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Review article: bloating in functional bowel disorders

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SUMMARY

Bloating is a frequently reported symptom in functional bowel disorders. It usually occurs in combination with other symptoms, but may also occur in isolation. The severity of bloating tends to worsen during the course of the day and improves overnight. Although frequently considered to be a subjective phenomenon, recent studies have shown that bloating is associated with a measurable increase in abdominal girth.

The pathophysiology of bloating remains elusive, but the evidence supports a sensorimotor dysfunction of the bowel. The possible mechanisms include abnormal gas trapping, fluid retention, food intolerance and altered gut microbial flora. Further studies are needed to define the sensorimotor abnormalities associated with bloating, which might be segmental and transient rather than generalized and persistent.

The lack of understanding of this symptom is paralleled by a limited availability of therapeutic options. Conventional medications used in functional bowel disorders are not helpful and may indeed worsen the symptoms. In future, new drugs with activity against serotonin and kappa receptors, or novel approaches such as the use of exclusion diets, probiotics and hypnotherapy, may prove to be useful.

INTRODUCTION

Functional bowel disorders present a difficult diagnostic and therapeutic challenge in clinical practice. Abdominal pain or discomfort, bloating, excessive flatulence and altered stool form and/or frequency are the common presenting symptoms. Despite being a frequent complaint, bloating remains the least understood of all the symptoms. The word 'bloating' means swelling or distension. There is considerable confusion in the literature with regard to the definition of 'bloating', and it has been used to describe both a subjective feeling of distension as well as an obvious swelling of the abdomen. Its pathogenesis remains elusive and there are few, if any, diagnostic and therapeutic options available.

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Patients with bloating frequently report a visible increase in abdominal girth. Characteristically, it worsens as the day progresses and tends to improve or disappear overnight. Post-prandial exacerbation of symptoms is also a common feature. The severity of symptoms may necessitate the change or loosening of clothes over the course of the day. Friends and family members frequently bear witness to the patients' testimony, but the doctors, more often than not, are sceptical.

Alvarez suggested that visible bloating is the result of a voluntary increase in lumbar lordosis.[1] It has been suggested that, if the patient is examined in the supine position with the legs flexed at the hips and knees, the distension disappears as the lordosis is reversed.[2] However, Maxton *et al.* demonstrated that the abdominal girth, measured at three anatomical levels, increased significantly during the day in female irritable bowel syndrome patients when compared with controls.[3] This was true both in the lying and standing

positions. Lateral standing plain X-rays showed no correlation between the changes in abdominal girth and the extent of lumbar lordosis. Abdominal computed tomography scans showed a significant increase in antero-posterior diameter during the day, which could not be attributed to either the position of the diaphragm or the volume of the bowel gas. This suggests that visible bloating is a real entity caused by an increase in the intra-abdominal volume.

EPIDEMIOLOGY OF BLOATING IN THE GENERAL POPULATION AND IN FUNCTIONAL BOWEL DISORDERS

Bloating or abdominal distension is a common complaint in the general population. In a recent cross-sectional survey of 2510 subjects in US households, 16% reported experiencing abdominal bloating in the month prior to the interview.[4] Sixty-five per cent of the respondents complained of moderate to severe bloating; 43% had taken medication for this symptom and 16% of the sufferers had sought medical advice. One-quarter of the sufferers reported a reduction in daily activity of ≥25% attributable to their symptoms. The prevalence of bloating was almost twice as high in females (males vs. females, 10.5% vs. 19.2%).

Abdominal bloating is second only to abdominal pain as the most frequently reported symptom in functional bowel disorders.[5, 6] In addition to irritable bowel syndrome, it is a common feature of functional dyspepsia, post-cholecystectomy syndrome and idiopathic constipation. Symptoms of 'dysmotility-like' functional dyspepsia are referable to the upper abdomen, including fullness, bloating, early satiety and/or nausea. Possible aetio-pathological mechanisms include impaired gastric accommodation,[7] delayed gastric emptying[8] and abnormal intragastric distribution of food.[9] Bloating, early satiety and nausea are frequently reported symptoms of post-cholecystectomy syndrome. Studies have demonstrated abnormalities in the fasting antroduodenal motility with increased duodenogastric reflux in this condition.[10] There is considerable overlap of symptoms between irritable bowel syndrome and dyspepsia, and amongst the dyspepsia subgroups in the general population.[11–14] A patient with dyspepsia may develop symptoms of irritable bowel syndrome over time and vice versa, although the irritable bowel syndrome patients, in general, show the highest stability and lowest disappearance of their symptoms.[13] Studies have demonstrated a generalized sensory abnormality common to both irritable bowel syndrome and functional dyspepsia, suggesting that these disorders have a common underlying pathophysiology.[15, 16]

IS BLOATING A SEPARATE DISORDER OR PART OF IRRITABLE BOWEL SYNDROME?

