CLINICAL—ALIMENTARY TRACT

Abdominal Distention Results From Caudo-ventral Redistribution of Contents

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See editorial on page 1487.

Background & Aims: Abdominal bloating is a frequent symptom in various categories of patients; however, its origin is unclear. Our aim was to establish the mechanisms of abdominal bloating. Methods: The study evaluated 56 patients whose predominant symptom was abdominal bloating. Of these, 47 (44 female and 3 male; aged 19–74 years) were diagnosed with functional intestinal disorder by Rome II criteria and 9 (7 female and 2 male; aged 18–64 years) were diagnosed with intestinal dysmotility by gastrointestinal manometry. Computed tomographic scans were obtained before (basal level) and during a severe bloating episode. Control scans were also obtained from 12 healthy subjects (11 female and 1 male; aged 19–62 years). Morphovolumetric differences between basal and severe bloating scans were measured using an original computer analysis program. Results: During severe bloating, patients with dysmotility exhibited anterior wall protrusion (23 ± 4 mm; $P < .001$ vs basal) associated with a marked increase in total abdominal volume (1.4 ± 0.3 L; $P = .002$ vs basal) and with cephalic displacement of the diaphragm. By contrast, in patients with functional intestinal disorder, total abdominal volume barely increased (0.3 ± 0.1 L; $P < .001$ vs dysmotility); in these patients, abdominal distention (14 ± 2 mm anterior wall protrusion; $P < .001$ vs basal) was related to diaphragmatic descent (−12 ± 3 mm; $R = -0.62$; $P < .001$). Conclusions: Abdominal distention might be caused by an increase in intra-abdominal volume or abdomino-phrenic displacement and ventro-caudal redistribution of contents.

Abdominal bloating is a frequent and troublesome symptom, and its origin and mechanism are poorly understood.[1,2] We have shown that patients with functional gut disorders[3] who report bloating have impaired intestinal gas transit and, in contrast to healthy subjects, retain exogenous gas infused into the gut.[4–9] However, using a highly sensitive, original method of abdominal computed tomography (CT) image analysis, we failed to detect differences in gas content between patients with functional bloating and healthy subjects.[10] We then realized that patients had been studied on a prefixed schedule, whereas real-life bloating runs a characteristic fluctuating course and develops during discrete episodes, usually after meals and at the end of the day.[1,11] Based on these data, we hypothesized that bloating episodes were due to transient accumulation of intestinal gas, and a study was designed to examine the relationship between bloating sensation and changes in abdominal walls and content. Our specific aims were to determine (1) whether the subjective bloating sensation is really associated with objective abdominal distention, (2) whether distention is due to volume increments in intestinal gas and/or intra-abdominal contents, and (3) to correlate changes in anterior abdominal wall and diaphragmatic position.

Abdominal morpho-volumetric changes associated with severe bloating, compared with basal conditions, were characterized in a large group of patients with functional bloating and in a thoroughly selected group of patients with "organic" bloating.

Subjects and Methods

Participants

Twelve healthy individuals without gastrointestinal symptoms (11 women and 1 man; age range, 19–62 years) and 56 patients whose predominant abdominal symptom was abdominal bloating (ie, subjective sensation of abdominal distention) participated in the study. Only patients in whom the bloating sensation exhibited fluctuations (ie, severe bloating episodes in contrast to mild or no sensation during basal conditions) were included in the study. Forty-seven patients (44 women and 3 men; age range, 19–74 years) were diagnosed based on Rome II criteria as having functional disorders,[11] 17 as functional bloating, and 30 as irritable bowel syndrome: 10 with alternating bowel habits and 20 constipation predominant (2.2 ± 0.7 bowel move-

Abbreviations used in this paper: **CT, computed tomography; HU, Hounsfield units.**

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ments per week, 1.4 ± 0.2 score in the Bristol stool form scale). No patient qualified as having diarrhea-predominant irritable bowel syndrome. Symptom duration was similar in the 3 groups (12 ± 2 years in functional bloating, 15 ± 2 years in alternating bowel habits, and 13 ± 4 years in constipation-predominant irritable bowel syndrome). Nine additional patients (7 women and 2 men; age range, 18–64 years) were diagnosed with intestinal dysmotility (7 neuropathic type and 2 myopathic type) using standard manometric criteria.[12] The study protocol had been approved by the Institutional Review Board of the University Hospital Vall d'Hebron, and all subjects gave their written informed consent to participate in the study.

CT Scanning

Abdominal CT scans were obtained with a helical multislice CT scanner (Mx8000; Philips Medical Systems, Best, The Netherlands) with the following specifications: exposure 120 kV and 50 mA/s, 2.5-mm section thickness reconstruction at 1.6-mm intervals, 1.5 pitch ratio, and 512 × 512 acquisition matrix. All participants were scanned in the supine position. Scans were performed blindly (see Experimental Design in the following text) following a standard procedure that includes instructing the participants to hold their breath. No oral or intravenous contrast material was administered. With these characteristics, the total effective dose was 2.4 mSv, similar to the dose of a CT colography and approximately one fourth of the dose of a standard CT scan.

