

REVIEW

Towards a better understanding of abdominal bloating and distension in functional gastrointestinal disorders

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Abstract Abdominal bloating is an extremely common symptom affecting up to 96% of patients with functional gastrointestinal disorders and even 30% of the general population. To date bloating has often been viewed as being synonymous with an actual increase in abdominal girth, but recent evidence suggests that this is not necessarily the case. This review examines the relationship between the symptom of bloating and the physical sign of abdominal distension, as well as examining the epidemiology, pathophysiology and treatment options available for this debilitating aspect of the functional gastrointestinal disorders. Pathophysiological mechanisms explored include psychological factors, intestinal gas accumulation, fluid retention, food intolerance and malabsorption of sugars, weakness of abdominal musculature, and altered sensorimotor function. Treatment options are currently rather limited but include dietary changes, pharmacological approaches, probiotics and hypnotherapy.

Keywords bloating, distension, intestinal gas, functional gastrointestinal disorders.

INTRODUCTION

Abdominal bloating is a common symptom affecting up to 96% of patients with functional gastrointestinal disorders and 10–30% of the general population.^{1–6} It

often co-exists with one or more of borborygmi, distension, excessive flatulence or frequent eructations,^{7,8} and in addition to occurring on its own, is often associated with other disorders such as irritable bowel syndrome (IBS), functional constipation, functional dyspepsia and even premenstrual syndrome.^{2,5,7,9–14} Sufferers typically report a worsening of bloating as the day progresses, particularly after meals, and it generally improves or disappears overnight.^{5,11,15–17} Some individuals relate bloating to ingestion of specific foods whilst others claim that any type of oral intake, even water can induce or exacerbate the problem.⁵ Furthermore, bloating appears to have a direct relationship to the intensity of abdominal pain.⁵

IS BLOATING SYNONYMOUS WITH ABDOMINAL DISTENSION?

Both the patient and their physician often view the subjective sensation of abdominal bloating as being synonymous with an actual increase in abdominal girth (i.e. distension). Indeed many authors still use the two terms interchangeably. This view was supported by early studies using a tape measure which suggested that the diurnal worsening of the sensation of bloating was accompanied by an increase in abdominal girth.^{18,19} However, a recent survey of patients with IBS, has suggested that only three quarters of patients believe their bloating is associated with visible abdominal distension, the rest describing a sensation of bloating alone.⁵ This inconsistency may in part be explained by the fact that tape measurements are not necessarily always reliable, as they are open to possible bias by both patient and investigator. For example, in an attempt to demonstrate the severity of their problem the patient may voluntarily protrude their abdomen during a single measurement or the investigator could inadvertently influence measurements by

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Figure 1 Photograph of a subject wearing the abdominal inductance plethysmography equipment. DL, the data logger; and TS, mercury tilt switches.

abdominal girth was measured with a tape measure before and after the infusion of as much as 2 L of peritoneal dialysis fluid into the peritoneal cavity of renal failure patients showed tape measurements to be both inaccurate and insensitive.^{20,21} These problems have recently been overcome with the development of more objective techniques to measure abdominal girth, such as extensometry²² and abdominal inductance plethysmography^{23,24} (Fig. 1). The latter technique, unlike extensometry, allows ambulatory measurements of abdominal girth over prolonged periods of time and has shown that girth is indeed greater at the end compared with the beginning of the day in many patients with IBS,¹⁵⁻¹⁷ and in some healthy subjects²³ (Fig. 2). This change in girth during the day however, is much greater in patients with IBS compared with healthy subjects, and shows a direct although weak correlation with the severity of bloating in IBS patients but not healthy subjects.^{15,17} Furthermore, comparison of the changes in girth in IBS patients with the normal 95% reference range for age and sex-matched healthy subjects shows that only approximately half of IBS patients actually physically distend beyond that seen in healthy subjects in addition to reporting that they feel bloated¹⁷ (LA Houghton & PJ Whorwell, unpublished data). Both the sensation of bloating as well as abdominal girth decrease overnight to values comparable with those observed on the previous morning.^{15-17,23} Thus the recent suggestion that the term bloating should be restricted to the subjective sensation of abdominal bloating, and distension reserved for an actual increase in abdominal girth has some validity. However, there are some problems with this especially with respect to bloating which

application of the tape measure in slightly different positions on the abdomen, or with varying degrees of tightness, particularly if the measurements are done at different times of the day. Indeed studies in which

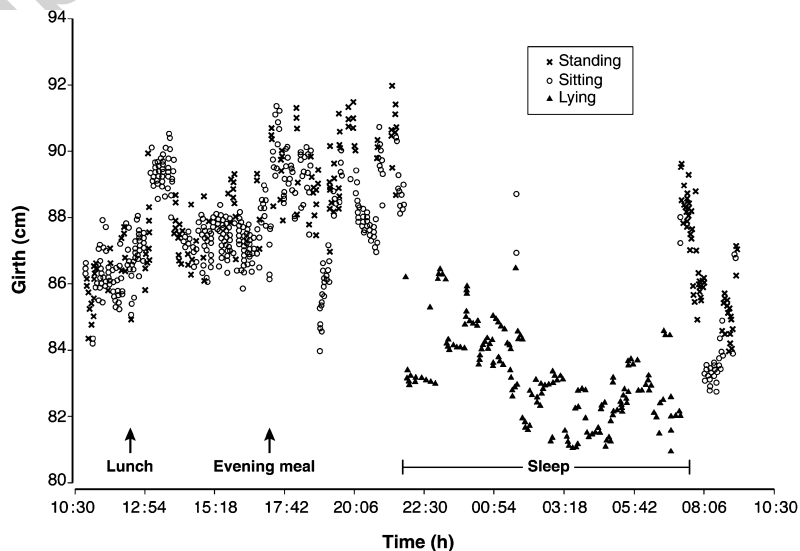


Figure 2 Typical recording of abdominal girth over 24 h in a normal healthy subject. Note the gradual increase in girth during the day, increase with meal ingestion and slow reduction in girth during sleep (reproduced by kind permission from Lewis *et al.*²³).

may or may not be associated with distension, means different things to different individuals and may not translate or even have the same meaning in different languages.

EPIDEMIOLOGY OF ABDOMINAL DISTENSION AND BLOATING

Up to 30% of the general population experience abdominal bloating,¹⁻⁴ with a significant proportion of sufferers claiming that it is so severe that it affects their daily activities (54%) or that they have had to resort to the use of medication (43%).¹ Some even seek medical advice about the problem even when it is unaccompanied by much else in the way of symptomatology (16%).¹ Healthy women are twice as likely to report distension and bloating than healthy men (19% vs 10%), and this does not appear to be related to menstruation.¹ However, bloating can be experienced perimenstrually by healthy women,²⁵ but it is unknown whether the mechanisms responsible for this form of bloating are similar to those which cause bloating at other times.