In irritable bowel syndrome patients, whilst bloating may be the predominant symptom, it usually occurs as a part of a symptom complex, with 80–90% of irritable bowel syndrome patients reporting excessive flatulence and abdominal distension.[17] In fact, bloating is rated as the most intrusive of all the symptoms by irritable bowel syndrome patients.[18] The recently revised Rome II criteria for the diagnosis of irritable bowel syndrome do not include bloating as an essential criterion, although its presence is considered to be supportive.[19] Patients who have bloating as an isolated or predominant symptom in the absence of other criteria of irritable bowel syndrome are classified as having 'functional bloating'. Functional bloating, in the absence of other symptoms of irritable bowel syndrome, is defined as at least 12 weeks, which need not be consecutive, in the preceding 12 months, of: (i) a feeling of abdominal fullness, bloating or visible distension; and (ii) insufficient criteria for a diagnosis of functional dyspepsia, irritable bowel syndrome or other functional disorder.

There is marked inter-individual variation of symptoms in irritable bowel syndrome patients. However, it is not clear whether these represent separate pathophysiological processes or constitute the variable expression of a single underlying disorder. Manning *et al.* reported four symptoms to be most frequent in irritable bowel syndrome patients: abdominal distension; pain relief with defecation; more frequent stools with the onset of pain; and looser stools with the onset of pain.[5] Only 31% of irritable bowel syndrome patients had all four symptoms, whereas 37.5% reported two or less symptoms. Although these symptoms are significantly correlated with the diagnosis of irritable bowel syndrome in female patients, the correlation in men is poor.[20] It has also been shown that irritable bowel syndrome patients tend to maintain an unchanged symptom profile over time.[13, 21] The observation that symptom complexes can vary greatly between different patients, but remain constant for an individual, supports the hypothesis that individual symptoms or combinations of symptoms may represent separate pathophysiological entities.

Ragnarsson and Bodemar used cluster analysis to identify irritable bowel syndrome subgroups using daily symptoms recorded in diary cards.[22] Two pain/bloating subgroups were identified, one with little and the other with considerable pain/bloating. There was no relationship between symptoms of pain/bloating and abnormality of defecation, suggesting that the underlying mechanisms in these may be different. The authors suggested that pain and bloating might be mediated by intestinal hypersensitivity, whereas abnormal defecation may be an expression of altered bowel motility.

Even though pain and bloating appear to be the manifestation of intestinal sensory dysfunction, they do not necessarily share a common pathophysiology. They can occur in isolation and, when they do co-exist, either may predominate, suggesting that the two do not constitute impaired regulation of a single process. In addition, a common clinical observation is that treatments aimed at controlling pain do not appreciably improve bloating. Clearly, the pathophysiology of bloating needs to be studied further.

MECHANISM OF BLOATING

Symptomatic bloating is frequently seen in disorders associated with obstruction to the normal flow of gas, fluid or faeces within the bowel lumen. The restoration of normal flow of intraluminal contents is associated with the relief of symptoms. 'Gas-bloat' syndrome following fundoplication is well recognized and is attributable to increased fundal pressure from excess gas in the stomach.[23, 24] The symptoms are relieved by balloon dilatation or, in extreme cases, by surgical reversal of the fundoplication. Gastric outflow obstruction, leading to fluid and food stasis in the stomach, also induces a feeling of distension. Small and large bowel obstruction can lead to similar symptoms. The symptoms may be generalized or localized to the upper or lower abdomen, depending on the level of obstruction. In all cases, the sensation of bloating is caused by the stimulation of bowel wall mechanoreceptors due to increased intraluminal pressure and subsequent distension of the obstructed segment.[25]

Bloating may also be induced by increased intra-abdominal pressure from causes other than bowel obstruction, such as rapidly developing ascites, pregnancy, etc.[2] The symptoms in these disorders may be caused either by the stimulation of abdominal wall stretch receptors or by the compression of various internal organs.