Bloating Sensation

At the time of each scan, bloating, defined as the subjective sensation of abdominal distention, was measured on a graphic rating scale graded from 0 (no perception) to 6 (extremely bothersome sensation).

Experimental Design

All patients underwent 2 CT scans. They were instructed to come to the hospital on 2 occasions: when they felt their bloating sensation was low (basal conditions) and during an episode of severe bloating. Patients were instructed to come immediately to the hospital when the bloating sensation reached a score of 4 on the 0–6 scale (see previous text). Waiting time for scanning was less than 30 minutes from arrival. Hence, the CT scan was performed within 60 minutes of bloating becoming severe. Healthy subjects underwent a CT scan during basal conditions as control. CT scanning and analysis were performed blindly, without knowing the category (patient or healthy subject) and condition (basal or bloating) of the participant.

Data Analysis

Abdominal morpho-volumetric analysis of CT images was performed using an original software program specifically developed in our laboratory.

To measure the volume and distribution of gas within the gut, a set of software modules, executed by a series of steps, was developed. The first step consists of converting the initial CT scan from the DICOM (Digital Imaging and Communications in Medicine) format provided by the manufacturer into a simple format (MetaImage) to account for the variability in DICOM files provided by CT manufacturers. The next step cleans the data by removing unwanted regions, such as the lungs and extracorporeal structures (air, table) present in the input data. Images are then filtered with a user-defined threshold to separate gas from tissues. Measurements of X-ray attenuation of a tissue are referenced to the relative attenuation of water and air using Hounsfield units (HU). In this scale, the attenuation produced by pure air is –1000 HU and the attenuation produced by water is 0 HU. Hence, 1 HU is 0.1% of the attenuation of water with respect to air. The attenuation of tissues depends on their composition.[13] The final step computes the volume of gas considering the proportion of gas in each pixel: pixels of –1000 HU are considered to contain 100% gas, pixels above a preselected threshold (T_{gas}) are considered to have no gas, and pixels between –1000 HU and the selected threshold (T_{gas}) are considered to have a part of gas, whose proportion depends on the attenuation, following a linear relation from –1000 HU (corresponding to 100% gas) to 0 HU (0% gas). The volume of gas in each pixel is accumulated using the formula $V_{gas} = \Sigma_i w(H_i, T_{gas}) V_{pixel}$, where V_{gas} is the accumulated gas volume, $w(H_i, T_{gas})$ represents the gas proportion in pixel i ($w = 0$ when $H_i >$ Tgas and $w = -H_i/1000$ otherwise), and V_{pixel} is the volume of each pixel. Previous validation studies showed that $T_{gas} = -500$ HU provides the best accuracy in detecting gas volumes infused into the gut with an error within the range of ±40 mL.[14] The program permits us to measure both the total gas volume within the abdominal cavity and segmental volumes in selected regions of the gut.

Total abdominal volume (gas, liquids, solids) was measured by a separate software program. The abdomen was bounded by bony structure planes: (1) a cranial plane perpendicular to the vertebral spine above the diaphragmatic domes and (2) a caudal plane defined by bony structures in the pelvis. Abdominal volume was measured as the body volume between the 2 planes, subtracting the volume of the lungs (pulmonary air below the cranial plane) and the heart.

Another software utility was also developed to measure girth by averaging the perimeter of the abdominal surface measured in 10 axial slices 4 mm apart, starting tangentially to the iliac crest in the cranial direction. At each site, girth was measured as the length of a polyline (series of connected segments) following the body contour. Anteroposterior abdominal diameter was measured as the distance (in the axial plane) between the anterior aspect of the vertebral bodies and the midline surface of the anterior abdominal wall. The average of the values measured at 6 levels (L_1 to S_1) was calculated in each subject. Position of the diaphragm was measured as the distance (in the vertical axis) between the left diaphragmatic dome and the line connecting the iliac crests. Lumbar lordosis was measured in the sagittal plane as the angle between lines perpendicular to the cranial end plate of the first and the caudal end plate of the fifth lumbar vertebra.[15] Thoracic perimeter was measured by averaging the perimeter of the chest measured in 10 axial slices 4 mm apart starting in T_{11} in the caudal direction. Anteroposterior diameter of the thorax was measured as the distance between the anterior aspect of T_{11} and the midline surface of the chest.

