with idiopathic constipation, with various combinations of pelvic floor dysfunction or slow colonic transit, have also demonstrated significant reductions in bloating symptoms following biofeedback therapy.<sup>108</sup> No studies have assessed the use of pelvic floor physical therapy to treat functional bloating alone.

### Complementary and Alternative Medicine

While no agent has been studied specifically for the treatment of bloating, some data are available from studies in FD and IBS. Iberogast may improve dyspeptic symptoms, though bloating was not a specific endpoint.<sup>109</sup> In a randomized trial of 72 patients with IBS with mixed bowel habits or IBS with diarrhea, peppermint oil (180 mg three times daily) resulted in significant improvement in symptoms of bloating or distension compared with placebo.<sup>110</sup> Rikkunshito, a Japanese herbal medicine, improved symptoms of bloating at 4 and 8 weeks of treatment compared with placebo as a secondary endpoint in a randomized trial of 125 FD patients.<sup>111</sup>

## Hypnotherapy

An audit of 250 patients with IBS found that 12 sessions of hypnotherapy over a 3-month period improved IBS symptoms, including bloating.<sup>112</sup> A comparator was not included in this study. Large prospective studies comparing hypnotherapy with other active treatments should be performed to help rank order treatment options for providers.

## Conclusions

Abdominal bloating and distension are highly prevalent symptoms that affect, and negatively impact, patients with a spectrum of underlying medical and surgical disorders. Unfortunately, symptoms of bloating and distension are inherently nonspecific. The practitioner is thus faced with a number of challenges when evaluating a patient with symptoms of bloating and distension. First, identify the underlying etiology, as this may help pinpoint required diagnostic tests. Second, recognize warning signs of other organic or more serious disorders. Third, assess dietary, medication, medical, surgical, and behavioral factors that could contribute to the pathogenesis of bloating. Fourth, consider the main underlying physiologic processes that underlie bloating and distension, with the goal of initiating specific treatments (eg, dietary interventions for carbohydrate intolerance vs behavioral therapy for an abnormal viscerosomatic reflex). Last, providers should have a thoughtful discussion regarding risks and benefits of different therapies, with a dialogue involving costs, efficacy, and safety.

## Supplementary Material

Note: To access the supplementary material accompanying this article, visit the online version of *Clinical Gastroenterology and Hepatology* at [www.cghjournal.org](http://www.cghjournal.org), and at <https://doi.org/10.1016/j.cgh.2020.03.056>.

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**Reprint requests**

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**Conflicts of interest**

This author discloses the following: Brian E. Lacy has served as a consultant for Ironwood, Urovant, Salix, and Viver. The remaining authors disclose no conflicts.

**Supplementary Table 1.** Diagnostic Tests

Possible etiology	Diagnostic test(s)
Celiac disease	Serum tTG antibody, serum IgA
Carbohydrate intolerance	Hydrogen breath test
Obstruction	EGD, CT, fluoroscopy
Gastroparesis	4-h solid phase scintigraphy
Abnormal gastric accommodation	SPECT
Small intestine diverticulosis	CTE, SBFT
Delayed intestinal transit capsule	nuclear imaging, wireless motility
Pelvic floor dysfunction	ARM, defecography

ARM, anorectal manometry; CT, computed tomography; CTE, computed tomography enterography; EGD, esophagogastroduodenoscopy; SBFT, small bowel follow-through; SPECT, single-photon computed tomography; tTG, tissue transglutaminase.

**Supplementary Table 2.** Therapeutic Options

Dietary modifications  
 Probiotics  
 Antibiotics  
 Antispasmodics  
 Secretagogues  
 Prokinetics  
 Neuromodulators  
 Biofeedback  
 Complementary and alternative medications  
 Pelvic floor biofeedback therapy  
 Behavioral therapy