An Understanding of Excessive Intestinal Gas

*Fabrizis L. Suarez, MD, PhD and Michael D. Levitt, MD**

Address
Minneapolis VA Medical Center (151), 1 Veterans Drive, Minneapolis, MN 55417, USA.

Current Gastroenterology Reports 2000, **2**:413–419
Current Science Inc. ISSN 1522–8037

Complaints of "excessive gas" from patients are very common but are difficult, if not impossible, for the physician to document. This review addresses the pathophysiology and management of such complaints, looking at the sources and routes of elimination, excessive eructation, bloating, and distention. In addition, common flatulence problems are summarized, including escessive flatus volume and noxious flatus.

Introduction

Complaints concerning what a patient assumes to be excessive gas in the stomach or intestines are extremely common. These complaints take the form of excessive eructation, bloating and distention, or abnormally voluminous or noxious flatus. Unfortunately, it is virtually impossible for the physician to objectively document the existence of excessive gas using currently available tests. Rather than admitting defeat in this regard, most physicians tend to order a series of biochemical, roentgenographic, and endoscopic studies that contribute little to the diagnosis or treatment of the problem. In addition, most gaseous problems are not readily treated. Given this unfortunate scenario, even the most astute and compassionate physician frequently feels frustrated when confronted with the patient complaining of "gas." Although we make no claims regarding our ability to cure most of these patients, this review provides a brief discussion of what is known about the pathophysiology of the various gas problems. Based on this information, we outline what we believe to be the most cost-effective approach to the management of patients complaining of "too much gas."

Sources and Routes of Elimination of Bowel Gas

The quantitatively important gases in the human gut are nitrogen (N_2), oxygen (O_2), hydrogen (H_2), carbon dioxide (CO_2), and methane (CH_4) [1,2]. These gases, which comprise more than 99% of the intestinal gas volume, are odorless. The characteristic unpleasant odor of intestinal gas appears to result primarily from the presence of trace gases that contain sulfur, such as hydrogen sulfide (H_2S), methanethiol (CH_3SH), and dimethylsulfide (CH_3SCH_3) [3].

Four mechanisms deliver gases to the intestinal lumen: 1) air swallowing (O_2 and N_2); 2) interaction of bicarbonate and acid (CO_2); 3) diffusion from the blood (CO_2, N_2, and O_2); and 4) bacterial metabolism (CO_2, H_2, CH_4, and a variety of trace gases including the sulfur-containing gases). Gases can be removed from the gut via convection out the esophagus or anus, diffusion into the blood, and consumption by bacteria. The net of these processes proximal to a given site in the gut determines the volume and composition of gas passing that site; the net of these processes throughout the entire gut determines the volume and mean composition of the entire gastrointestinal gas volume.

Excessive Eructation

Pathophysiology

Although patients usually believe that their excessive eructation reflects abnormal gas production in the stomach, analyses indicate that virtually all gastric gas is of atmospheric origin (N_2 and O_2) [4]. Consequently, belching is a manifestation of air swallowing. Air can enter the stomach in association with the swallowing of liquids or solids. Studies using ultrafast computed tomography show that a mean of 17 mL of air accompanies the swallowing of 10 mL of water [5]. Given the quantity of fluid ingested (1 to 2 liters per day), several liters per day of air should enter the stomach by this mechanism. If not eructed, most swallowed air (the N_2 component) would pass through the gut with minimal absorption and hence be passed per rectum. Because less than 500 mL of N_2 per day is passed per rectum [6], it follows that most swallowed air is eructed, even though many patients deny knowledge of even a single belch a day. Air also can be swallowed in the absence of food or liquid ingestion via the propulsion of a bolus of air and perhaps a small amount of saliva into the hypopharnx. This mechanism is consciously employed by individuals who practice laryngeal speech and unconsciously by most those who complain of chronic eructation.

Patient management

There are no data that allow the physician to distinguish between normal and excessive eructation. However, the

patient complaining of this problem usually claims near constant belching, which clearly is excessive. Such patients are virtually always repeatedly swallowing boluses of air. Although the eructed air does not directly result from gastroesophageal pathology, for reasons that are not clear, gastric or esophageal discomfort may induce the patient to swallow air. Thus, appropriate diagnostic and therapeutic maneuvers are indicated if associated symptoms suggesting esophagitis or peptic ulcer are present. Treatment of such a problem may reduce the patient's desire to swallow air.