The X-ray attenuation spectrum of abdominal contents was analyzed by measuring in each scan the abdominal volume in the interval between –200 HU and –30 HU, where fat is represented.[14]

The software developed in this work was built on 2 open-source toolkits: the Insight Segmentation and Registration toolkit[16] and the Visualization toolkit, both used for 3-dimensional computer graphics, image processing, and visualization.[17]

Statistical Analysis

Mean values (±SE) of the parameters measured were calculated in each group of subjects. Within each group, normality was tested by the Kolmogorov–Smirnov test. Comparisons of parametric, normally distributed data were made by the Student *t* test, paired tests for intragroup comparisons, and unpaired tests for intergroup comparisons; otherwise, the Wilcoxon signed rank test was used for unpaired data between groups and the Mann–Whitney *U* test for unpaired data between groups. Correlations of paired data were examined using linear regression analysis.

Results

Morpho-volumetric Characterization of Participants

No significant differences were observed between healthy subjects, patients with functional bloating, and patients with intestinal dysmotility (Table 1). Both abdominal

Table 1. Abdominal Morpho-volumetric Features

	Healthy subjects	Patients	
		Functional	Dysmotility
Total abdominal volume (*L*)	12.5 ± 1.2	14.4 ± 0.7	14.0 ± 1.7
Fat volume (*L*)	4.9 ± 1.0	6.7 ± 0.5	5.6 ± 1.4
Perimeter (girth) (*cm*)	80 ± 4	89 ± 2	84 ± 6
Anteroposterior diameter (*cm*)	9.7 ± 0.7	11.2 ± 0.4	10.9 ± 1.1
Diaphragmatic position (*cm*)[a]	193 ± 7	192 ± 4	207 ± 9
Lumbar lordosis	39 ± 2	40 ± 2	32 ± 2
Thorax perimeter (*cm*)	83 ± 3	87 ± 1	85 ± 4

NOTE. No statistical differences between groups were observed.
[a]Distance between left diaphragmatic dome and line connecting iliac crests.

Table 2. Intestinal Gas Content

	Gas content (*mL*)	
	Stomach and small bowel	Colon
Healthy subjects	32 ± 7	99 ± 17
Functional disorders		
Basal	35 ± 4	79 ± 10
Distention	34 ± 3	111 ± 12
P vs basal	.824	.008
Intestinal dysmotility		
Basal	282 ± 214[a]	267 ± 114[a]
Distention	673 ± 370[a]	529 ± 225[a]
P vs basal	.145	.08

[a]$P \leq .006$ vs functional disorders.

girth and the volume of abdominal contents in the X-ray attenuation spectrum of fat (−200 HU to −30 HU) tended to be larger in functional patients than in healthy subjects, but the differences did not reach statistical significance ($P = .074$ and $P = .112$, respectively). Patients with intestinal dysmotility were in between (Table 1). Pooling all subjects together, girth was strongly related to the volume of abdominal contents in the fat spectrum ($R = 0.92$; $P < .001$) and total abdominal volume ($R = 0.86$; $P < .001$).

Patients With Functional Bloating

Basal conditions. During basal conditions, the volume and distribution of intestinal gas in functional patients were similar to those in healthy subjects. Gas volume in the different gut compartments was similar in both groups (Table 2). At the time of the basal scan, the subjective bloating sensation was relatively mild (Figure 1).

Bloating episode. At the time of the scan obtained during a severe bloating episode, the subjective bloating sensation was significantly higher than at the time of the basal scan (Figure 1). Bloating sensation was associated with objective abdominal distention, measured as a significant increase in girth (21 ± 4 mm; $P < .001$) and in the anteroposterior (spine to anterior wall) diameter (Figures 2 and 3). During severe bloating, lumbar lordosis remained unchanged (40° ± 2°). Severe bloating was associated with significant increases in abdominal gas, particularly in the colon ($P = .008$), in all colonic segments alike. Total abdominal volume was also significantly greater during bloating ($P = .002$; Figure 2). However, the increases in both gas and total volume observed were relatively small and did not correlate with the degree of abdominal distention; the correlation of abdominal protrusion (increase in anteroposterior diameter) to gas increment was $R = 0.17$ ($P = .26$) and to total volume increment was $R = 0.18$ ($P = .29$). Interestingly, severe bloating was associated with a marked and significant diaphragmatic descent ($P < .001$ vs basal) (Figures 2 and 3), which correlated with the degree of anterior wall protrusion ($R = -0.62$; $P < .001$) (Figure 4). Both the anteroposterior diameter and thorax perimeter measured at T_{11} level significantly increased (by 5 ± 1 mm and 12 ± 2