In functional gastrointestinal disorders, distension and bloating affect as much as 96% of individuals.^{5,6} Factor analyses have shown distension and bloating to cluster with IBS,^{9,26} functional constipation^{10,27} and functional dyspepsia,^{28,29} and it has also been suggested that bloating, distension, early satiety and the sensation of gastric retention should be defined as a distinct subgroup, termed functional bloating.² Wiklund *et al.*³⁰ have recently suggested a subgroup of IBS patients characterized by the symptoms of bloating, visible abdominal distension and the passage of gas, while Ragnarsson and Bodemar³¹ proposed two other subgroups of IBS, one with and the other without considerable pain or bloating. Although some studies have reported a lack of clustering with IBS,^{10,27,32,33} bloating is one of the most frequent symptoms^{8,9,34-36} and often ranked by patients as the most bothersome feature^{34,37} Bloating with visible distension is generally more common in females than males with IBS,^{5,38-40} and is reported more commonly by patients with constipation (c-IBS) rather than diarrhoea (d-IBS) predominant IBS^{5,34,35,38} However, recent evidence has suggested that the mean severity of bloating and degree of abdominal distension observed during the day is not necessarily different between bowel habit subgroups,^{16,17,31} although it is only in c-IBS patients that there is a direct correlation between the two (c-IBS $r = 0.6$; $P = 0.01$ vs d-IBS $r = 0.2$; $P = 0.42$),¹⁷ (LA Houghton & PJ Whorwell, unpublished data) suggesting that different pathophysiological mechanisms may

underlie bloating and distension in different types of IBS. Just as is in healthy women, bloating can also worsen perimenstrually in females with IBS, irrespective of bowel habit subtype^{5,12,25}

IS IT ALL IN THE MIND?

Early investigators believed that distension and bloating were all in the mind resulting from deliberate protrusion of the abdomen.^{7,41} However, only approximately one-third of patients report a relationship between bloating or distension and daily stress and anxiety,^{5,11} although some do claim that it is better when they are in a relaxed mood.⁵ Studies looking for a specific relationship between bloating or distension and psychological factors have resulted in somewhat conflicting results. In two population surveys, one reported a correlation with depression, insomnia and coping capacities⁴² and the other a link with depression, panic disorder and agoraphobia.⁴³ Heaton *et al.*⁴⁴ have reported a temporal relationship between anxiety and depression and bloating, but not other symptoms, in females with IBS. Most other studies have been unable to identify any clear-cut psychological contribution to the pathogenesis of bloating. For example, patients who present with bloating as their primary complaint are no more anxious than those who only admit to suffering from bloating on questioning.¹⁹ Similarly patients with either functional bloating or inflammatory bowel disease have elevated but similar levels of anxiety and depression when compared with healthy subjects.⁴⁵ A number of investigators have used the SCL-90-R psychometric instrument to look for any possible association between psychological factors and bloating in the various subgroups of IBS.^{5,34,35,37,38} They showed that despite bloating being more prevalent in IBS patients who are female and/or constipated, their SCL-90-R scores are no different from those who are male and/or have diarrhoea,^{34,35,38} while SCL-90-R scores were similar in IBS patients who described either bloating or pain as their most bothersome symptom³⁷ and also whether they do or do not exhibit concomitant distension.⁵ Similarly in functional dyspeptic patients who complain of fullness, bloating or belching no particular psychosocial profile has been identified.⁴⁶

IS IT ALL DUE TO GAS ACCUMULATION?

Excessive gas might seem to be the most obvious cause of bloating and distension but evidence in support of this hypothesis is far from conclusive. The gut of a normal adult contains approximately 200 mL of gas⁴⁷

and an abnormal accumulation can result from air swallowing, over production within the lumen or a failure of efficient expulsion. Studies using an argon washout technique have shown no difference in endogenous gas production or composition between patients with gas-related complaints and healthy subjects.⁴⁷ More recent investigations using a labelled sulphurhexafluoride technique^{48,49} or calorimetry⁵⁰ in patients with IBS have lent support for this view. Furthermore, despite females reporting more severe distension and bloating than males the mean daily flatus volume and frequency is no different between sexes.^{51,52} Computer tomography of the abdomen in distended IBS patients has also been unable to identify excessive volumes of intestinal gas,¹⁸ and was also able to show that distension was not due to voluntary protrusion of the abdomen, depression of the diaphragm or an exaggerated lumbar lordosis.¹⁸ Even in the few studies where increased volumes of intestinal gas have been demonstrated on plain abdominal radiographs in IBS patients, there was little or no correlation with symptoms.^{53,54} Pharmacological studies aimed at modifying gas production or flora in the gut have also been unable to show an association between gas or flatus and bloating or distension.⁵⁵⁻⁶² This is especially clearly demonstrated by the finding that the fermentable fibre, psyllium, the non-fermentable fibre, methylcellulose and lactulose all cause significant bloating but only lactulose causes increased flatus episodes and feelings of rectal gas.⁶³ It is also noteworthy that if approximately 10 times the normal amount of gas present in the gut is infused into the intestine it can result in a less than 2 cm change in abdominal girth^{48,64} (Fig. 3). This is in contrast to the average change in girth in an IBS patient during the day which is approximately twice this value and in some cases can be up to 10–12 cm.¹⁵⁻¹⁷ Thus these observations would suggest that increased volumes of gas cannot be the sole cause of distension and bloating, although it has been shown that abdominal distension does directly correlate with retained gas volume following experimental infusion^{48,64-67} (Fig. 3).

In recent years the Barcelona group have undertaken a series of novel studies examining the possibility that gas handling may be abnormal in IBS even if it is not necessarily excessive. They have shown that infusion of relatively large volumes of gas into the jejunum of normal individuals can be well tolerated as a result of rapid transit and evacuation^{48,68} (Fig. 4). In contrast, patients with IBS or functional bloating exhibit gas retention which is accompanied by a variety of abdominal symptoms including distension^{48,49,65} (Figs 4 and 5). In addition patients with constipation

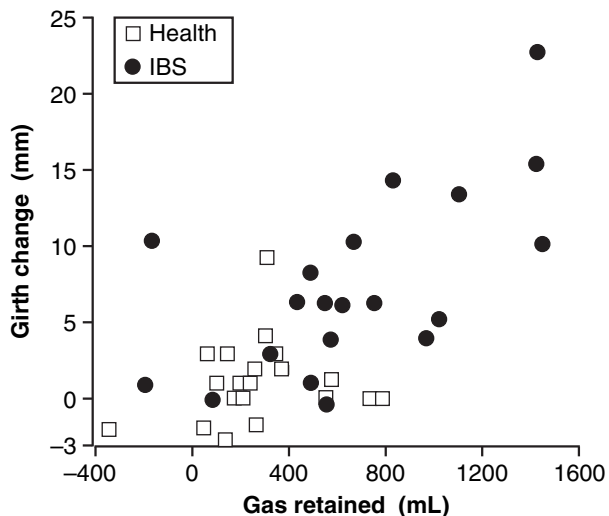


Figure 3 Individual measurements of abdominal girth (i.e. distension) and gas retention after a 2-h intestinal gas infusion in patients with IBS and healthy volunteers. Gas retained is volume infused minus volume evacuated. Data suggests that extrapolation of the data points to approximately 2000 mL gas retention would lead to a <20 mm change in girth. Note IBS patients retained greater volumes of gas and distended more than the healthy volunteers (reproduced by kind permission from Serra *et al.*⁴⁸).