The vast majority of chronic eructors have no detectable organic problem, however, and their belching is simply a deeply ingrained air-swallowing habit. Thus, no diagnostic evaluation is indicated in the absence of symptoms of upper gastrointestinal disease. In these patients, treatment is largely educational. The air- swallowing origin of the problem should be clearly delineated, and the possibility that the gases are being produced in the gut should be thoroughly debunked. Unfortunately, most chronic air swallowers vigorously deny that they are gulping air, and even if this concept is accepted, control of the habit is difficult. Holding an object, such as a tongue blade, between the teeth is said to prevent air swallowing. However, in our experience, this intervention can be overcome by the determined air swallower [7•]. Education is most effective in the subset of patients who fear that their eructation is a sign of serious underlying disease. Reassurance of the benign basis of the eructation often results in a "cure" of the problem in these patients. Because air swallowing frequently appears to increase during periods of stress, therapy directed toward stress management and/or tranquilizers may be helpful [8].

Bloating and Distention

Pathophysiology

Exactly what is responsible for sensations of bloating and distention remains controversial. Patients have no doubt that excessive intestinal gas is the cause of the problem. This belief is based on several lines of evidence. Most convincing is the observation that passage of gas per rectum or movement of gas in the gut is seemingly associated with transient relief of the bloating sensation. In addition, protuberance of the abdomen, requiring loosening of belts or tight garments, frequently is associated with the sensation of bloating. Gas is presumed to be the source of the increased volume of abdominal contents responsible for the enhanced abdominal girth.

A large body of evidence suggests, however, that excessive intestinal gas may have little to do with bloating and distention. In our experience, roentgenograms of the abdomen of patients complaining of bloating seldom show excessive bowel gas. We obtained a more quantitative assessment of intestinal gas volume in experiments in which we "washed out" all intestinal gas through an argon infusion at the ligament of Trietz [2]. Quantitation of the native bowel gases washed out at the rectum showed that both healthy control subjects and patients with bloating and distention had similar volumes of bowel gas (mean of 100 mL, maximum of 200 mL). Maxton *et al*. [9] assessed the volume of bowel gas in 20 female patients complaining of bloating by measuring the area of all visible abdominal gas bubbles observed on radiographs of the abdomen. No significant increase in gas was observed during periods of bloating versus asymptomatic periods. Chami *et al*. [10] found that the area of abdominal gas bubbles was greater in constipated IBS patients than in control subjects, but no correlation was seen between the volume of gas observed and the complaints of bloating and distention. These data seem to indicate that excessive gas is not the cause of bloating. However, Serra *et al*. [11], in a very careful study, found that some patients have defective transport of gas through the gut. These authors postulate that such individuals could have an accumulation of gas, causing bloating and distention. Acceptance of this hypothesis will require some explanation for the apparent lack of excessive bowel gas in roentgenographic studies of bloating patients.

If no increase in abdominal gas is observed in patients who present with bloating, why do they so commonly report what appears to be a clear-cut increase in abdominal girth? Alvarez [12] made an extensive study of the protuberant abdomen associated with bloating and concluded that the protuberance had a large volitional component. When asked to reduce their abdominal girth, the majority of distended patients were able to respond. Several who could not voluntarily decrease their abdominal girth were anesthetized, and an immediate reduction in abdominal protrusion was observed. Alvarez concluded that the increase in abdominal girth was not caused by increased volume of bowel contents but rather represented an unconscious tendency of bloating patients to protrude their abdomen. Exactly how the bloating patient produces this abdominal protrusion is not clear. Conventionally, it was thought that distention resulted from lowering the diaphragm and assuming a lumbar lordotic posture. However, neither of these anatomic alterations was observed in computerized tomographic studies of bloating patients [9].

These observations illustrate that our understanding of the pathophysiology of bloating and distention is far from perfect. However, the preponderance of evidence seems to favor the concept that excessive gas probably has little to do with these symptoms. More likely, these symptoms are indicative of an "irritable" bowel that causes the patient to perceive that the intestine is over-distended when no such distention actually exists. This concept is supported by the observation that bloating patients have an enhanced pain response to balloon-induced bowel distention [13]. It is also possible that feelings of distention reflect increased volume of solid or liquid luminal contents rather than gas. For example, we found that healthy subjects complained of mild distention and "gas" following ingestion of a nonfermentable fiber [14]. These subjects had no increase of

flatus frequency or breath H_2 excretion, and a fiber-induced increase in luminal bulk appeared to be responsible for the sensation of distention.