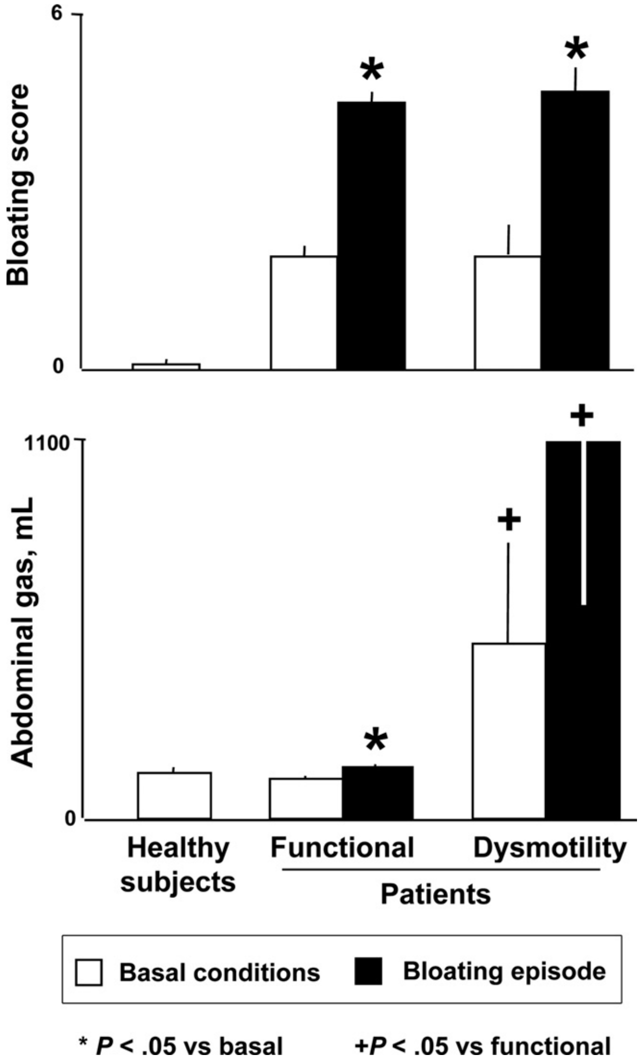

Figure 1. During basal conditions, bloating sensation was low in all groups, but patients with intestinal dysmotility had significantly more gas in the gut. During bloating, intestinal gas increased markedly (although not significantly; $P = .075$) in dysmotility patients but only marginally (although significantly; $P = .013$) in patients with functional disorders. Data are expressed as mean ± SE.

mm, respectively; $P < .001$ for both). None of the previously described parameters differed between the subgroups of patients with alternating bowel habits, constipation-predominant irritable bowel syndrome, and functional bloating (data not shown). Neither age nor sex influenced the results.

Bloating sensation developed in 19 patients within 240 minutes after ingestion of a meal (127 ± 18 minutes between meal ingestion and scanning) and in the remainder bloating was not related to meals (309 ± 13 minutes between meal ingestion and scanning); most of the latter (23 of 28) corresponded to the typical clinical pattern where bloating increases through the day, peaks in the late afternoon/evening, and subsides later. None of the characteristics of bloating (bloating score, abdominal gas, girth increment, increase in anteroposterior diameter, and diaphragmatic descent) differed in these 2 subgroups of patients, and the proportion of constipation, alternating bowel habits, and functional bloating was similar. Conversely, the proportion of postprandial versus late-day bloating was similar regardless of bowel habit. Similar results were observed using cutoffs different from 240 minutes.

Basal girth, abdominal fat volume, and lumbar lordosis

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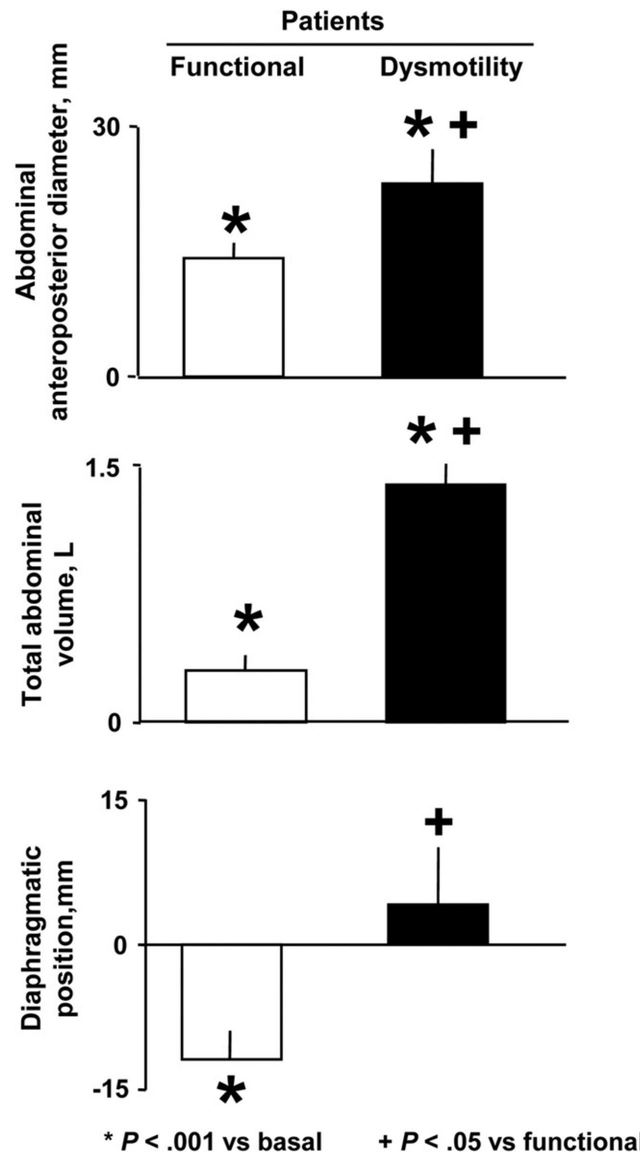