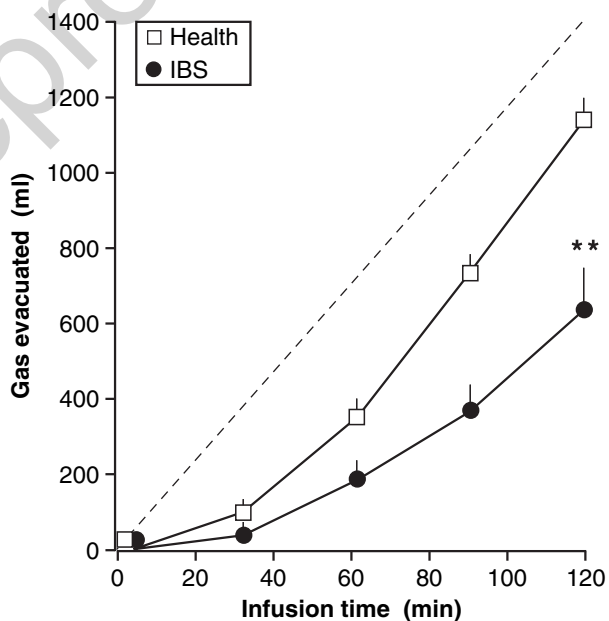


Figure 4 Evacuation of intestinal gas in patients with IBS and healthy volunteers. Gas was infused into the intestine at a constant rate (represented by the broken line) for 2 h and collected via an anal cannula. IBS patients expelled a significantly lower volume of gas than healthy volunteers. Data are expressed as mean (SEM), ***P* < 0.01 (reproduced by kind permission from Serra *et al.*⁴⁸).

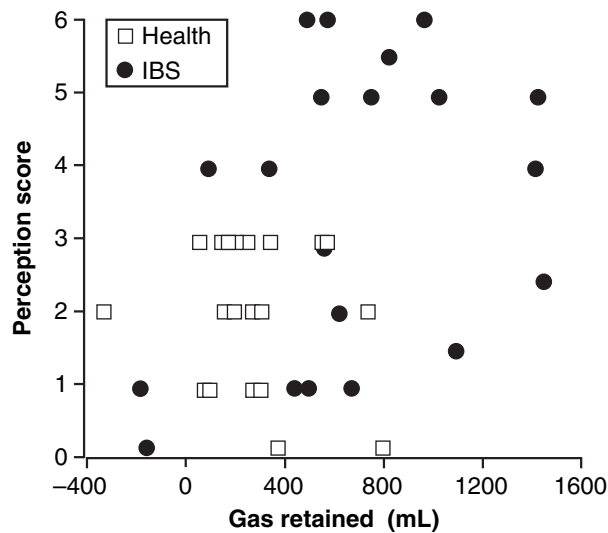


Figure 5 Individual perception scores and gas retention after 2-h intestinal gas infusion in patients with IBS and healthy volunteers. Perception of abdominal symptoms was scored on a seven-point scale (0–6). Gas retained is volume infused minus volume evacuated. Most healthy volunteers had a symptom score of <3 (mild to moderate) and retained <400 mL. In contrast, IBS patients generally retained >400 mL of gas and developed symptoms with a score >3 (moderate to severe) (reproduced by kind permission from Serra *et al.*⁴⁸).

tend to retain more gas than those with diarrhoea^{48,49} with the implication that gas handling may be related to differences in gastrointestinal transit or motility between the two patient subgroups.⁶⁹ This concept is further supported by the observation that gas retention is exacerbated by glucagon, an inhibitor of motility, and reversed by the prokinetic agent, neostigmine.^{49,70} Additional studies addressing the possible mechanism of impaired gas handling in IBS patients have shown that by comparing the expulsion of infused gas either from the anus or rectum that it cannot be due to anal retention.⁴⁸ Both by voluntarily obstructing evacuation or by relaxation of the gut with intravenous glucagon it is possible to induce gas retention.⁷⁰ However only in those who voluntarily inhibit the passage of flatus do symptoms develop,⁷⁰ suggesting that functional obstruction to the passage of gas may yet turn out to be important in the development of symptoms, such as bloating. It has also been shown that the severity of symptoms, but not distension, appears to depend on the intestinal distribution of infused gas, as a 720 mL gas load in the rectum is considerably better tolerated than a similar load in the jejunum.⁶⁷ This has been attributed to a concomitant increase in duodenal tone occurring at the time of jejunal but not rectal gas infusion, while in contrast rectal gas infusion was

associated with rectal relaxation alone.⁶⁷ Distension of both proximal^{66,71} and distal⁷¹ regions of the gut at fixed wall tension levels by means of a computerized tensostat, results in accelerated transit of infused gas and the prevention or reduction of gas retention, while the infusion of nutrients, such as intralipid into the duodenum, delays transit, increases gas retention and abdominal girth although with minimal perception in healthy subjects.⁶⁶ Ileal as opposed to duodenal intralipid appears to have an even greater effect on gas retention and abdominal girth, especially when the gas is infused into the jejunum, resulting in symptoms.⁶⁴ In patients with IBS, not only are they unable to evacuate experimentally infused gas, with the development of symptoms and abdominal distension,^{48,49,65} they also appear to be hypersensitive to a given intraduodenal lipid load, exhibiting greater gas retention, abdominal symptoms and distension than healthy subjects.⁶⁵ This is in accord with the observation that IBS patients exhibit a greater reduction in sensory threshold to gut distension during duodenal lipid infusion than do healthy subjects.⁷² Thus, based on current evidence it would appear that abnormalities of gas handling are more likely to be associated with bloating rather than distension.

WHAT'S THE EVIDENCE FOR SENSORIMOTOR DYSFUNCTION?

The observation that the sensation of bloating is not always accompanied by an actual increase in girth allows patients to be divided into two groups, those with bloating alone and those with bloating and distension. We have shown that compared with healthy subjects, patients with bloating alone have lower sensory thresholds whereas those with bloating and distension have normal or slightly higher sensory thresholds.⁷³ Interestingly bloating alone is more common in d-IBS whereas bloating accompanied by distension seems to be associated with c-IBS.^{5,34,35,38} Thus it appears that there may be more of a sensory component in patients whose sole complaint is bloating whereas mechanical factors may contribute more to distension. If this is the case it could lead to the speculation that successfully treating constipation might lead to some improvement in bloating or distension as a result of relieving the mechanical component of the problem. It is therefore of interest that treating c-IBS with the prokinetic 5-HT₄ receptor partial agonist, tegaserod seems to lead to reduction of bloating.^{74–76} Other data suggesting a role for heightened visceral sensation as contributing to the sensation of bloating includes the observation that

bloating but not distension increases during the perimenstrual phase of the menstrual cycle,⁷⁷ at which time rectal sensitivity has been shown to be increased compared with the other phases of the cycle in patients with IBS.¹³ Perimenstrual hypersensitivity may be related to the release of prostaglandins which is known to occur at this time as a result of red blood cell destruction^{78,79} and platelet activation.⁸⁰ Prostaglandins can sensitize afferent nerves^{81,82} which may compound the hypersensitivity of the gut that already exists in many patients with IBS. Alternatively, prostaglandin release can also lead to diarrhoea⁸³ which has been shown to have the potential for sensitizing the gut,⁸⁴ especially in IBS patients who are known to be more susceptible to such events compared with healthy volunteers.⁷² Whether sex hormones themselves contribute in any way to bloating and distension at times other than menses in women is unknown, but the observations that this problem maybe more prevalent after the menopause but is apparently reduced by hormone replacement therapy, supports such a role.⁸⁵ However, others have been unable to confirm these observations.⁵

The motor correlates of bloating and distension are not so clear. We did not find any changes in rectal tone or compliance in patients with premenstrual bloating¹³ although this obviously does not exclude changes elsewhere in the gut. Galati *et al.*⁸⁶ also failed to identify a difference in small intestinal motility during gas infusion in patients with IBS compared with controls despite increased perception of the gas. Likewise we were unable to show any differences in small and large bowel phasic motor activity between occasions when IBS patients felt bloated and when they did not.⁸⁷ There are some data to suggest that small bowel transit⁶⁹ and ileal emptying and clearance⁸⁸ may be delayed in IBS patients with bloating and distension, and delayed gastric emptying has been found in functional dyspeptics complaining of postprandial fullness and bloating.⁴⁶ However, other studies have reported opposing findings^{89,90} and it is probably reasonable to conclude that much more work needs to be done with respect to motor events especially as we now have better ways of defining bloating and distension.