Patient management

Although the vast majority of patients complaining of bloating and distention have no anatomic abnormality of the gut, partial bowel obstruction can cause similar symptomatology [15]. The diagnostic work-up for these complaints of bloating and distention is determined by the duration of the problem, the age of the patient, and other associated symptoms. The patient aged under 50 years with a multiple-year history and no other symptoms (*eg*, diarrhea or weight loss) is very unlikely to have an obstructing lesion, and a minimal work-up is indicated. In contrast, an elderly patient with a brief history of symptoms requires a more thorough evaluation.

Treatment of patients with complaints of bloating and distention is hampered by our poor understanding of the pathophysiology responsible for this problem. To the extent that intestinal gas volume usually is normal in these patients, it might seem irrational to employ treatments designed to reduce bowel gas. However, the possibility remains that normal volumes of gas may produce discomfort in these patients due to their irritable bowel. In these patients, the passage of swallowed air through the gut could cause discomfort even though their intestinal gas volume appears to be within normal limits. We have observed patients who have a vicious cycle of abdominal discomfort—air swallowing—discomfort—air swallowing. Education of these patients about the need to avoid swallowing air is indicated. Gas is also produced during the fermentation of non-absorbed carbohydrates in the colon (refer to following section, "Flatulence Problems") [16]. Although reduction of gas from this source also might be useful in patients with bloating, our studies suggest that colonic gas production does not play a major role in inducing symptoms of bloating and distention [14].

A number of pharmaceutical products have been employed for patients with bloating and distention. Some allegedly influence gas dynamics in the gut, whereas others are directed toward treatment of an underlying irritable bowel. Proof of efficacy of any manipulation of bloating patients requires rigorous blinded testing because these symptoms are notoriously susceptible to placebo effects. As an example, many bloating patients are certain that their abdominal symptoms are severely aggravated by the ingestion of lactose and that they are much improved with strict lactose avoidance. In a blinded study of a self-identified severely lactose-intolerant patient, we found that the ingestion of one cup of conventional milk per day (12.5 g of lactose) for a week resulted in no more abdominal symptomatology than was observed with lactose-hydrolyzed milk [17]. Prior to the study, most of these individuals believed that milk would result in such severe discomfort that they would have to withdraw from the study.

One compound that has been extensively advertised for use in the treatment of gaseous complaints is simethicone, a defoaming agent that reduces surface tension and thus would be expected to convert small bubbles into larger gas collections. Why this should be beneficial is not obvious; however, one could speculate that the large gas collection might be more effectively propelled through the gut and expelled per rectum. Such an effect has not been demonstrated, and to the contrary, there are claims that simethicone ingestion actually decreases the amount of gas passed per rectum. A few independent controlled trials of simethicone have been conducted; some showed efficacy and others found no benefit. On the basis of their double-blind trial, Jain *et al*. [18] conclude that simethicone was not an effective treatment for bloating. In contrast, Holtmann *et al*. [19•] studied 177 patients with functional dyspepsia (bloating) in a randomized, double-bind protocol. The patients received either simethicone or cisapride. The global perception of symptoms was significantly less with simethicone than with cisapride (cisapride was recently removed from the market except under special circumstances), but only during the first two weeks of treatment (no significant differences were observed at 4-week follow-up). Several controlled trials have suggested that motility enhancing agents may reduce symptoms of bloating, with Van Outryve *et al*. [20] showing benefit with cisapride and Johnson *et al*. [21] with metoclopramide. Given the apparent toxicity of both of these drugs, neither is recommended for the long-term treatment of bloating and distention.

Activated charcoal is another product that has been claimed to influence the volume of gut gas, presumably through the binding of gases by the charcoal. However, our in vitro studies indicate that charcoal adsorbs negligible amounts of the quantitatively important gut gases [22•]. Whereas two clinical studies showed that ingestion of activated charcoal with baked beans reduced breath hydrogen excretion (a measure of gut hydrogen) [18,23], we were unable to demonstrate that this compound reduced intestinal hydrogen production [24].

Although pancreatic supplements were developed for the treatment of pancreatic insufficiency, these preparations have been widely used for functional abdominal problems such as bloating and distention. Because ingestion of these preparations seemingly should add little to the enzyme output of the normal pancreas of these patients, no solid rationale exists for their use in patients with bloating problems. Nevertheless, we recently found in a blinded study that a microencapsulated pancreatic enzyme preparation significantly reduces the postprandial symptoms experienced by healthy volunteers after ingestion of a high-calorie, high-fat meal [25•]. This finding suggests that these supplements might also be beneficial in bloating patients.

Our opinion is that complaints of bloating and distention generally are a manifestation of an underlying

irritable bowel syndrome and have very little to do with bowel gas. Thus, treatment should be directed toward the irritable bowel, a topic that is beyond the scope of this review.