Figure 2. Changes associated with bloating (differences vs basal). Bloating was associated with significant anterior wall protrusion both in functional and dysmotility patients. In dysmotility patients, distention was associated with marked intra-abdominal volume increment ($P = .002$ vs basal) and cephalic displacement of the diaphragm ($P = .279$ vs basal). By contrast, in patients with functional disorders, abdominal volume barely increased ($P = .002$ vs basal; $P < .001$ vs dysmotility) and anterior wall protrusion was associated with significant diaphragmatic descent ($P < .05$ vs basal and vs dysmotility). Data are expressed as mean ± SE.

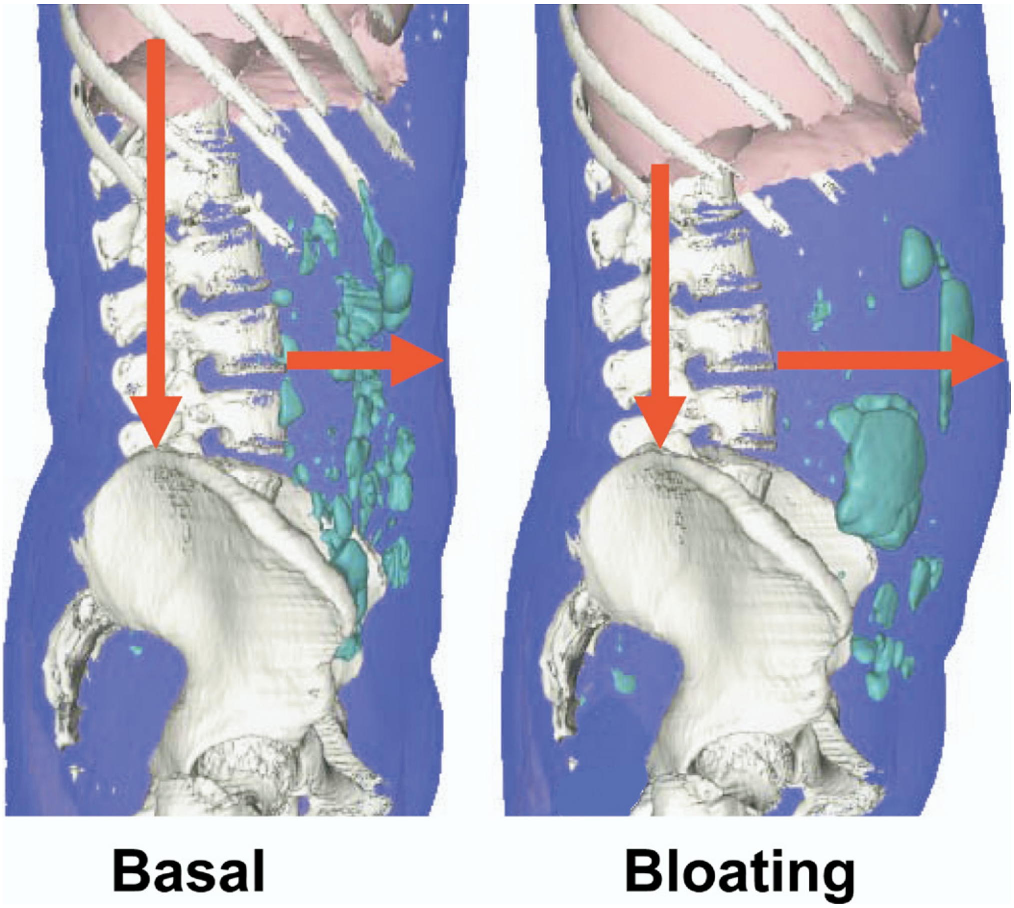

Figure 3. Abdominal imaging in a patient with functional gut disorder. Note anterior abdominal wall protrusion and diaphragmatic descent during bloating compared with basal with only a small increase (by 22 mL) in gas content.

did not correlate with girth increment ($R = -0.072$, $R = -0.129$, and $R = -0.148$, respectively; $P > .3$ for all) or abdominal protrusion during bloating ($R = -0.128$, $R = -0.274$, and $R = 0.026$, respectively; $P > .1$ for all).