IS IT RELATED TO INTRALUMINAL BULKING OR FLUID RETENTION INSIDE OR OUTSIDE OF THE GUT?

Hebden *et al.*⁸⁹ showed that addition of bran to a radiolabelled meal increased small bowel transit in normals but did not further accelerate an already rapid

transit in patients with IBS. This lack of affect of bran on transit in IBS patients was associated with an exacerbation of pain and bloating, which led the authors to conclude that it was caused by an increased bulking effect in the proximal colon. As with 'trapped' gas this might possibly stimulate mechanoreceptors within the gut wall, giving rise to symptoms such as bloating and there is certainly some evidence that bran can exacerbate the symptoms of IBS.⁹¹

Abdominal bloating is particularly severe perimenstrually^{12,13} and although this may in part be related to the heightened sensitivity of the gut at this time,¹³ another possible mechanism could be sex hormone induced fluid retention. However, this would not explain why there is a diurnal pattern to bloating and distension. Furthermore, other studies have been unable to account for the diurnal pattern of these symptoms by changes in body weight,^{18,19} which might be expected to increase if there was significant retention of fluid. Sequestration of fluid within bowel segments leading to stimulation of mechanoreceptors and consequently the sensation of bloating has been proposed⁹² but is unlikely to be the cause of distension as it is known from observations on ascites that relatively large fluid volumes need to be retained before there is a noticeable change in girth.⁹³ Thus fluid retention is unlikely to be involved in the development of distension, although it is conceivable that sequestration of fluid could cause the sensation of bloating.

WHAT IS THE ROLE OF FOOD INTOLERANCE OR ABNORMAL GUT FLORA?

Lactose intolerance is a common disorder and therefore it is not surprising that it is frequently found in IBS.⁹⁴ Evidence for it playing a significant role in the development of bloating however is poor, as it has been shown that IBS patients with lactase deficiency who are on a lactose-free diet have similar bloating scores to IBS patients without lactose intolerance.⁹⁵ In addition supplementing the diet of IBS lactose malabsorbers with lactase has no effect on their symptoms.⁹⁶ Furthermore, Haderstorfer *et al.*⁹⁷ have shown that bloating is more common in patients with IBS compared with subjects who just have lactose malabsorption. Malabsorption of other carbohydrates such as fructose and sorbitol is common in IBS but no more prevalent than in the general population. However their elimination from the diet in patients with IBS does sometimes lead to improvement of symptoms,^{98,99} suggesting that the IBS gut may react

differently to the consequences of carbohydrate malabsorption. It has recently been reported that IgG food antibodies may also have a role in IBS¹⁰⁰ and this may be by contributing to the low grade inflammatory process that has been demonstrated in the mucosa of some patients with the condition¹⁰¹ and which possibly contributes to heightened visceral sensation. Whether changes in bacterial flora could also contribute to this situation is unknown but it has been shown that some patients seem to have different patterns of colonization with coliforms, lactobacilli and bifidobacteria compared with controls.¹⁰² In addition, individuals who do not possess methanogenic bacteria and produce low levels of methane do experience symptoms¹⁰³ and this has been attributed to a lack of utilization of gut hydrogen, which is required in the production of methane. Interestingly, one calorimetry study has shown IBS patients to produce more hydrogen than healthy subjects, although total gas volume was normal.⁵⁰ It has also been suggested that methane production is associated with constipation in IBS.¹⁰⁴ So far attempts to modify gut flora by the use of probiotics have proved disappointing^{58,62} in terms of relieving symptoms although one study did show an effect on bloating.⁶² However it should be pointed out that there are a very large number of potential probiotic strains with varying activity and recently Quigley *et al.*¹⁰⁵ have identified a promising bifidobacteria. Attempts to alter gut flora by the use of antibiotics has resulted in no improvement in bloating,^{57,60,61} although the non-absorbable antibiotic rifaximin is associated with a reduction in number of flatus episodes and abdominal girth.⁵⁷

IS IT DUE TO WEAK ABDOMINAL MUSCULATURE?

Patients with abdominal distension have been reported to be more likely to have recently gained weight, do little regular exercise, have weak abdominal muscles and increased girth during episodes of distension.¹⁹ The concept that abdominal distension may be related to dysfunction of the anterior abdominal musculature is also supported by the recent observation that rectal gas infusion in patients with abdominal bloating was associated with failed muscular contraction and paradoxical relaxation of the internal oblique abdominal muscle on electromyography (EMG) compared with the increase in muscular tone seen in healthy subjects.¹⁰⁶ EMG studies under non-gas infused conditions have been unable to show a difference in recordings between IBS patients with a history of visible distension and healthy subjects.¹⁰⁷ Furthermore, age which

might be expected to be associated with general weakening of the abdominal musculature, is not related to increased bloating;^{4,42,108} although there is no data on its relationship to measurable abdominal distension. More research is required in this area, but it would seem reasonable to assume that weakness of the abdominal musculature might be expected to be related to abdominal distension, but it is unlikely to have much effect on the symptom of bloating.

DIFFERENTIAL DIAGNOSIS

Organic conditions such as ascites or abdominal malignancy, particularly ovarian cancer can present with distension although this usually differs from the functional variety in that it does not exhibit diurnal variation. In contrast, intermittent subacute obstruction can present with fluctuating distension which may sometimes lead to diagnostic uncertainty although there is usually a history of previous abdominal surgery or some other predisposing cause such as Crohn's disease. On examination, it is usually relatively easy to differentiate distension from ascites but this can be difficult in more obese subjects. It is also important to remember that superficially, a large ventral hernia can mimic distension unless the integrity of the rectus abdominis is assessed. However, if there is any doubt about the diagnosis especially if the abdomen is very prominent and the problem fails to subside at night, it is sensible to undertake a scan or any other relevant investigation to eliminate possibilities other than a functional gastrointestinal disorder.

SO IS THERE A TREATMENT?

It is clear that there is growing evidence that although bloating and distension overlap they do not always co-exist. Furthermore their pathophysiology may, at least in part, be different with bloating probably having more of a sensory component involving complex interactions between local sensory mechanisms and central perception. In contrast distension could be more of a mechanical problem. However, until the exact mechanisms involved are better delineated treatment will necessarily be empirical.

It is quite common for cereal fibre, which is so frequently advocated for IBS, to actually make the symptoms, including bloating, worse.⁹¹ It might be expected that the exacerbation might only occur when supplementation is first initiated but this is not our experience and therefore this approach should not be pursued if it is clearly not leading to any improvement.

Similarly, it is worthwhile offering dietary advice about how a patient may want to try avoiding lactose and fructose as well as vegetables, such as beans and broccoli, that can lead to excessive gas.¹⁰⁹ Beano which aids the digestion of complex carbohydrates may reduce flatulence but is disappointing with respect to bloating.¹¹⁰ There is one report suggesting that pancreatic enzyme supplementation may be useful¹¹¹ and probiotics certainly do no harm, although so far they have proved rather disappointing.^{58,62} Surfactants, such as simethicone have shown some benefit in some but not all studies,^{55,112,113} while activated charcoal,^{55,114} exercise and weight loss^{92,115} have shown to be of little benefit.