Flatulence Problems

Complaints of rectal gas can be related to excessive volume and/or odor. Because the quantitatively important gases have no odor and the odoriferous gases are present in only trace quantities, volume and odor should be considered as separate problems.

Excessive flatus volume

Pathophysiology

Healthy subjects pass from 400 to 2500 mL of gas per day [6], with a frequency of 10 passages per day (upper limit of normal is 22 times day) [14]. There are two potential origins of rectal gas: air swallowing and intraluminal production. These two sources can be distinguished through an analysis of flatus. N_2 is the predominant gas when air swallowing is the major source [7•], whereas H_2, CO_2, and CH_4 are predominant when most of the gas is derived from bacterial metabolism [16].

Although air swallowing is not commonly considered to be a major cause of flatulence, relatively enormous quantities of air can enter the gut through this mechanism (refer to "Excessive Eructation" section). If not eructed, the N_2 component will pass through the gut with minimal absorption and be excreted in flatus. Thus, excessive air swallowing and/or ineffective eructation theoretically leads to the appearance of excessive quantities of swallowed air in flatus. For example, we studied a patient who had a Nissen repair of a hiatus hernia, a procedure that prevents eructation, followed by a laryngectomy for laryngeal carcinoma several years later. This patient passed rectal gas at a rate of 5 liters per hour when he actively carried on esophageal speech.

The gas produced by intestinal bacteria is located primarily in the colon [2]. Gas production is determined by the amount of metabolites that reach these bacteria, primarily ingested carbohydrates that are incompletely absorbed in the small bowel. Patients with a diseased gut (*eg*, celiac sprue, pancreatic insufficiency, or short bowel) may have excessive gas caused by the malabsorption of carbohydrates that are well absorbed by healthy subjects. However, a variety of carbohydrates are malabsorbed by individuals who have no evidence of bowel disease. Table 1 summarizes the carbohydrates that may be malabsorbed by healthy subjects. Most ingested carbohydrates are polymers that require digestion to simple sugars before absorption can occur. An exception is fructose, which is ingested as a simple sugar. Fructose is present in low concentrations in some fruits and vegetables and in very high concentrations in most soft drinks (up to 23 g/12-ounce can) [26]. Fructose, which is absorbed by a facilitated (rather than an active) process may be incompletely absorbed by healthy subjects. Lactose is a disaccharide found only in milk and milk products. About 30% of US adults (and the majority of the adult population of the world) have a genetically programmed decrease in lactase synthesis which results in malabsorption of this disaccharide [27]. Trehalose, a disaccharide found in mushrooms and insects, is malabsorbed by individuals with a trehalase deficiency [28]. Legumes store carbohydrates via the linkage of one or two galactose molecules to sucrose to produce raffinose and stachyose, respectively. Humans lack the α- galactosidase enzyme required to split these galactososide linkages; consequently, these sugars are not absorbable [29]. The common ingested starches frequently are not completely absorbed by healthy subjects [30]. This malabsorption appears to reflect the resistance of these starches to amylase digestion in the small bowel. An example of a naturally resistant starch is that present in green bananas. The majority of this starch may be indigestible and thus pass to the colon, where it is readily fermented. Recently, interest has been shown in a process called retrogradation of starch, the crystallization that occurs with refrigeration of cooked products (such as pasta) [31]. This crystallization, which is not reversible with reheating, inhibits amylase digestion and produces malabsorption. The colonic bacteria have the ability to rapidly metabolize all forms of malabsorbed carbohydrate. Consequently, this material provides substrate for gas production.

Colonic bacteria may consume as well as produce gas. Thus, the volume of gas available for excretion is the net of production minus consumption. Such consumption is particularly important for H_2 in that, normally, over 90% of the total production of this gas is consumed by other bacteria [32]. There are individual differences in the efficiency of the H_2 consuming flora, and patients with ineffective consumption excrete more gas per unit of carbohydrate malabsorbed [32]. Thus, excessive rectal gas of bacterial origin may reflect increased carbohydrate malabsorption and/or ineffective gas consumption.

Patient management

When confronted with the patient complaining of excessive rectal gas, most physicians tend to reflexively order a series of tests that generally include expensive radiographic and/or endoscopic studies of the gut. We discourage the use of such studies because they virtually never uncover an anatomic lesion that would explain the increased gas.

