Gastrointestinal gas volume measurement with CT: Patients with functional GI disorders have less gas

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**Purpose**

*Functional Gastro-intestinal Disorders - An Overview of Symptoms*

Functional gastro-intestinal disorders (FGIDs) including irritable bowel syndrome (IBS) are a diverse group of disorders. FGIDs are characterised by gastrointestinal (GI) tract dysfunction in the absence of organic disease, macro- or microscopic, and may affect the GI tract from oesophagus to anus.

The classical symptom of FGIDs is bloating. [1, 2] In layman's terms, bloating is considered synonymous with distension. Yet, in the FGID literature, the two are very separate entities; distension is an objective increase in abdominal girth and bloating is a subjective perception of increased abdominal gas. Much research effort has been focused on characterising the relationship between distension and bloating and determining the cause of bloating. [3, 4] Bloating is a symptom that displays a diurnal variation, worsening later in the day and improving overnight. There is a correlation between bloating and distension as bloating has been associated with increased abdominal girth. [5] Furthermore, different subtypes of IBS have different patterns of bloating and distension. In a questionnaire-based study of 714 patients with IBS, patients with constipation-predominant IBS complained of both bloating and distension whereas those with diarrhoea-predominant IBS only complained of bloating. [6]

*The Aetiology of Symptoms*

Gas is normally present in the GI tract of all humans. Using different techniques, the volume of human intestinal gas (intestinal gas volume, IGV) has been calculated to be about 100 ml, ranging from 31 to 200 ml. [7] The handling of this gas in patients with FGIDs is known to be impaired. These patients' GI tracts display a variety of defective or deficient homeostatic mechanisms for the normal transit of gas from mouth to anus.

Knowledge of gas handling by the GI tract in FGIDs is based on empirical evidence. Firstly, when transit was pharmacologically decreased in the non-functional gut symptoms arose akin to those in FGIDs. [8] Decreased transit is only part of the picture, however, as obstructed evacuation of gas causes more symptoms than decreased transit. [9] Abnormal retention is also a factor; by infusing gas into the jejunum, Serra et al. showed patients with IBS retained gas more than healthy controls and this retention caused symptoms. [10] In a further study by Salvioli, scintigraphy was used to determine the location of this retained gas in IBS patients. [11] Gas was seen to be retained within the small intestine more often in IBS when compared to non-FGID controls. These results were congruent with Harder et al. who demonstrated jejunal gas infusion caused more symptoms than rectal infusion despite absolute abdominal distension being the same.
The unifying mechanism causing symptoms in these patients is focal dilatation of the GI tract induced by gas. In FGIDs, the distribution of intestinal gas determines a patient’s bloating symptoms; the absolute IGV determines distension.

Neurological dysfunction is a postulated mechanism in the pathogenesis of bloating and is seen in the musculoskeletal system. In an early study on IBS patients by Maxton et al., the known diurnal variation in symptoms was not reflected in a change in IGV but rather by an altered AP diameter of the abdomen. In a recent CT-based study investigating the change in distribution of gas during symptomatic periods, Accarino described caudo-ventral redistribution of abdominal contents in 47 patients with FGIDs. The diaphragm descended and anterior abdominal wall protruded yet there was no significant change in IGV when these patients were symptomatic. This study with relatively large patient numbers yielded interesting results yet confounders such as co-morbid organic disorders were not considered.

Gas Quantification in Patients with FGIDs

Normal values for IGV range from 31-200ml according to early studies. The seminal work done in IGV in IBS patients was done in the 1970s by Lasser et al. They used a washout technique to show IGV to be no different in IBS patients than in healthy controls. More recently, various in vivo techniques to estimate IGV have been studied including abdominal inductance plethysmography, radioisotope studies, plain film radiography, and computed tomography (CT).

It is tempting to use imaging for estimation or direct quantification of IGV as one may visualise gas collections directly without relying on surrogate markers such as symptoms. Using plain abdominal radiography, Chami et al. showed IGV to be increased in IBS patients compared to healthy controls. Seike et al. considered the case of FGIDs which developed post anterior resection and showed these patients to have increased gas in the left colon although this study was confounded greatly by the patients' bowel surgery. Koide et al. developed a scoring system to grade IGV and showed the score to be increased in patients with IBS. In opposition to these results, Morken et al. showed a gas score calculated from plain abdominal radiographs to be unchanged after lactulose challenge which provoked symptoms in patients with IBS. The studies that employed plain radiography are contradictory, have relied on small numbers, and are inherently limited by projection radiography.