With respect to pharmacological approaches, it is only relatively recently that bloating or distension has been included as end points in clinical trials. Therefore there is little or no data on the effect of the more established medications on these particular symptoms.¹¹⁶ The more recently introduced 5-HT₃ receptor antagonist, alosetron did not seem to improve bloating¹¹⁷ although the kappa receptor agonist, fedotozine did appear to have some effect but this particular drug is not being developed further.¹¹⁸ One of the most promising drugs for improving bloating is the 5-HT₄ receptor partial agonist, tegaserod which has shown this effect in a number of trials.⁷⁴⁻⁷⁶ It is tempting to speculate that this might be as a result of its prokinetic activity, as bloating and distension seem to be most closely linked in constipated patients.^{5,17,33,34,37} Non-absorbable antibiotics may have some beneficial effects on bloating⁵⁷ but there would be concerns about their long-term usage.

Lastly, hypnotherapy has been shown to relieve the symptoms of functional dyspepsia and IBS including bloating¹¹⁹⁻¹²⁵ and this may be as a result of its beneficial effect on visceral sensitivity.^{126,127} Whether other behavioural treatments have similar effects has yet to be determined.

CONCLUSION

The reporting of the sensation of abdominal bloating does not necessarily imply a concomitant physical increase in abdominal girth, and indeed this is the case for 50% of patients with IBS. An understanding that abdominal distension represents a true change in abdominal girth, while bloating may mean different things to different patients within the same or different functional gastrointestinal disorder and even across different cultures and languages is required. Further research correctly differentiating between these two terms, together with the use of objective methods to measure abdominal girth are likely to identify some of

the pathophysiological processes involved and consequently hopefully result in better ways of managing these often debilitating complaints. However, it is unlikely that distension and bloating are the result of a single pathophysiological process but are multifactorial in origin and consequently successful treatment is unlikely to be uniform from patient to patient. We are making progress but there is much more to be done.

REFERENCES

- 1 Sandler RS, Stewart WF, Liberman JN, Ricci JA, Zorich NL. Abdominal pain, bloating, and diarrhoea in the United States. Prevalence and impact. *Dig Dis Sci* 2000; **45**: 1166-71.
- 2 Talley NJ, Boyce P, Jones M. Identification of distinct upper and lower gastrointestinal symptom groupings in an urban population. *Gut* 1998; **42**: 690-95.
- 3 Kay L, Jorgensen T, Jensen KH. The epidemiology of irritable bowel syndrome in a random population: prevalence, incidence, natural history and risk factors. *J Intern Med* 1994; **236**: 23-30.
- 4 Drossman DA, Li Z, Andruzzi E, Temple R *et al*. US householder survey of functional gastrointestinal disorders: prevalence, sociodemography and health impact. *Dig Dis Sci* 1993; **38**: 1569-80.
- 5 Chang L, Lee O-Y, Naliboff B, Schmulson M, Mayer EA. Sensation of bloating and visible abdominal distension in patients with irritable bowel syndrome. *Am J Gastroenterol* 2001; **96**: 3341-47.
- 6 Talley NJ, Phillips SF, Melton LJ, Wiltgen C, Zinsmeister R. A patient questionnaire to identify bowel disease. *Ann Intern Med* 1989; **111**: 671-4.
- 7 Zar S, Benson MJ, Kumar D. Review article: bloating in functional bowel disorders. *Aliment Pharmacol Ther* 2002; **16**: 1867-76.
- 8 Hungin APS, Whorwell PJ, Tack J, Mearns F. The prevalence, patterns and impact of irritable bowel syndrome: an international survey of 40,000 subjects. *Aliment Pharmacol Ther* 2003; **17**: 643-50.
- 9 Manning AP, Thompson WG, Heaton KW, Morris AF. Towards positive diagnosis of the irritable bowel. *Br Med J* 1978; **2**: 653-4.
- 10 Talley NJ, Holtmann G, Agreus L, Jones M. Gastrointestinal symptoms and subjects cluster into distinct upper and lower groupings in the community: a four nations study. *Am J Gastroenterol* 2000; **95**: 1439-47.
- 11 Maxton DG, Whorwell PJ. Abdominal distension in irritable bowel syndrome: the patients perspective. *Eur J Gastroenterol Hepatol* 1992; **4**: 241-3.
- 12 Heitkemper MM, Cain KC, Jarrett ME, Burr RL, Hertig V, Bond EF. Symptoms across the menstrual cycle in women with irritable bowel syndrome. *Am J Gastroenterol* 2003; **98**: 420-30.
- 13 Houghton LA, Lea R, Jackson NA, Whorwell PJ. The menstrual cycle affects rectal sensitivity in patients with irritable bowel syndrome but not healthy volunteers. *Gut* 2002; **50**: 471-4.
- 14 Westbrook JI, Talley NJ. Empiric clustering of dyspepsia into symptom sub-groups: a population based study. *Scand J Gastroenterol* 2002; **37**: 917-23.