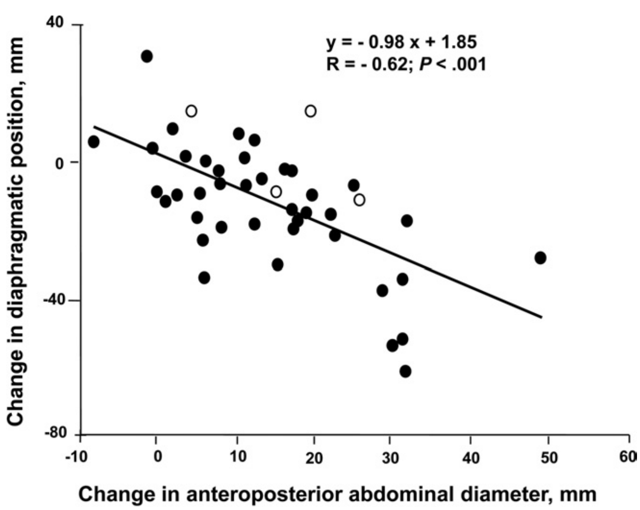

Figure 4. Bloating in patients with functional gut disorders (individual differences vs basal). Anterior wall protrusion correlated with diaphragmatic descent. Note responses in the 4 subjects (*open circles*) with more than 150 mL (168–325 mL range) gas increment during bloating; all developed distention, but the diaphragmatic response was variable.

Patients With Intestinal Dysmotility

Basal conditions. At the time of the basal scan, patients reported relatively mild bloating sensation, similar to that in functional patients during basal conditions (Figure 1). However, the volume of intestinal gas in dysmotility patients was significantly greater in all segments of the gut than in functional patients and in healthy subjects (Table 2).

Bloating episode. At the time of the scan obtained during a severe bloating episode, the severity of the sensation was markedly and significantly higher than during basal conditions; however, it was not different from that observed in functional patients during distention (Figure 1). Bloating sensation in patients with dysmotility was also associated with objective abdominal distention, determined by an increase in girth (42 ± 6 mm; $P < .001$ vs basal and $P = .026$ vs functional) and anterior wall protrusion while lumbar lordosis remained unchanged (32° ± 2°) (Figures 2 and 5). In contrast to functional patients, severe bloating in dysmotility patients was associated with a marked increment in total abdominal volume ($P = .002$ vs basal) (Figures 1 and 2). Total abdominal volume increase correlated with anteroposterior diameter increment ($R = 0.79$; $P = .01$). Of note, almost half the total volume increase corresponded to gas, although the change in gas content did not reach