The first step in evaluation of the patient complaining of excessive rectal gas is to determine whether the patient truly has excessive gas. Because quantitative collection of rectal gas is nearly impossible, it has been necessary to use the frequency of gas passage as an indirect indicator of volume. We have found that healthy adult subjects (on their usual diet) pass gas up to 22 times per day, and this frequency is not appreciably influenced by age or gender [14]. The patient

Table 1. Carbohydrates That May Be Incompletely Absorbed by Healthy Subjects

Carbohydrate	Dietary sources	Cause of malabsorption
Simple sugars		
Fructose	Fruit, honey, vegetables, soft drinks	Slow intestinal transport
Disaccharides		
Lactose	Milk, ice cream, yogurt, soft cheese	Low lactase activity
Trehalose	Mushrooms, insects	Low trehalase acitivity
Oligosaccharides		
Raffinose	Legumes and other vegetables	Absence of α-galactosidase activity
Stachose	Legumes and other vegetables	Absence of α-galactosidase activity
Complex carbohyrates		
Resistant starch	Fruits, flours, vegetables	Naturally resistant to amylase
Retrograde starch	Refrigerated wheat products	Crystallization–amylase resistance
Fiber	Whole grains, vegetables, fruits	Absence of β-glucosidase activity

Table 2. Symptoms and Signs Suggesting That Air Swallowing or Bacterial Fermentation Is the Cause of Excessive Rectal Gas

Symptom or sign	Air swallowing	Bacterial fermentation
Increased eructation	Yes	No
Increased salivation	Yes	No
Gas is stress related	Yes	No
Gas is meal related	No	Yes
Abdominal bloating	Yes	No
Highly malodorous gas	No	Yes
Nocturnal gas	No	Yes

should be asked to keep a very careful record of each flatus passage for several weeks [7•,17,33,34]. Although this may sound complicated, we have found that patients rather enjoy keeping such a record. Many patients will have a frequency of less than 22 times per day, and they simply have to be informed of their "normality." No further diagnostic or therapeutic manipulations are indicated.

If the patient passes gas more than 22 times per day, presumably excessive quantities of swallowed air or bacterial gases are passing through the gut. We have encountered patients with enormous flatus frequency caused by both of these mechanisms [7•,35]. Analysis of carefully collected flatus samples will differentiate between these two causes; however, such testing is not readily available. Symptoms and signs suggestive of air swallowing versus excessive bacterial fermentation are listed in Table 2.

If air swallowing is the cause of the excessive rectal gas, the patient should be informed of the benign nature of the problem and counseled on how air swallowing might be minimized. When the source of rectal gas is bacterial fermentation, treatment theoretically might be directed toward altering the colonic flora in the direction of less net gas liberation. Unfortunately, there is little evidence that the colonic flora can be manipulated in such a way as to allow less gas to be released. Antibiotics often produce greater inhibition of gas consumption than gas production; hence, net gas release increases. There are claims that supplementation of the diet with large quantities of lactose reduces gas production through induction of a colonic flora rich in lactobacilli, organisms that do not liberate H_2 during fermentation. However, a controlled trial of this approach in lactose malabsorbers suggests that the beneficial effect on gaseous symptoms was largely attributable to a placebo effect, because patients ingesting sucrose reported a decrease in gaseous symptoms comparable with that observed with lactose supplementation [36]. Little solid evidence supports the use of probiotics, such as lactobacilli, to reduce flatus excretion.

Dietary alterations that decrease the quantity of carbohydrate reaching the colon reduce the quantity of gas produced in the colon and diminish flatus excretion. For example, Tomlin *et al*. [6] found that flatus excretion fell markedly when poorly absorbed carbohydrates were removed from the diet of healthy subjects. A low-gas diet would not contain the various carbohydrates listed in Table 2. Unfortunately, such a diet is extremely restrictive (particularly with regard to starches), and in our experience most patients would rather pass gas than adhere to such a diet.

A small subset of flatulent patients may benefit from reduction of lactose intake. We have carried out extensive testing of the flatulence that results from ingestion of milk by patients who have low intestinal lactase [17,33,34]. Despite malabsorption of lactose, flatulence was minimal when these patients ingested one cup of milk (12 g of lactose) with breakfast and dinner [33]. However, if sufficient milk is ingested over a short period, flatulence results. For example, when two 15-g doses of lactose in a dietary supplement were ingested at breakfast and lunch without other food, very appreciable flatulence (30 passages per 12 hours) resulted (Submitted data). In this situation, it appears that lactose was delivered to the colon very rapidly, and the rate of gas production far outstripped the consumption and absorption mechanisms. As a result, a very large quantity of gas was available for rectal excretion. Flatulence problems from lactose can be treated by simply reducing the quantity of milk ingested. Alternatively, patients who elect to consume large quantities

of milk can purchase low-lactose milk, produce their own low-lactose milk through treatment of their milk with lactase, or take lactase tablets at the time of milk ingestion.