- 15 Lea R, Whorwell PJ, Reilly B, Houghton LA. Abdominal distension in irritable bowel syndrome (IBS): diurnal variation and its relationship to abdominal bloating. *Gut* 2003; **32** (Suppl. VI): A32.
- 16 Lea R, Houghton LA, Whorwell PJ, Reilly B. Relationship of abdominal bloating to physical distension on irritable bowel syndrome (IBS): effect of bowel habit. *Neurogastroenterol Motil* 2003; **15**: 587.
- 17 Lea R, Reilly B, Whorwell PJ, Houghton LA. The effect of bowel habit and time of day on the relationship between bloating and visible distension in patients with irritable bowel syndrome. *Gastroenterology* 2004; **126**: 1634.
- 18 Maxton DG, Martin DF, Whorwell PJ, Godfrey M. Abdominal distension in female patients with irritable bowel syndrome: exploration of possible mechanisms. *Gut* 1991; **32**: 662–4.
- 19 Sullivan SN. A prospective study of unexplained visible abdominal bloating. *NZ Med J* 1994; **107**: 428–30.
- 20 Aitken RJ, Clifford PC. Girth measurement is not a reliable investigation for the detection of intra-abdominal fluid. *Ann R Coll Surg Engl* 1985; **67**: 241–2.
- 21 Castren M, Liukka K, Nurmi J, Honkanen E, Lindgren L. Measurement of the abdominal circumference for the detection of intra-abdominal hemorrhage has no diagnostic value. *Acta Anaesthesiol Scand* 2004; **48**: 592–4.
- 22 Basilisco G, Marino B, Passerini L, Ogliari C. Abdominal distension after colonic lactulose fermentation recorded by a new extensometer. *Neurogastroenterol Motil* 2003; **15**: 427–33.
- 23 Lewis M, Reilly B, Houghton LA, Whorwell PJ. Ambulatory abdominal inductance plethysmography: towards objective assessment of abdominal distension in irritable bowel syndrome. *Gut* 2001; **48**: 216–20.
- 24 Reilly B, Bolton M, Houghton LA, Lewis M, Whorwell PJ. A device for 24 hour ambulatory monitoring of abdominal girth using inductive plethysmography. *Physiol Meas* 2002; **23**: 661–70.
- 25 Heitkemper MM, Jarrett M, Cain KC, Shaver J, Walker E, Lewis L. Daily gastrointestinal symptoms in women with and without a diagnosis of IBS. *Dig Dis Sci* 1995; **40**: 1511–9.
- 26 Neri M, Laterza F, Howell S *et al.* Symptoms discriminate irritable bowel syndrome from organic gastrointestinal diseases and food allergy. *Eur J Gastroenterol Hepatol* 2000; **12**: 981–8.
- 27 Shaw M, Talley NJ, Adlis S, Beebe T, Tomshine P, Healey M. Development of a digestive health status instrument: tests of scaling assumptions. Structure and reliability in a primary care population. *Aliment Pharmacol Ther* 1998; **12**: 1067–78.
- 28 Schlemper RJ, van der Werf SD, Vandebrouke JP, Biemond I, Lamers CB. Peptic ulcer, non-ulcer dyspepsia and irritable bowel syndrome in The Netherlands and Japan. *Scand J Gastroenterol Suppl* 1993; **200**: 33–41.
- 29 Kwan AC-P, Bao TN, Chakkaphak S *et al.* Functional gastrointestinal disorders. Validation of Rome II criteria for functional gastrointestinal disorders by factor analysis of symptoms in Asian patient sample. *J Gastroenterol Hepatol* 2003; **18**: 796–802.
- 30 Wiklund IK, Fullerton S, Hawkey CJ *et al.* An irritable bowel symptom-specific symptom questionnaire: development and validation. *Scand J Gastroenterol* 2003; **38**: 947–54.
- 31 Ragnarsson G, Bodemar G. Division of the irritable bowel syndrome into subgroups on the basis of daily recorded symptoms into two outpatient samples. *Scand J Gastroenterol* 1999; **34**: 993–1000.
- 32 Taub E, Cuevas JL, Cook EW, Crowell MD, Whitehead WE. Irritable bowel syndrome defined by factor analysis. Gender and race comparisons. *Dig Dis Sci* 1995; **40**: 2647–55.
- 33 Whitehead WE, Crowell MD, Bosmajian L *et al.* Existence of irritable bowel syndrome supported by factor analysis of symptoms in two community samples. *Gastroenterology* 1990; **98**: 336–40.
- 34 Schmulson M, Lee O-Y, Chang L, Naliboff B, Mayer EA. Symptom difference in moderate to severe IBS patients based on predominant bowel habit. *Am J Gastroenterol* 1999; **94**: 2929–35.
- 35 Lee O-Y, Mayer EA, Schmulson M, Chang L, Naliboff B. Gender-related differences in IBS symptoms. *Am J Gastroenterol* 2001; **96**: 2184–93.
- 36 Maxton DG, Morris JA, Whorwell PJ. Ranking of symptoms by patients with irritable bowel syndrome. *Br Med J* 1989; **299**: 1138.
- 37 Lembo T, Naliboff B, Munakata J *et al.* Symptoms and visceral perception in patients with pain-predominant irritable bowel syndrome. *Am J Gastroenterol* 1999; **94**: 1320–6.
- 38 Talley NJ, Dennis EH, Schettler-Duncan VA, Lacy BE, Olden KW, Crowell MD. Overlapping upper and lower gastrointestinal symptoms in irritable bowel syndrome patients with constipation or diarrhea. *Am J Gastroenterol* 2003; **98**: 2454–59.
- 39 Thompson WG. Gender differences in irritable bowel syndrome. *Eur J Gastroenterol Hepatol* 1997; **9**: 229–302.
- 40 Smith RC, Greenbaum DS, Vancouver JB *et al.* Gender differences in Manning criteria in the irritable bowel syndrome. *Gastroenterology* 1991; **100**: 591–95.
- 41 Alvarez W. Hysterical type of nongaseous abdominal bloating. *Arch Intern Med* 1949; **84**: 217–45.
- 42 Johnsen R, Jacobsen BK, Forde OH. Associations between symptoms of irritable colon and psychological and social conditions and lifestyle. *Br Med J* 1986; **292**: 1633–5.
- 43 Walker EA, Katon WJ, Jemelka RP, Roy-Byrne PP. Comorbidity of gastrointestinal complaints, depression, and anxiety in the epidemiologic catchment area (ECA) study. *Am J Med* 1992; **92**: 26S–30S.
- 44 Heaton KW, Ghosh S, Braddon FEM. How bad are the symptoms and bowel dysfunction of patients with the irritable bowel syndrome? A prospective, controlled study with emphasis on stool form. *Gut* 1991; **32**: 73–9.
- 45 Song JY, Merskey H, Sullivan S, Noh S. Anxiety and depression in patients with abdominal bloating. *Can J Psychiatry* 1993; **38**: 475–9.
- 46 Fischler B, Tack J, De Gucht V *et al.* Heterogeneity of symptom pattern, psychosocial factors, and pathophysiological mechanisms in severe functional dyspepsia. *Gastroenterology* 2003; **124**: 903–10.
- 47 Lasser RB, Bond JH, Levitt MD. The role of intestinal gas in functional abdominal pain. *N Engl J Med* 1975; **293**: 524–26.
- 48 Serra J, Azpiroz F, Malagelada J-R. Impaired transit and tolerance of intestinal gas in the irritable bowel syndrome. *Gut* 2001; **48**: 14–19.