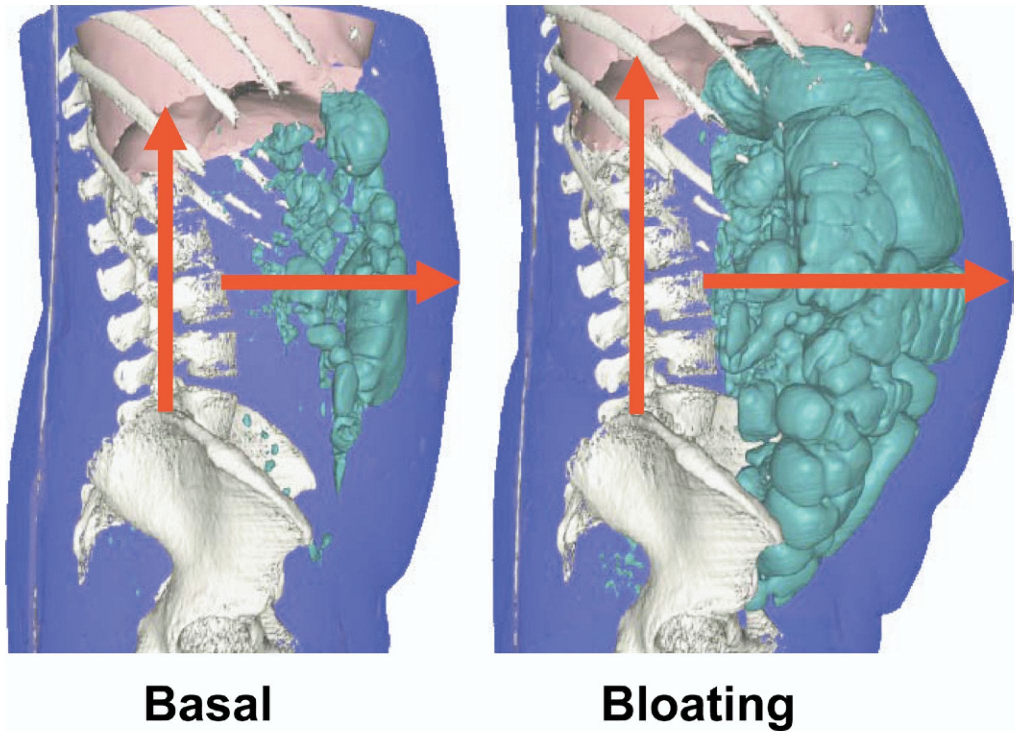

Figure 5. Abdominal imaging in a patient with intestinal dysmotility. Note the marked increment in gas content (by 3352 mL) associated with anterior abdominal wall protrusion and diaphragmatic ascent.

statistical significance (P = .075 vs basal). The increases in total abdominal volume and gas content were both significantly greater than in functional patients (P = .0001 and P = .001, respectively). Gas content increased overall in the gut (Table 2). In contrast to functional patients, the diaphragm did not descend (P = .042 vs functional bloating) (Figures 2 and 5). The change versus basal condition did not reach statistical significance (P = .279), but the degree of diaphragmatic displacement was related to the increase in total abdominal volume (R = 0.79; P = .01), and higher volumes were associated with cephalic displacement. As in functional patients, both the anteroposterior diameter and perimeter of the thorax significantly increased (by 12 ± 3 mm and 24 ± 4 mm, respectively; $P < .001$ for both). In dysmotility patients, severe bloating developed 202 ± 23 minutes after ingestion of a meal (5 patients within and 4 patients after the 240-minute postingestion period). Lumbar lordosis did not correlate with changes in anteroposterior abdominal diameter (R = 0.015; P = .970) or diaphragmatic position ($R = -0.311$; P = .415).

Discussion

Our data indicate that in patients whose predominant symptom is abdominal bloating, the subjective sensation of abdominal distention reflects true changes in the abdominal wall. However, only in rare cases of severe intestinal dysmotility is distention related to a real increase in intra-abdominal content, whereas in the vast majority of patients (ie, those with functional gut disorders) distention is due to dysregulation of abdominal wall activity and caudo-ventral displacement of content (Figure 6).

The primary objective of this study was to unravel the origin and mechanism of bloating; for this purpose, 2 distinctly different groups of patients reporting abdominal bloating were selected: a large pool of patients with functional gut disorders (ie, with no detectable cause of their symptoms) and a very select group of patients with severe intestinal motility impairment and abnormal intestinal manometry who were studied as disease controls. While the former represent the vast majority of patients consulting for bloating, the latter are, by contrast, extremely rare and only seen with relative frequency at specialized referral centers.

During basal conditions (ie, when the bloating sensation was absent or mild), the abdominal conditions differed in these 2 patient groups: intra-abdominal gas volume was normal in functional patients but was increased in dysmotility patients, who nevertheless did not perceive it as abnormal. In a previous study using the same methodology, we had already observed that during basal conditions (ie, on a normal day), the volume and distribution of intestinal gas in patients with functional gut disorders were similar to those in healthy subjects.[18] The finding that during basal conditions intestinal gas content in patients with functional gut disorders did not exceed that observed in healthy subjects was originally rather unexpected, because functional patients, specifically those with irritable bowel syndrome and functional bloating,[3] have impaired handling of intestinal gas and develop retention and abdominal distention in response to exogenous gas loads that are well tolerated by healthy subjects.[4–8,19] Furthermore, other studies had shown that this type of patient has increased gas surface on plain abdominal radiographs,[20–22] although the accuracy of