Noxious Flatus

Physiology

Our evaluation of human flatus indicates that three sulfur-containing gases produced by fecal bacteria—H_2S, methanethiol, and dimethylsulfide—are the major malodorous components of human flatus [3]. The total of these three sulfur-containing gases represents less than one part in 10,000 of flatus; however, even these low concentrations would be very offensive to the human nose. Moore *et al*. [37] conclude that sulfur-containing gases are the main malodorants in human feces, and find no evidence to support the commonly held belief that indole and skatole are responsible for the unpleasant odor of feces. The rate of production of sulfur gases by the colonic bacteria presumably is a function of the availability of sulfur-containing substrates. These substrates include sulfate and cysteine, which are of dietary origin, or taurocholate and mucin, which are endogenous to the gut [38].

The sulfur gases are very rapidly absorbed and/or metabolized by the colonic mucosa; thus, the quantity of these gases excreted per rectum is only a trivial fraction of the volume produced in the colon [39]. As a result, the residence time of these gases in the colon is a major determinant of the quantity passed per rectum; *ie*, a greater fraction of total production is excreted per rectum when production occurs in the distal colon or rectum versus the more proximal colon.

Patient management

The first problem facing the physician when a patient complains of excessively noxious flatus is to determine whether there truly is an "abnormality." Some patients claim to have become social outcasts, and some believe that they are continually exuding an odor, in the absence of gas passage. Study of several patients with these complaints failed to reveal a persistent odor. Forceful explanations to the effect that no odor exists were unaccepted, and it was clear that the patient was delusional in this regard. Although psychotherapy might be helpful for these patients, there is no role for the gastroenterologist.

A second subset of patients state that their flatus expulsion arouses more than the usual level of complaint by their close associates. Objective studies showing that such patients actually excrete excessively noxious flatus have not been performed. However, it seems likely that some individuals excrete far more sulfur gas than do others, and treatment to reduce sulfur gas release in these patients would be desirable. The little that is known about the influence of various manipulations on the production of odoriferous fecal gases is summarized below. Antibiotic administration often is associated with the more malodorous flatus, suggesting that such therapy enhances, rather than reduces, sulfur gas production in the colon. Administration of probiotics (lactobacilli plus fructooligosaccharide) had no benefit on sulfur gas production by feces (Personal observation). Most healthy individuals who complain of noxious flatus are at least minimally constipated, and a bowel movement seems to temporarily reduce the passage of malodorous gas. Thus, treatment designed to induce a normal or even supra-normal frequency of bowel movements may be useful. Surprisingly, some patients claim that non-absorbed carbohydrate laxatives such as lactulose, which increase total gas production, may actually reduce odor. A possible explanation is that the acid environment induced by fermentation of the carbohydrate inhibits sulfur gas production. Presumably, a diet that is low in non-absorbable sulfur-containing compounds (primarily sulfate) would be helpful, but there are no studies to document this concept. Foods rich in sulfur-containing compounds include cruciferous vegetables (such as broccoli) and beer. However, if endogenous substrates such as mucin are the primary source of sulfur for the colonic bacteria, dietary manipulations are unlikely to be beneficial.

A charcoal-lined cushion (Ultratech, Houston, TX) has been developed to combat excessively noxious flatus. According to the manufacturers, if gas is passed while the individual is sitting on this cushion, sulfur gases will be adsorbed by the charcoal. We assessed the effectiveness of this cushion using specially designed gas-tight mylar pantaloons. Gas containing the three sulfur gases found in flatus was infused at the anus through a narrow-caliber tube. When subjects sat on the cushion, less than 10% of the infused sulfur gas reached the gas space of the pantaloons, whereas, in the absence of the cushion, almost 100% of the infused sulfur gas was recovered in the pantaloons [3].

Lastly, the ability of several orally administered compounds to absorb the sulfur gases has been tested. Activated charcoal effectively binds sulfur gases in vitro, but we found that fecal liberation of sulfur gases was not reduced when subjects ingested two 260-mg charcoal tablets four times a day [22•]. Bismuth also avidly binds sulfides in vitro. In contrast with the results with charcoal, the feces of patients ingesting two tablets of bismuth subsalicylate four times a day released virtually no H_2S and greatly reduced volumes of CH_3SH [40•]. Consequently, this treatment should provide effective therapy for fecal or flatus odor. However, one must keep in mind that long-term therapy with bismuth subsalicylate on very rare occasions has been associated with absorption of sufficient bismuth to induce bismuthism [41,42]. Serum bismuth levels should be monitored with long-term use of bismuth subsalicylate.