Similarly, bloating in functional bowel disorders may be caused by a number of possible mechanisms. Firstly, it may be induced by the stimulation of bowel and/or abdominal wall mechanoreceptors due to excess gas, fluid retention or reduced abdominal muscle tone—a mechanism similar to that seen in various obstructive conditions. Secondly, triggers such as food hypersensitivity, inflammation and changes in gut microbial flora may alter the sensory threshold of the gut to normal luminal contents. Alternatively, it may represent a primary sensorimotor dysfunction of the enteric nervous system.

Intestinal gas

The production of excess intestinal gas has been proposed as the likely mechanism in bloating.[17] The excess gas may be produced *de novo* from bacterial fermentation of undigested carbohydrates in the colon or from excess swallowed air. Ninety-nine per cent of colonic gas comprises a mixture of nitrogen, hydrogen, carbon dioxide and methane.[26, 27] In healthy individuals, most of the nitrogen gas comes from swallowed air, whereas hydrogen and methane are produced by colonic bacterial fermentation of undigested carbohydrates. A post-prandial increase in the passage of flatus is due to the increased production of carbon dioxide and hydrogen by the latter mechanism.[28]

Measurement of intestinal gas. It is difficult to quantify the intestinal gas in clinical practice. The complaints related to excess intestinal gas include bloating, excessive flatulence, foul smell and the inability to control it in socially unacceptable situations. Most studies have focused on the measurement of the volume of gas produced in the gut. Various techniques employed for this purpose include plethysmography,[29] hydrodensitometry,[30] intestinal washout techniques using argon infusion into the duodenum[26, 27, 31] and manual geometric measurements on abdominal roentgenograms.[32] Either due to methodological flaws or technical complexities, none of these techniques has a role in routine clinical practice. The normal quantity of bowel gas, as measured by these techniques, has been reported to range from 0.03 to 1.0 L. However, most researchers agree that the normal quantity of bowel gas is below 0.2 L. More recently, bowel gas has been measured with

a computer digitizing board using abdominal radiographs.[33] Two independent evaluators agreed well on the measured areas of bowel gas ($r = 0.96$), suggesting that the technique was reliable. The method provides an inexpensive and objective measure of abdominal gas with little inter-observer variation, but even with this technique, the measured volume was dependent upon the subject's position (erect vs. supine).

'Gas trapping'—a possible mechanism in bloating. Bloating may be caused by impaired transit and/or segmental sequestration of gas, with or without an increase in the absolute volume of intestinal gas. Healthy individuals tolerate jejunal gas infusion without developing symptomatic abdominal distension, except in a small proportion who retain >400 mL of the infused gas.[34] In comparison, the majority of irritable bowel syndrome patients develop gas retention (> 400 mL), increased abdominal girth and a feeling of distension in response to jejunal gas infusion.[35] The retention of infused jejunal gas in healthy individuals has been shown to produce symptoms in response to obstructive evacuation (self-restrained), but not with impaired gut propulsion (intravenous glucagon).[36] This suggests that symptom perception in response to intestinal gas retention depends on the underlying mechanism, i.e. hypotonic gut vs. obstructive retention. It is a common clinical observation that the passage of flatus improves bloating in irritable bowel syndrome patients. Both excessive flatulence and bloating are common symptoms in irritable bowel syndrome. It is possible that these two symptoms are caused by different mechanisms, with 'gas trapping' as the predominant mechanism in 'bloaters', as opposed to increased intestinal transit of gas in flatulent patients. The evaluation of these subgroups will help in the understanding of the mechanism of bloating.

Is bloating caused by excess intestinal gas? It is a widely held misconception that bloating is caused by the production of excess bowel gas. In a comparative study of 12 patients with chronic complaint of excess gas and 10 controls, using a washout technique with intestinal infusion of an inert gas mixture, no significant difference in the composition or accumulation rate of intestinal gas was observed.[37] However, the patients complained of abdominal pain at a significantly lower volume of infused gas and had a significantly longer intestinal transit time when compared with controls. This suggests that the symptoms may be caused by altered sensory perception or abnormal intestinal motility rather that an excess of absolute volume of bowel gas.