The studies that employed CT to measure IGV may be expected to quantify the volume most accurately. An early study by Maxton suggested a non-significant trend for increased IGV in IBS. Accarino showed patients with IBS have similar IGVs to healthy controls at baseline in random samples. In a later study studying the difference in volume between IBS patients at baseline and when symptomatic, Accarino showed.
no difference in IGV but did detect significant differences in abdominal distension and
diaphragmatic descent. [15] The only study to date that included an appropriate validation
was that by Perez et al. who assessed the affect of meals on gas distribution in both
FGID patients and in healthy subjects. This study showed that meals increase IGV among
FGID patients. [23] To date, the CT studies of IGV in IBS suggest there is no difference
in IGV between IBS patients and healthy controls. However, they have relied on well
controlled circumstances and have not accounted for any co-morbid GI diagnoses that
may confound IGV measurement.

**Treatment of Excess Intestinal Gas:**

If increased intestinal gas is present in patients with FGIDs, it is a realistic therapeutic
target. Several trials of therapy to reduce intestinal gas have not had encouraging results.
Caldarella et al. showed the acetylcholinesterase inhibitor neostigmine to be no better
than a normal saline placebo in both patients with FGIDs and normal controls. [24] In
a review in 2004, Azpiroz et al. found poor evidence to support the use of simethicone
or charcoal to reduce IGV. [25] Using CT to measure gas volume, Accarino et al. found
pyridostigmine (another acetylcholinesterase inhibitor) to improve bloating symptoms in
patients with FGIDs but have no effect on IGV. [22] Thus, increased IGV may be a viable
therapeutic target yet there is a lack of efficacious therapies to reduce it.

**Purpose:**

Empirical studies have shown patients with FGIDs to retain gas (distend) and become
symptomatic (bloat). Outside these experimental conditions, it is difficult to assess what
underlies symptoms. Ideally, one would be able to detect focally regional abnormalities
when the patients are symptomatic. Yet, before this is attempted, the effect of GI co-
morbidities on IGV must be determined to allow one to adequately control for this factor.

**Methods and Materials**

All patients (n=1909) attending a gastroenterology out-patients clinic with a special focus
on FGIDs over a ten year period (January 1999-January 2009) were identified. Their
records were reviewed and their diagnoses were coded according to the International
Classification of Disease (ICD-10) classification and the Rome III criteria for FGIDs.
Patients were grouped into three groups based on diagnoses: Organic GI Disorders
(OGID, n=84) for patients with an organic, non-functional disorder, Functional GI Disorder
(FGID, n=36) for patients with a functional disorder but no organic disorder, and Organic-
Functional GI Disorder (OFGID, n=87) for patients with both organic and functional
disorders. (Figure 1 on page 5)
Those with abdominopelvic CT during the study period (n=207) were selected and their CTs (n=312) were retrieved in DICOM format. Institutional abdominopelvic CT protocol involves fasting from midnight and oral and rectal contrast. CTs for all indications were included and patients with prior bowel surgery or a pathological gas collection (inflammation, obstruction, and abscess) were excluded. For patients with more than one study the most recent study was considered for analysis.

Two independent readers blinded to diagnostic group calculated IGV using OsiriX (OsiriX Foundation, Geneva, Switzerland) with 3D region growing (threshold -1024HU to -300HU). (Figure 2 on page 6) To calculate the Intestinal Gas Volume (IGV), firstly all intraluminal gas in the abdominal cavity was included in the region growing. Seed points were entered manually in all collections of gas and the total Gastro-intestinal Gas Volume (GGV) was determined. (Figure 3 on page 6) Then, to calculate IGV, the volume of gas in the stomach was subtracted from the total GGV to yield only intra-intestinal gas.

To control for body habitus which is correlated with IGV, the BMI was calculated from anthropometric measures of a single image at the L1 vertebral level according to published research. [26] (Figure 4 on page 7)

All data were stored and cleaned in a Microsoft Access 2007 database and statistical analysis of results was performed in SPSS version 14. For continuous variables, either ANOVA and Student's t-test or the Kruskall-Wallis and Mann-Whitney U-tests were used depending on the normality of the sample distribution. Spearman correlations for age and IGV were performed. Simple linear regression with IGV as the dependent variable was performed to assess factors including BMI, gender, age at CT study, and diagnostic group. In addition, stepwise inclusion of factors was performed to identify those factors most strongly determinant of IGV.