Conclusions

The gastroenterologist treating patients who present with complaints of excessive intestinal gas is faced with many challenges. Recent research on the pathophysiology and management provides a helpful context for the practitioner dealing with such patients and suggests ways to

respond to their questions and concerns. Further examination should provide promising results.

Acknowledgements

This work was supported in part by the Department of Veterans Affairs and The National Institute of Diabetes and Digestive and Kidney Diseases grant R01 DK 13309-25.

References and Recommended Reading

Papers of particular interest, published recently, have been highlighted as:

- • Of importance
- •• Of major importance

1. Kirk E: **The quantity and composition of human colonic flatus.** *Gastroenterology* 1949,**12:**782–794.
2. Levitt MD: **Volume and composition of human intestinal gas determined by means of an intestinal washout technique.** *N Engl J Med* 1971, **284:**1394–1398.
3. Suarez FL, Furne JK, Springfield JR, Levitt MD: **Identification of gases responsible for the odor of human flatus and evaluation of a device purported to reduce this odor.** *Gut* 1998, **43:**100–104.
4. Maddock WG, Bell JL, Tremaine MJ: **Gastrointestinal gas: observation of belching during anesthesia, operations, pyelography and rapid passage of gas.** *Ann Surg* 1949, **130:**512.
5. Pouderoux P, Gulchin AE, Shezhang L, Kahrilas PJ: **Esophageal bolus transit imaged by ultrafast computerized tomography.** *Gastroenterology* 1996,**110:**1422–1428.
6. Tomlin J, Lowis C, Read NW: **Investigation of normal flatus production in healthy volunteers.** *Gut* 1991, **32:**665–669.
7. • Levitt MD, Furne JK, Aeolus MR, Suarez FL: **Evaluation of an extremely flatulent patient: case report and proposed diagnostic and therapeutic approach.** *Am J Gastroenterol* 1998, **93:**2276–2281.

A recent case report that may be of interest.

8. Baume P, Tracey M, Dawson L: **Efficacy of two minor tranquilizers in relieving symptoms of functional gastrointestinal distress.** *Aust NZ J Med* 1975, **5:**503–506.
9. Maxton DG, Martin DF, Whorwell PJ, Godfrey M: **Abdominal distention in female patients with irritable bowel syndrome: exploration of possible mechanisms.** *Gut* 1991, **32:**662–664.
10. Chami TN, Schuster MM, Bohlman ME, *et al.*: **A simple radiologic method to estimate the quantity of bowel gas.** *Am J Gastroenterol* 1991, **86:**599–602.
11. Serra J, Azpiroz F, Malagelada J-R: **Intestinal gas dynamics and tolerance in humans.** *Gastroenterology* 1998, **115:**542–550.
12. Alvarez WC: **Hysterical type of nongaseous abdominal bloating.** *Arch Intern Med* 1949, **84:**217–245.
13. Ritchie J: **Pain from distention of pelvic colon by inflating a balloon in the irritable bowel syndrome.** *Gut* 1973, **14:**125–132.
14. Levitt MD, Furne J, Olsson S: **The relation of passage of gas and abdominal bloating to colonic gas production.** *Ann Intern Med* 1996, **124:**422–424.
15. Schuffler MD, Sinanan MN: **Intestinal obstruction and pseudo-obstruction.** In *Gastrointestinal and Liver Diseases,* edn 6. Edited by Sleisenger MH, Fordtran JS. Philadelphia: WB Saunders; 1998:898–916.
16. Strocchi A, Levitt MD: **Intestinal gas.** In *Gastrointestinal and Liver Diseases,* edn 6. Edited by Sleisenger MH, Fordtran JS. Philadelphia: WB Saunders; 1998:153–160.
17. Suarez FL, Savaiano DA, Levitt MD: **A comparison of symptoms in people with self-reported severe lactose intolerance after drinking milk or lactose-hydrolyzed milk.** *N Engl J Med* 1995, **333:**1–4.
18. Jain NK, Patel VP, Pitchumoni S: **Activated charcoal, simethicone, and intestinal gas: a double-blind study.** *Ann Intern Med* 1986, **105:**61–62.
19. • Holtmann G, Gschossmann J, Karaus M, *et al.*: **Randomised double-blind comparison of simethicone with cisapride in functional dyspepsia.** *Aliment Pharmacol Ther* 1999, **13:**1459–1465.

Results from a notable recent study.