- 49 Caldarella M, Serra J, Azpiroz F *et al.* Prokinetic effects in patients with intestinal gas retention. *Gastroenterology* 2002; **122**: 1748–55.
- 50 King TS, Elia M, Hunter JO. Abnormal colonic fermentation in irritable bowel syndrome. *Lancet* 1998; **352**: 1187–89.
- 51 Tomlin J, Lewis C, Read NW. Investigation of normal flatus production in healthy volunteers. *Gut* 1991; **32**: 665–9.
- 52 Furne JK, Levitt MD. Factors influencing frequency of flatus emission by healthy subjects. *Dig Dis Sci* 1996; **41**: 1631–5.
- 53 Chami TN, Schuster MM, Bohlman M *et al.* A simple radiologic method to estimate the quantity of bowel gas. *Am J Gastroenterol* 1991; **86**: 599–602.
- 54 Koide A, Yamaguchi T, Odaka T *et al.* Quantitative analysis of bowel gas using plain abdominal radiograph in patients with irritable bowel syndrome. *Am J Gastroenterol* 2000; **95**: 1735–41.
- 55 Jain NK, Patel VP, Pitchumoni S. Activated charcoal, simethicone, and intestinal gas: a double-blind study. *Ann Intern Med* 1986; **105**: 61–2.
- 56 Fardy J, Sullivan S. Gastrointestinal gas. *CMAJ* 1988; **139**: 1137–42.
- 57 Di Stefano M, Strocchi A, Malservisi S, Veneto G, Ferrieri A, Corazza GR. Non-absorbable antibiotics for managing intestinal gas production and gas-related symptoms. *Aliment Pharmacol Ther* 2000; **14**: 1001–8.
- 58 Nobaek S, Johansson ML, Molin G, Ahrne S, Jeppsson B. Alteration of intestinal microflora is associated with reduction in abdominal bloating and pain in patients with irritable bowel syndrome. *Am J Gastroenterol* 2000; **95**: 1231–8.
- 59 O'Sullivan MA, O'Morain CA. Bacterial supplementation in irritable bowel syndrome. A randomised double-blind placebo-controlled crossover study. *Dig Liver Dis* 2000; **32**: 294–301.
- 60 Pimentel M, Chow EJ, Lin HC. Eradication of small intestinal bacterial overgrowth reduces symptoms of irritable bowel syndrome. *Am J Gastroenterol* 2000; **95**: 3503–6.
- 61 Pimentel M, Chow EJ, Lin HC. Normalization of lactulose breath testing correlates with symptom improvement in irritable bowel syndrome. A double-blind, randomized, placebo-controlled study. *Am J Gastroenterol* 2003; **98**: 412–9.
- 62 Kim HJ, Camilleri M, McKinzie S *et al.* A randomized controlled trial of a probiotic, VSL 3, on gut transit and symptoms in diarrhea-predominant irritable bowel syndrome. *Aliment Pharmacol Ther* 2003; **17**: 895–904.
- 63 Levitt MD, Furne J, Olsson S. The relation of passage of gas and abdominal bloating to colonic gas production. *Ann Intern Med* 1996; **124**: 422–4.
- 64 Hernando-Harder AC, Serra J, Azpiroz F, Malagelada J-R. Sites of symptomatic gas retention during intestinal lipid perfusion in healthy subjects. *Gut* 2004; **53**: 661–5.
- 65 Serra J, Salvioli B, Azpiroz F *et al.* Lipid-induced intestinal gas retention in irritable bowel syndrome. *Gastroenterology* 2002; **123**: 700–6.
- 66 Serra J, Azpiroz F, Malagelada J-R. Gastric distension and duodenal lipid infusion modulate intestinal gas transit and tolerance in humans. *Am J Gastroenterol* 2002; **97**: 2225–30.
- 67 Harder H, Serra J, Azpiroz F, Passos MC, Aguade S, Malagelada J-R. Intestinal gas distribution determines abdominal symptoms. *Gut* 2003; **52**: 1708–13.
- 68 Serra J, Azpiroz F, Malagelada J-R. Intestinal gas dynamics and tolerance in humans. *Gastroenterology* 1998; **115**: 542–50.
- 69 Cann PA, Read NW, Brown C, Hobson N, Holdsworth CD. Irritable bowel syndrome: relationship of disorders in the transit of a single solid meal to symptom patterns. *Gut* 1983; **24**: 405–11.
- 70 Serra J, Azpiroz F, Malagelada J-R. Mechanisms of intestinal gas retention in humans: impaired propulsion versus obstructed evacuation. *Am J Physiol* 2001; **281**: G138–43.
- 71 Harder H, Serra J, Azpiroz F, Malagelada J-R. Reflex control of intestinal gas dynamics and tolerance in humans. *Am J Physiol* 2004; **286**: G89–94.
- 72 Simren M, Abrahamsson H, Bjornsson ES. An exaggerated sensory component of the gastrocolonic response in patients with irritable bowel syndrome. *Gut* 2001; **48**: 20–27.
- 73 Lea R, Reilly B, Whorwell PJ, Houghton LA. Abdominal bloating in the absence of physical distension is related to increased visceral sensitivity. *Gastroenterology* 2004; **126** (Suppl. 2): 432.
- 74 Muller-Lissner SA, Fumagalli I, Bardhan KD *et al.* Tegaserod, a 5-HT₄ receptor partial agonist, relieves symptoms in irritable bowel syndrome patients with abdominal pain, bloating and constipation. *Aliment Pharmacol Ther* 2001; **15**: 1655–66.
- 75 Novick J, Miner P, Krause R *et al.* A randomized, double-blind, placebo-controlled trial of tegaserod in female patients suffering from irritable bowel syndrome with constipation. *Aliment Pharmacol Ther* 2002; **16**: 1877–88.
- 76 Kellow J, Lee OY, Chang FY *et al.* An Asia-Pacific, double blind, placebo controlled randomized study to evaluate the efficacy, safety, and tolerability of tegaserod in patients with irritable bowel syndrome. *Gut* 2003; **52**: 671–76.
- 77 Lea R, Reilly B, Whorwell PJ, Houghton LA. Peri-menstrual bloating in patients with irritable bowel syndrome is not associated with abdominal distension and may be related to visceral hypersensitivity. *Gastroenterology* 2004; **126** (Suppl. 2): M1611.
- 78 Ramwell PW, Shaw JE. Biological significance of prostaglandins. *Recent Prog Horm Res* 1970; **26**: 139–87.
- 79 Schwertz A, Zor U, Linder HR. Primary dysmenorrhea: alleviation by an inhibitor of prostaglandin synthesis and action. *Obstet Gynaecol* 1974; **44**: 709–12.
- 80 Harris RH, Ramswell PW. Cellular mechanisms of prostaglandin action. *Annu Rev Physiol* 1979; **41**: 653–68.
- 81 Cohen RH, Perl ER. Contributions of arachidonic acid derivatives and substance P to the sensitization of cutaneous nociceptors. *J Neurophysiol* 1990; **64**: 457–64.
- 82 Crunkhorn P, Willis AL. Cutaneous reactions to intradermal prostaglandins. *Br J Pharmacol* 1971; **41**: 49–56.
- 83 Arthur C, Ament ME, Song MK. Prostaglandin metabolism in relation to bowel habit of women. *Prostaglandins Leukot Essent Fatty Acids* 1992; **46**: 257–9.
- 84 Houghton LA, Wych J, Whorwell PJ. Acute diarrhoea induces rectal sensitivity in women but not men. *Gut* 1995; **37**: 270–3.