Images for this section:

<table>
<thead>
<tr>
<th>Group</th>
<th>Patients</th>
<th>CT studies</th>
</tr>
</thead>
<tbody>
<tr>
<td>FGID</td>
<td>36</td>
<td>51</td>
</tr>
<tr>
<td>OGIS (organic, non-FGID GI disorder)</td>
<td>84</td>
<td>145</td>
</tr>
<tr>
<td>OFGID (both)</td>
<td>87</td>
<td>116</td>
</tr>
<tr>
<td>Total</td>
<td>207</td>
<td>312</td>
</tr>
</tbody>
</table>
Fig. 1: Study groups: Number of subjects and CT studies

Fig. 2: Sample gas volume calculation with threshold-based segmentation.
Fig. 3: Volume rendering of the total gas in a sample human colon
Fig. 4: Anthropometric measures for derived BMI
Results

When comparing variables by diagnostic group, inter-group differences were seen to be significant for age and IGV. (Figure 1 on page 10) BMI was seen to be comparable between diagnostic groups and within diagnostic group by gender. (Figure 2 on page 10)

To further investigate the significant differences, post hoc Student's t-tests showed ages to be similar within diagnostic groups by gender yet significantly different between groups. (Figure 3 on page 10) Patients in the FGID group were younger at the time of the study (mean 36.2yrs, 56.6yrs, and 46.7yrs for FGID, OGID, and OFGID, respectively; p<0.001 for all differences).

The median calculated GGVs were as follows: OGID, 283.7mls; FGID, 250.5mls; OFGID, 207.2mls. After removing the stomach volume, median IGVs were determined to be 220.6ml, 197.6ml, and 155.0ml for the OGID, FGID, and OFGID groups respectively. The difference between OGID and OFGID was significant (p=0.017). (Figure 4 on page 11)

When considering the effect of the time of day on IGV, there was no difference by diagnostic group in IGV between studies performed before 1200 and those after 1200. When grouping by gender within diagnostic groups, only the males with FGIDs scanned before 1200 have significantly higher IGV than those scanned after 1200. (Figure 5 on page 11)

For those patients with IBS, there was no difference by diagnostic subgroup (constipation predominant, diarrhoea predominant, and alternating) in IGV, age at study, BMI, or time of study.

There was a significant positive correlation between age and IGV for patients in OGID (Spearman's=0.253, p=0.02); the correlation was non-significant in the other groups.

As IGV was non-parametric, the linear regression model was computed with log(IGV) as the dependant variable. Factors included in the model were age at study, BMI, gender, diagnostic group, and time of study. (See Figure 6 on page 11)

The $r^2$ of 0.119 suggests 11.9% of the variation in log(IGV) is explained by the model. A stepwise model was also computed and revealed age at study, gender, and calculated BMI to predict the log IGV with an $r^2$ of 0.116. (See Figure 7 on page 11)
Images for this section:

<table>
<thead>
<tr>
<th>Diagnostic Group</th>
<th>Gender</th>
<th>n</th>
<th>Mean Age at Study (years) [SD]</th>
<th>Mean BMI (kg/m^2) [SD]</th>
<th>Median IGV (ml) [IQR]</th>
<th>Median IGV (ml) [IQR]</th>
</tr>
</thead>
<tbody>
<tr>
<td>FGID</td>
<td>Female</td>
<td>26 (72%)</td>
<td>35.0 [16.8]</td>
<td>26.8 [5.5]</td>
<td>202.8 [136.7]</td>
<td>197.6 [138.2]</td>
</tr>
<tr>
<td>O gid</td>
<td>Female</td>
<td>48 (57%)</td>
<td>56.6 [19.7]</td>
<td>24.2 [6.6]</td>
<td>170.9 [204.0]</td>
<td>220.6 [279.3]</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>36 (43%)</td>
<td>56.6 [17.9]</td>
<td>26.1 [5.7]</td>
<td>304.3 [231.2]</td>
<td></td>
</tr>
<tr>
<td>OFGID</td>
<td>Female</td>
<td>55 (63%)</td>
<td>45.1 [15.0]</td>
<td>25.0 [5.1]</td>
<td>150.7 [145.1]</td>
<td>155.0 [170.0]</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>32 (37%)</td>
<td>49.4 [14.4]</td>
<td>27.2 [5.7]</td>
<td>180.6 [187.8]</td>
<td></td>
</tr>
</tbody>
</table>

p-value* <0.001 0.18 0.008 0.05

*ANOVA for Age at Study, BMI; Kruskall-Wallis for IGV

Fig. 1: Summary inter-group differences

<table>
<thead>
<tr>
<th>Diagnostic Group</th>
<th>Gender</th>
<th>n</th>
<th>Mean BMI (kg/m^2) [SD]</th>
<th>p-value*</th>
<th>p-value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>FGID</td>
<td>Female</td>
<td>26 (72%)</td>
<td>26.8 [5.5]</td>
<td>0.88</td>
<td>0.18</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>10 (18%)</td>
<td>26.5 [6.7]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ogid</td>
<td>Female</td>
<td>48 (57%)</td>
<td>24.2 [6.6]</td>
<td>0.16</td>
<td>0.38</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>36 (43%)</td>
<td>26.1 [5.7]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>OFGID</td>
<td>Female</td>
<td>55 (63%)</td>
<td>25.0 [5.1]</td>
<td>0.06</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>32 (37%)</td>
<td>27.2 [5.7]</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Student's t-test