The relationship between colonic symptoms and the volume of abdominal gas has also been challenged recently. Chami *et al.* measured abdominal gas in 19 constipation-predominant irritable bowel syndrome patients and 24 healthy controls using a computerized digitizing board.[33] Although abdominal gas was significantly greater in the patient group, the variation in magnitude of the symptoms of bloating, pain or flatulence correlated poorly with the volume of estimated abdominal gas. Other studies have shown that strategies aimed at reducing bowel gas fail to relieve the symptoms of bloating.[38, 39]

Bloating may be induced by intestinal contents other than excess bowel gas, such as dietary fibre. In a controlled study of 25 healthy volunteers, significant increases in breath hydrogen excretion, frequency of gas passage and subjective impression of increased rectal gas were reported in response to oral lactulose, but not to psyllium (a fermentable fibre) or methylcellulose (a non-fermentable fibre).[40] However, a significant increase in abdominal bloating scores was reported in response to lactulose as well as both of the fibres. Whilst symptoms following lactulose challenge can clearly be attributed to excess colonic bacterial fermentation, the induction of bloating by both fermentable and non-fermentable fibres suggests that the actual bulking effect of fibre on the intestinal contents was responsible for bloating.

In summary, bloating is not synonymous with excess intestinal gas. Abnormal handling of bowel gas and the influence of other dietary constituents may contribute to the pathogenesis of bloating.

Food intolerance

Lactose intolerance is a common disorder and the symptoms associated with it, such as nausea, fullness, bloating, pain and diarrhoea, are very similar to those found in irritable bowel syndrome patients.[41, 42] It has been suggested that lactose intolerance may play a role in the symptoms of irritable bowel syndrome. Tolliver *et al.* evaluated the possible role of lactose intolerance in 166 irritable bowel syndrome patients.[43] Forty-seven (29%) irritable bowel syndrome patients had lactose intolerance as determined by lactose hydrogen breath testing. After a mean of 44 months, the re-evaluation of symptoms showed no difference in the symptom scores

(including bloating) between the patients with and without lactose intolerance, despite the fact that lactose malabsorbers avoided dietary lactose. Another study showed that bloating was more common in irritable bowel syndrome patients compared to lactose malabsorbers and healthy controls, but this could not be attributed to increased gas production from bacterial fermentation of undigested carbohydrates.[44]

Fluid retention

The increased incidence of bloating in women compared to men and the pre-menstrual worsening of symptoms suggests a possible perturbing role of female sex hormones.[20, 45, 46] Functional bowel disorders are common in women with dysmenorrhoea[47] and during pregnancy.[48] Bloating is the most common reason for the discontinuation of hormone replacement therapy in post-menopausal women.[49] In addition, female irritable bowel syndrome patients complain of gynaecological symptoms frequently.[50] A possible mechanism for these observations is sex hormone-induced fluid retention in the body. However, this alone would not account for the diurnal variation reported in bloating.

The effect of other hormones involved in fluid homeostasis, such as corticosteroids, mineralocorticoids, renin–angiotensin system, etc., may play a role. In one study, 'diarrhoea-predominant' irritable bowel syndrome patients were shown to have significantly increased cortisol levels post-prandially compared to controls or 'constipation-predominant' irritable bowel syndrome patients.[51]

However, most studies have shown no diurnal change in the total body fluid content in patients with bloating.[3, 52, 53] The fact that there are few symptom differences between pre- and post-menopausal women also supports this argument.[54]

The sequestration of fluid within the bowel loops, without alteration in the total body fluid content, has been proposed as a possible mechanism in bloating.[55] This hypothesis has not been studied so far.

Altered abdominal muscle tone

A reduced abdominal muscle tone has been suggested as a cause of the increased abdominal girth seen in bloating.[1] Patients with bloating give a history of recent weight gain, lack of regular exercise and have reduced abdominal muscle strength.[53, 56] However, such a mechanism should result in a soft and flabby abdominal wall. On the contrary, many patients with bloating have a tense, uncomfortable abdomen. McManis *et al.* measured the electromyographic activity of the abdominal wall musculature in irritable bowel syndrome patients with visible distension to test this hypothesis.[52] In healthy individuals, there was an increase in electromyographic activity of the abdominal musculature on standing. No difference was seen in the electromyographic voltage between irritable bowel syndrome patients and controls in either the erect or supine positions and both showed increased activity on standing up. This study suggests that abdominal muscle tone does not play a primary pathogenetic role in bloating.