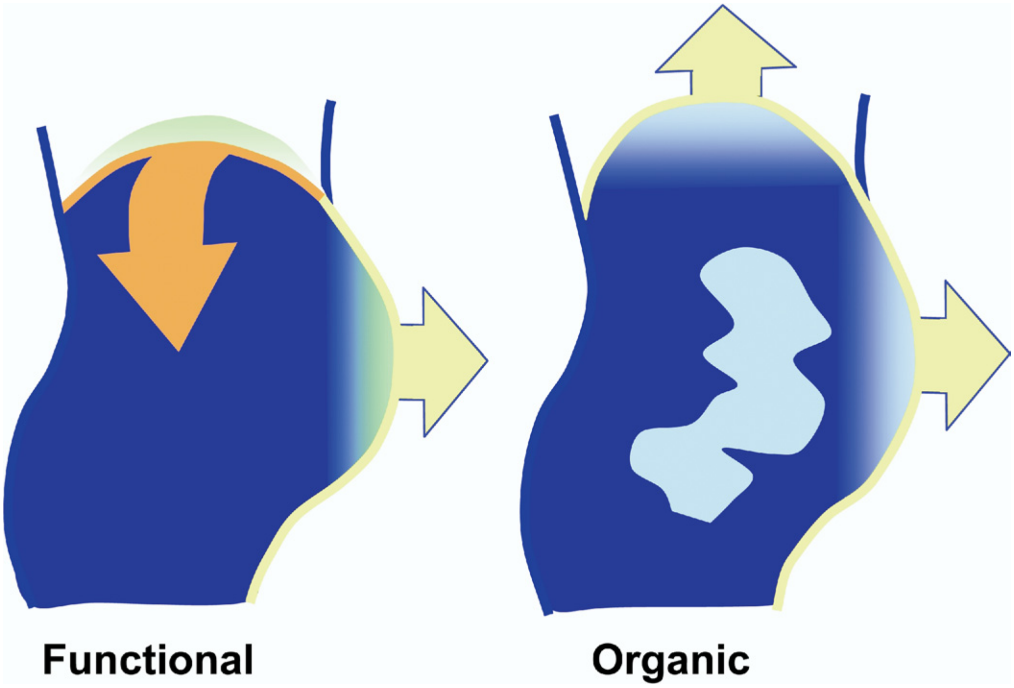

Figure 6. Mechanisms of bloating. In functional patients, abdominal bloating and distention are related to abdomino-phrenic displacement and ventro-caudal redistribution of contents, whereas organic bloating involves a true increment in intestinal content and abdominal expansion.

intestinal gas estimations based on radiologic observations is low.[23] However, these data were in contrast to direct measurements of intestinal gas using the washout technique, performed originally by Levitt's group[19] and later in our laboratory.[5,6,24–27]

The association of subjective bloating sensation and objective abdominal distention has been previously reported.[25–28] This association may depend on the clinical characteristics of the patients and hence the inclusion criteria of each study, being more patent in patients whose predominant symptom is bloating, as in our study, and less in diarrhea-predominant irritable bowel syndrome.[26] Because bloating is usually episodic, the association of bloating with distention may be clearer when comparing a severe bloating episode with basal than when measuring diurnal variations during a nonselected day.[26,27]

Our study shows major differences in the origin of abdominal distention between functional and organic bloating. In patients with dysmotility, the situation seems clear-cut: bloating is due to an increase in gas content and intra-abdominal volume that produces displacement of the abdominal walls (Figure 6). In contrast, patients with functional gut disorders showed during bloating episodes only a minor, although significant, increase in intestinal gas and total intra-abdominal volume. Recent data on abdominal accommodation of intestinal gas loads in healthy subjects[29] indicate that these volume increments observed in functional patients do not justify their increase in girth and anterior wall protrusion. However, if not from the gut, where does distention come from? The present study shows for the first time that abdominal distention in functional patients is consistently associated with a significant diaphragmatic descent.

The volume of the abdominal cavity exhibits physiologic variations, and the walls adapt to its content. In a series of studies measuring the activity of the anterior abdominal wall and the diaphragm by electromyography, we showed that abdominal accommodation to volume loads is an active process controlled by a coordinated abdomino-phrenic response, so that, regardless of body posture, an increase in intra-abdominal volume is associated with both diaphragmatic ascent (ie, cephalic displacement) and anterior wall protrusion. In the upright position, this response is instrumented by an adaptive diaphragmatic relaxation coupled to a compensatory anterior wall contraction.[29] In a previous study, we showed that patients with functional gut disorders who reported bloating had an abnormal response to intra-abdominal volume increments; with the same volume of gas infused into the colon, they exhibited a paradoxical relaxation and exaggerated anterior wall protrusion.[30] Subsequent data using the same model further indicate that patients also respond with an abnormal diaphragmatic contraction.[31] Hence, abdominal distention in functional patients seems related to abdomino-phrenic dyssynergia and incoordinated abdominal accommodation (altered balance of forces between diaphragmatic contraction and anterior wall relaxation) resulting in caudo-ventral displacement of abdominal walls and content (Figure 6).

Patients with functional gut disorders who report bloating have been shown to have abnormal visceral reflexes leading to impaired handling of intestinal gas,[4,8,9] visceral hypersensitivity,[27,32,33] and abnormal viscerosomatic responses with impaired abdominal accommodation to volume loads.[30,31] How would all these diverse pieces of information fit with the current data to explain the mechanism of bloating? Impaired visceral reflexes may result in abnormal propulsion of gaseous, and probably also nongaseous, intestinal contents with focal pooling and segmental stretch of the gut. In patients with visceral hypersensitivity this may originate bloating sensation, even without a true increment in girth.[27] Pooling of gut content may also release abnormal viscerosomatic responses with abdomino-phrenic incoordination and anterior wall protrusion.[30,31]

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Conflicts of interest

The authors disclose no conflicts.

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