Fig. 2: BMI by diagnostic group and gender

<table>
<thead>
<tr>
<th>Diagnostic Group</th>
<th>Gender</th>
<th>n</th>
<th>Mean Age at Study (years)</th>
<th>p-value*</th>
<th>Mean Age at Study (years)</th>
<th>p-value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>FGID</td>
<td>Female</td>
<td>26 (72%)</td>
<td>35.0 [16.8]</td>
<td>0.45</td>
<td>36.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>10 (18%)</td>
<td>39.4 [11.6]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ogid</td>
<td>Female</td>
<td>48 (57%)</td>
<td>56.6 [19.8]</td>
<td>0.99</td>
<td>56.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>36 (43%)</td>
<td>56.6 [17.2]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>OFGID</td>
<td>Female</td>
<td>55 (63%)</td>
<td>45.1 [15.0]</td>
<td>0.19</td>
<td>46.7</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>32 (37%)</td>
<td>49.4 [14.4]</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Student's t-test
**Fig. 3:** Age at study by diagnostic group and gender

<table>
<thead>
<tr>
<th>Diagnostic Group</th>
<th>Gender</th>
<th>n</th>
<th>Median IGV (ml) [IQR]</th>
<th>p-value*</th>
<th>Total Median IGV (ml) [IQR]</th>
<th>p-value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>FGID</td>
<td>Female</td>
<td>26 (72%)</td>
<td>202.8 [136.7]</td>
<td>0.9</td>
<td>197.6 [138.2]</td>
<td>0.23</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>10 (18%)</td>
<td>184.6 [216.4]</td>
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</tr>
<tr>
<td>OGDID</td>
<td>Female</td>
<td>48 (57%)</td>
<td>170.9 [204.0]</td>
<td>0.01</td>
<td>220.6 [279.3]</td>
<td>0.017</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>36 (43%)</td>
<td>304.3 [231.2]</td>
<td></td>
<td></td>
<td>0.29</td>
</tr>
<tr>
<td>OFGDID</td>
<td>Female</td>
<td>55 (63%)</td>
<td>150.7 [145.1]</td>
<td>0.12</td>
<td>155.0 [170.0]</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>32 (37%)</td>
<td>180.6 [187.8]</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Mann-Whitney U-test

**Fig. 4:** IGV by diagnostic group and gender

<table>
<thead>
<tr>
<th>Diagnostic Group</th>
<th>Gender</th>
<th>n Pre-Noon</th>
<th>Pre-Noon Median IGV (ml)</th>
<th>n Post-Noon</th>
<th>Post-Noon Median IGV (ml)</th>
<th>p-value* (pre vs post by gender)</th>
<th>p-value* (post vs post by gender)</th>
</tr>
</thead>
<tbody>
<tr>
<td>FGID</td>
<td>Female</td>
<td>5</td>
<td>187.7 [202.4]</td>
<td>21</td>
<td>209.6 [175.8]</td>
<td>0.466</td>
<td>0.49</td>
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<tr>
<td></td>
<td>Male</td>
<td>3</td>
<td>465.8</td>
<td>7</td>
<td>143.3 [123.2]</td>
<td>0.03</td>
<td></td>
</tr>
<tr>
<td>OGDID</td>
<td>Female</td>
<td>9</td>
<td>149.5 [281.7]</td>
<td>39</td>
<td>173.8 [198.3]</td>
<td>0.316</td>
<td>0.57</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>9</td>
<td>217.3 [304.2]</td>
<td>27</td>
<td>334.6 [209.5]</td>
<td>0.23</td>
<td></td>
</tr>
<tr>
<td>OFGDID</td>
<td>Female</td>
<td>12</td>
<td>116.6 [108.2]</td>
<td>43</td>
<td>154.4 [181.9]</td>
<td>0.209</td>
<td>0.21</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>6</td>
<td>224.4 [244.8]</td>
<td>26</td>
<td>180.6 [190.6]</td>
<td>0.91</td>
<td></td>
</tr>
</tbody>
</table>