Altered gut microflora

Gut microbial flora is unique to each individual. The foetal gut is sterile, but colonization of the gut begins during transit through the birth canal and is later modified by environmental factors. Once established, the microbial flora in an individual remains more or less stable throughout life. Alteration may occur as a result of various disease processes and with the use of antibiotics. As the colonic bacteria differ in their ability to ferment undigested carbohydrates, it is conceivable that overgrowth of one type of microbe may result in excess fermentation and gas production with resultant bloating. It has been shown that irritable bowel syndrome patients have a decreased concentration of coliforms, lactobacilli and bifidobacteria compared to controls.[57] Based on this hypothesis, attempts have been made to influence gas-related symptoms in irritable bowel syndrome patients by modifying the gut flora using probiotics containing lactobacilli.[58, 59] Although improvement was seen in some of the symptoms, including flatulence, pain and stool form, the bloating scores remained unchanged. In another study, a non-absorbable antimicrobial agent active against anaerobes was used to treat gas-related symptoms.[60] A significant reduction in the mean number of flatus episodes and abdominal girth was achieved, but no improvement was observed in bloating and pain scores. Further studies are needed to evaluate the usefulness of this approach.

Inflammation and/or food hypersensitivity

Recent studies have suggested that irritable bowel syndrome patients have microscopic inflammation of

the small and large bowel.[61, 62] This primarily involves mucosal infiltration by mast cells. It has been suggested that infection and/or food hypersensitivity may be responsible for the sensitization of mast cells, which in turn can secrete chemical mediators capable of causing sensorimotor abnormalities in the gut. Although exclusion diets improve irritable bowel syndrome symptoms, the identification of the offending foods is a cumbersome process. Food-specific serum immunoglobulin E antibodies and skin prick tests have not been proven to be useful in identifying the incriminating foods.[63, 64] Food-specific immunoglobulin G4 antibodies are elevated in irritable bowel syndrome patients, and exclusion diets based on these may help to control the symptoms of irritable bowel syndrome, including bloating.[65–67] Future developments in the diagnosis and treatment of food hypersensitivity will help in determining the role of this approach in bloating.

BLOATING — A PRIMARY SENSORIMOTOR DYSFUNCTION OF THE BOWEL?

Altered mucosal sensitivity in bloating

Irritable bowel syndrome has been associated with visceral sensory dysfunction of both the small and large bowel. Kellow *et al.* showed increased awareness of physiological small bowel motor activity in irritable bowel syndrome patients.[68] Rectal afferent hypersensitivity in response to phasic balloon distension has been reported in irritable bowel syndrome patients.[69, 70] In addition, rectal hyperalgesia can be induced in irritable bowel syndrome patients by repetitive high-pressure mechanical stimulation of the rectosigmoid area.[71] It has also been shown that irritable bowel syndrome patients have selective hypersensitivity of small intestinal mechanoreceptors, as determined by jejunal balloon distension, whereas the perception of small intestinal transmucosal electrical nerve stimulation and small intestinal compliance was not significantly different from controls.[72] Whilst jejunal mechanoreceptors can be sensitized by the dietary lipid content,[73] the effect of modulators, such as infection, inflammation and microbial flora, needs to be evaluated further.

To date, the sensorineural pathways mediating pain and bloating have not been clearly defined. In a recent study, repetitive sigmoid balloon distension was shown to induce rectal hypersensitivity in irritable bowel syndrome patients with pain as the predominant symptom, but not in those with bloating as the predominant symptom.[18] This suggests that pain and bloating are modulated by separate neurological mechanisms. At present, the clinical significance of these findings remains undetermined. Future studies will help in the understanding of the neurophysiological mechanisms operating in healthy individuals and irritable bowel syndrome patients.