- 85 Lewis M, Houghton LA, Whorwell PJ. Abdominal distension in pre- and post-menopausal females with irritable bowel syndrome (IBS): the effect of contraceptive pill and hormone replacement therapy. *Gastroenterology* 2000; **118**: 140, 832.
- 86 Galati JS, McKee DP, Quigley EM. Response to intraluminal gas in irritable bowel syndrome. Motility versus perception. *Dig Dis Sci* 1995; **40**: 1381–7.
- 87 Lewis MJV, Houghton LA, Whorwell PJ. Changes in small and large bowel phasic activity do not explain the increased perception of distension in patients with IBS. *Gut* 2001; **49** (Suppl. III): 3088.
- 88 Trotman IF, Price CC. Bloating irritable bowel syndrome defined by dynamic ^{99m}Tc bran scan. *Lancet* 1986; **2**: 364–6.
- 89 Hebden JM, Blackshaw E, D'Amato M, Perkins AC, Spiller RC. Abnormalities of GI transit in bloated irritable bowel syndrome: effect of bran on transit and symptoms. *Am J Gastroenterol* 2002; **97**: 2315–20.
- 90 Hutchinson R, Notghi A, Smith NB *et al.* Scintigraphic measurement of ileocaecal transit in irritable bowel syndrome and chronic idiopathic constipation. *Gut* 1995; **35**: 585–9.
- 91 Francis CY, Whorwell PJ. Bran and irritable bowel syndrome: time for reappraisal. *Lancet* 1994; **344**: 39–40.
- 92 Sullivan SN. Functional abdominal bloating. *J Clin Gastroenterol* 1994; **19**: 23–7.
- 93 Cattau EI, Benjamin SB, Knuff TE *et al.* The accuracy of the physical exam in the diagnosis of suspected ascites. *JAMA* 1982; **247**: 1164.
- 94 Tolliver BA, Herrera JL, DiPalma JA. Evaluation of patients who meet clinical criteria for irritable bowel syndrome. *Am J Gastroenterol* 1994; **89**: 176–8.
- 95 Tolliver BA, Jackson MS, Jackson KL, Barnett ED, Chastang JF, DiPalma JA. Does lactose maldigestion really play a role in the irritable bowel? *J Clin Gastroenterol* 1996; **23**: 15–7.
- 96 Lisker R, Solomons NW, Perez BR, Ramirez MM. Lactase and placebo in the management of the irritable bowel syndrome: a double-blind, cross-over study. *Am J Gastroenterol* 1989; **84**: 756–62.
- 97 Haderstorfer B, Psycholgin D, Whitehead WE, Schuster MM. Intestinal gas production from bacterial fermentation of undigested carbohydrate in irritable bowel syndrome. *Am J Gastroenterol* 1989; **84**: 375–8.
- 98 Fernandez-Banares F, Esteve-Pardo M, de Lean R *et al.* Sugar malabsorption in functional bowel disease: clinical implications. *Am J Gastroenterol* 1993; **88**: 2044–50.
- 99 Goldstein R, Braverman D, Stankiewicz H. Carbohydrate malabsorption and the effect of dietary restriction on symptoms in irritable bowel syndrome and functional bowel complaints. *Isr Med Assoc J* 2000; **2**: 583–7.
- 100 Atkinson W, Sheldon TA, Shaath N, Whorwell PJ. Food elimination based on IgG antibodies in IBS: a randomized controlled trial. *Gut* 2004; **53**: 1459–64.
- 101 Collins SM, Piche T, Rampal P. The putative role of inflammation in the irritable bowel syndrome. *Gut* 2001; **49**: 743–5.
- 102 Balsari A, Ceccarelli A, Dubini F, Fesce E, Poli G. The fecal microbial population in the irritable bowel syndrome. *Microbiologica* 1982; **5**: 185–94.
- 103 Kajs TM, Fitzgerald JA, Buckner RY *et al.* Influence of methanogenic flora on breath H₂ and symptom response to ingestion of sorbitol or oat fiber. *Am J Gastroenterol* 1997; **92**: 89–94.
- 104 Pimentel M, Mayer AG, Park S, Chow EJ, Hasan A, Kong Y. Methane production during lactulose breath test is associated with gastrointestinal disease presentation. *Dig Dis Sci* 2003; **48**: 86–92.
- 105 Quigley E, O'Mahony L, McCarthy J *et al.* Probiotics for the irritable bowel syndrome (IBS): A randomized, double-blind, placebo-controlled comparison of *Lactobacillus* and *Bifidobacterium* strains. *Gastroenterology* 2002; **112** (Suppl.): A59, 498.
- 106 Tremolaterra F, Serra J, Azpiroz F, Villoria A, Malagelada J-R. Bloating and abdominal wall dystonia. *Gastroenterology* 2004; **126** (Suppl. 2): 431.
- 107 McManis PG, Newall D, Talley NJ. Abdominal wall muscle activity in irritable bowel syndrome with bloating. *Am J Gastroenterol* 2001; **96**: 1139–42.
- 108 Heaton KW, Radvan J, Cripps H, Mountford RA, Braddon FE, Hughes AO. Defecation frequency and timing, and stool form in the general population: a prospective study. *Gut* 1992; **33**: 818–24.
- 109 Nanda R, James R, Smith H, *et al.* Food intolerance and the irritable bowel syndrome. *Gut* 1989; **30**: 1099–104.
- 110 Ganiats TG, Norcross WA, Halverson AL, *et al.* Does Beano prevent gas? A double-blind, crossover study of oral alpha-galactosidase to treat dietary oligo-saccharide intolerance. *J Fam Pract* 1994; **39**: 441–5.
- 111 Suarez F, Levitt MD, Adshear J, Barkin JS. Pancreatic supplements reduce symptomatic response of healthy subjects to a high fat meal. *Dig Dis Sci* 1999; **44**: 1317–21.
- 112 Friis H, Bode S, Rumessen JJ, Gudmand-Hoyer E. Effect of simethicone on lactulose-induced H₂ production and gastrointestinal symptoms. *Digestion* 1991; **49**: 227–30.
- 113 Holtmann G, Gschossmann J, Karaus M *et al.* Randomised double-blind comparison of simethicone with cispripide in functional dyspepsia. *Aliment Pharmacol Ther* 1999; **13**: 1459–65.
- 114 Suarez FL, Furne J, Springfield J, *et al.* Failure of activated charcoal to reduce the release of gases produced by the colonic flora. *Am J Gastroenterol* 1999; **94**: 208–12.
- 115 Rao SS. Belching, bloating, and flatulence. How to help patients who have troublesome abdominal gas. *Postgrad Med* 1997; **101**: 263–9.
- 116 Fardy J, Sullivan SN. Recent advances in pharmacotherapy: gastrointestinal gas. *Can Med Assoc J* 1988; **139**: 1137–42.
- 117 Camilleri M, Chey WY, Mayer EA *et al.* A randomized controlled clinical trial of the serotonin type 3 receptor antagonist alosetron in women with diarrhea-predominant irritable bowel syndrome. *Arch Intern Med* 2001; **161**: 1733–40.
- 118 Dapoigny M, Abitbol JL, Fraïtag B. Efficacy of peripheral kappa agonist fedotozine versus placebo in treatment of irritable bowel syndrome. A multicenter dose-response study. *Dig Dis Sci* 1995; **40**: 2244–9.
- 119 Whorwell PJ, Prior A, Faragher EB. Controlled trial of hypnotherapy in the treatment of severe refractory irritable bowel syndrome. *Lancet* 1984; **ii**: 1232–4.
- 120 Whorwell PJ, Prior A, Colgan SM. Hypnotherapy in severe irritable bowel syndrome: further experience. *Gut* 1987; **28**: 423–5.
- 121 Houghton LA, Heyman DJ, Whorwell PJ. Symptomatology, quality of life and economic features of irritable

- bowel syndrome – the effect of hypnotherapy. *Aliment Pharmacol Ther* 1996; **10**: 91–5.
- 122 Gonsalkorale WM, Houghton LA, Whorwell PJ. Hypnotherapy in irritable bowel syndrome: a large-scale audit of a clinical service with examination of factors influencing responsiveness. *Am J Gastroenterol* 2002; **97**: 954–61.
- 123 Gonsalkorale WM, Miller V, Afzal A, Whorwell PJ. Long term benefits of hypnotherapy for irritable bowel syndrome. *Gut* 2003; **52**: 1623–29.
- 124 Palsson OS, Turner MJ, Johnson DA, Burnelt CK, Whitehead WE. Hypnosis treatment for severe irritable bowel syndrome: investigation of mechanism and effects on symptoms. *Dig Dis Sci* 2002; **47**: 2605–14.
- 125 Calvert EL, Houghton LA, Cooper P, Morris J, Whorwell PJ. Long-term improvement in functional dyspepsia using hypnotherapy. *Gastroenterology* 2002; **123**: 1778–85.
- 126 Lea R, Houghton LA, Calvert EL *et al*. Gut focused hypnotherapy normalizes rectal hyper-sensitivity in patients with irritable bowel syndrome. *Aliment Pharmacol Ther* 2003; **17**: 635–42.
- 127 Prior A, Colgan SM, Whorwell PJ. Changes in rectal sensitivity after hypnotherapy in patients with irritable bowel syndrome. *Gut* 1990; **31**: 896–8.