20. Van Outryve M, Milo R, Toussaint J, *et al.*: **'Prokinetic' treatment of constipation predominant irritable bowel syndrome: a palcebo-controlled study of cisapride.** *J Clin Gastroenterol* 1991, **13:**49–57.
21. Johnson AG: **Controlled trial of metoclopramide in the treatment of flatulent dyspepsia.** *Br Med J* 1971, **2:**12–16.
22. • Suarez FL, Furne JK, Springfield J, Levitt MD: **Failure of activated charcoal to reduce the release of gases produced by the colonic flora.** *Am J Gastroenterol* 1999, **94:**208–212.

Results from a study assessing the effectiveness of activated charcoal.

23. Hall RG, Thompson H, Strother A: **Effects of orally administered activated charcoal on intestinal gas.** *Am J Gastroenterol* 1981, **75:**192–196.
24. Potter T, Ellis C, Levitt M: **Activated charcoal: in vivo and in vitro studies of effect on gas formation.** *Gastroenterology* 1985, **88:**620–624.
25. • Suarez FL, Levitt MD, Adshead J, Barkin JS: **Pancreatic supplements reduce the symptomatic response of healthy subjects to a high fat meal.** *Dig Dis Sci* 1999, **44:**1317–1321.

Recent study with important clinical implications.

26. Bowel A, Church H: *Food Values of Portions Commonly Used,* edn 16. Philadelphia: JB Lippincott; 1994.
27. Scrimshaw NS, Murray EB: **The acceptability of milk and milk products in populations with a high prevalence of lactose intolerance.** *Am J Clin Nutr* 1988, **48:**1083–1159.
28. Bergoz R: **Threhalase malabsorption causing intolerance to mushrooms.** *Gastroenterology* 1971, **60:**909–912.
29. Suarez FL, Springfield J, Furne JK, *et al.*: **Gas production in humans ingesting a soybean flour derived from beans naturally low in oligosaccharides.** *Am J Clin Nutr* 1999, **69:**135–139.
30. Anderson IH, Levine AS, Levitt MD: **Incomplete absorption of the carbohydrate in all-purpose wheat flour.** *N Engl J Med* 1981, **304:**891–892.
31. Englyst HN, Wiggins HS, Cummings JH: **Determination of the non-starch polysaccharides in plant foods by gas-liquid chromatography of constituent sugars as alditol acetates.** *Analyst* 1982, **107:**307–318.
32. Strocchi A, Levitt MD: **Factors affecting production and consumption by human fecal flora: the critical role of hydrogen tension and methanogenesis**. *J Clin Invest* 1992, **89:**1304–1311.
33. Suarez FL, Savaiano DA, Arbisi P, Levitt MD: **Tolerance to the daily ingestion of two cups of milk by individuals claiming lactose intolerance.** *Am J Clin Nutr* 1997, **65:**1502-1506.
34. Suarez FL, Adshead J, Furne JK, Levitt MD: **Lactose maldigestion does not represent an impediment to the intake of 1500 mg of calcium/day as dairy products.** *Am J Clin Nutr* 1998, **68:**1118–1122.
35. Sutalf LO, Levitt MD: **Follow up of a flatulent patient.** *Dig Dis Sci* 1979, **24:**652–654.
36. Briet F, Pochart P, Marteau P, *et al.*: **Improved clinical tolerance to chronic lactose ingestion in subjects with lactose intolerance: a placebo effect?** *Gut* 1997, **41:**632–635.
37. Moore JG, Jessop LD, Osborne DN: **Gas-chromatographic and mass-spectrometric analysis of the odor of human feces.** *Gastroenterology* 1987, **93:**1321–1329.
38. Levine J, Furne JK, Levitt MD: **Fecal hydrogen sulfide production in ulcerative colitis.** *Am J Gastroenterol* 1998, **93:**83–87.
39. Suarez FL, Furne JK, Springfield JR, Levitt MD: **Insights into human colonic physiology obtained from study of flatus composition.** *Am J Physiol* 1997, **272:**G1028–G1033.
40. • Suarez FL, Furne JK, Springfield J, Levitt MD**: Bismuth subsalicylate markedly decreases hydrogen sulfide release in the human colon.** *Gastroenterology* 1998, **114:**923–929.

Of clinical interest.

41. Hasking GJ, Duggin JM: **Encephalopathy from bismuth subsalicylate.** *Med J Aust* 1982, **2:**167.
42. Gordon MF, Abrams RI, Rubin DB, *et al.*: **Bismuth subsalicylate toxicity as a cause of prolonged encephalopathy with myoclonus.** *Mov Disord* 1995, **10:**220–222.