Altered bowel tonic and phasic motility

Various abnormalities of fasting and post-prandial small bowel motility have been identified in irritable bowel syndrome patients.[74–76] Although frequent episodes of clustered contractions have been associated with abdominal discomfort, no specific abnormality has been reported in association with bloating.[74] Abnormalities of colonic motility have also been identified in some but not all studies.[77–79] These include differences in the frequency and amplitude of contractions and colonic tone.[80, 81] However, motility patterns associated with different symptom subgroups need to be characterized further. The diurnal and post-prandial variations seen in irritable bowel syndrome will require prolonged ambulatory studies of the small and large bowel to enable the motility to be characterized and to enable the effect of various factors known to influence the symptoms to be studied.

Motility abnormalities observed in bloating caused by various pathological conditions, such as pseudo-obstruction, diabetic autonomic neuropathy and partial small bowel obstruction, might provide a framework for comparison.[82–86] Studies are needed to define the interaction between intestinal tone, compliance, motility and sensory abnormalities in relation to patients' symptoms. Whilst progress is being made, much remains to be done.[87–89] It should be recognized that motor abnormalities associated with bloating might be segmental and transient rather than generalized and persistent, and this should be addressed in future study designs. An atonic or spastic bowel segment can result in the impaired transit of gas and fluid, which may have important pathophysiological significance.

MEASUREMENT OF BLOATING — 'A CLINICAL BLOATOMETER'

The objective clinical assessment of bloating is fraught with difficulties. Routine use of radiological imaging,

including computed tomography scans, cannot be advocated due to the antecedent risk from radiation exposure in this benign condition. Simple clinical methods, such as the measurement of abdominal girth, are difficult to standardize and, at best, provide a snapshot view of a dynamic condition. Recently, the technique of ambulatory inductance plethysmography has been used to record changes in abdominal girth over a 24-h period.[90] A study of a group of healthy controls has demonstrated that this method correlates well with tape measurements and subjective assessment using visual analogue scores. It also allows the accurate assessment of diurnal variations in the abdominal girth. Comparative studies between healthy controls and patients with bloating are awaited with interest. Such a device will help in the understanding of the pathogenesis of bloating and the evaluation of various therapeutic interventions in bloating.

MANAGEMENT

The management of irritable bowel syndrome, after the exclusion of organic diseases, is usually aimed at controlling symptoms. This involves the use of antispasmodics for pain, laxatives for constipation and bulking agents and opiates for diarrhoea.

Treatment options for the management of bloating, however, are limited and unrewarding. Strategies aimed at improving other symptoms are ineffective and, in some cases, counterproductive, e.g. bran and opiates. Regular exercise, weight loss, healthy eating habits and restriction of fibre in the diet are frequently recommended, but are of no proven benefit.[2, 55] Dietary exclusion of gas-producing foods and the use of activated charcoal and simethicone to reduce bowel gas have shown limited efficacy.[38, 39, 60] The use of antibiotics and probiotics has so far proven to be ineffective.[58–60] Prokinetic agents, such as cisapride, have shown no efficacy over placebo.[91] A few studies have shown improvement with hypnotherapy.[92] In a preliminary study, aloe vera has not been shown to be beneficial for bloating, although it may improve pain severity.[93] Patients frequently relate their symptoms to food and may have tried various exclusion diets by the time they seek consultation. In the absence of definite evidence of lactose intolerance, the exclusion of milk and other dairy products is unlikely to be successful.[43] Serum immunoglobulin E and skin prick tests have not been of any value in identifying suspect foods. Exclusion diets based on raised serum immunoglobulin G4 antibodies to common food antigens may be useful in improving the symptoms of irritable bowel syndrome, including bloating.[94] However, further work is needed to evaluate the usefulness of this approach. The new generation of drugs with activity against serotonin and kappa receptors have shown promising results in initial trials, but need further evaluation to establish their efficacy and safety.[95, 96]

CONCLUSIONS

Bloating remains the most poorly understood symptom in functional bowel disorders. Despite being rated as the most bothersome symptom, very few studies have addressed it in a meaningful way. The common perception that it is caused by excess gas, fluid retention or weak abdominal musculature is not supported by the available data. Therapies aimed at other symptoms in irritable bowel syndrome are either ineffective or counterproductive. Further studies are needed to identify sensorimotor abnormalities of the small and large bowel in order to gain an insight into its pathophysiology. In essence, bloating may represent a separate aetiopathological entity from other functional abdominal disorders, and should be the subject of future research.

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