*Mann-Whitney U-test

**Fig. 5:** Pre-Noon and Post-Noon IGV by diagnostic group and gender

**Fig. 6:** Simple linear regression model

\[
\log_{10}(IGV) = -0.159a + 0.055b - 0.047c - 0.01d + 0.233e + 0.004f + 2.306
\]

\[a = \text{Gender (0=male, 1=female)}\]
\[b = \text{FGID (1=yes, 0=no)}\]
\[c = \text{OFGID (1=yes, 0=no)}\]
\[d = \text{BMI (kg/m2)}\]
\[e = \text{Time stamp (Decimal form ie 12:00 = 0.5, 18:00 = 0.75)}\]
\[f = \text{Age at study (years)}\]

\[r^2 = 0.119\]
**Fig. 7**: Stepwise linear regression model

<table>
<thead>
<tr>
<th>Stepwise Linear Regression Model</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\log_{10}(IGV) = 0.004a - 0.154b - 0.010c + 2.435$</td>
</tr>
<tr>
<td>$a = \text{Age at study (years)}$</td>
</tr>
<tr>
<td>$b = \text{Gender (0=male, 1=female)}$</td>
</tr>
<tr>
<td>$c = \text{BMI (kg/m2)}$</td>
</tr>
<tr>
<td>$r^2 = 0.116$</td>
</tr>
</tbody>
</table>
Conclusion

BMI was seen to be uniform by group and gender, suggesting that the study population was fairly homogeneous in terms of body habitus. It is reasonable to expect BMI to be correlated with IGV as the larger a person is, the greater the volume of the abdomen and the greater the volume of abdominal gas. The effect of BMI on IGV would be expected to be the same for all diagnostic groups and genders.

Different diagnostic groups are seen to have significantly different mean ages (56.6yrs for OGID, 36.2yrs for FGID, and 46.7yrs for OFGID). This difference may be explained in context of the demographics of the patient groups. Those with functional disorders tend to be younger and those with organic disorders tend to be older. Indeed, the longer one lives, the more likely one is to acquire a diagnosis of any kind. However, many clinicians are slow to diagnose a functional disorder in a patient of advanced age and this may confound the results.

The median IGVs (220.6ml for OGID, 197.6ml for FGID, and 155.0ml for OFGID) were significantly different from one another (p<0.05, Kruskall-Wallis). Post hoc Mann-Whitney U-tests with Bonferroni correction revealed the underlying difference between OGID and OFGID to be significant (p=0.017). This result is somewhat difficult to rationalise when only considering diagnosis as a determinant of IGV. However patients in OFGID have more diagnoses by definition and so are more likely to be investigated and treated. This may contribute to selection bias in terms of those patients that have a CT abdomen and pelvis and also may reflect the action of therapy that alters IGV.

IGV is known to vary in a diurnal pattern with it being higher in the evening; this normal increase is known to correlate with an increase in reported symptoms in the evenings for patients with FGIDs. [4] The only difference, that for FGID males between pre- and post-noon studies, is likely an artefact of small numbers (n=3 for pre-noon group). Maxton et al. considered the difference between pre- and post-noon studies in patients with IBS and found no difference in IGV. [14] As the vast majority of studies included in this study were outpatient studies for patients that underwent standard fasting and bowel prep, the expected rise in IGV may have been masked by factors in the CT study preparation.

The most common FGID in the study population, IBS, is a syndrome with several diagnostic subgroups. Constipation-predominant, diarrhoea-predominant, and alternating IBS are recognised entities and the patients in this study were grouped accordingly. Accarino et al. only considered constipation predominant and alternating IBS in their analysis as they observed the diarrhoea-predominant to be less associated with bloating symptoms. [15] In this study, there was no difference by diagnostic subgroup in
age at study, BMI, or IGV. As there was no difference in IGV by IBS subtype, the bloating symptoms of the constipation predominant group Accarino investigated are likely due to a factor other than IGV.

There was a significant positive correlation between age and IGV for patients in OGID (Spearman's=0.253, p=0.02) yet the correlation was non-significant in the other groups. This reflects the advanced age of some of the subjects in OGID. A postulated increase in compliance of the muscular wall of the gut explains this relationship.

Factors included in the regression model were age at study, BMI, gender, diagnostic group, and time of study. The $r^2$ of 0.119 suggests only 11.9% of the variation in IGV can be explained by these factors. The unexplained variation may be due to inherent variation and inadequate control within the study methodology or due to the presence of lurking variables not considered (for example, fasting status, medications, actual diagnoses). The stepwise model computed showed age at study, gender, and calculated BMI to predict the log IGV with an $r^2$ of 0.116. When considering the stepwise model does not include diagnostic group and still maintains a very similar $r^2$, diagnostic group is seen to be poorly predictive of IGV.

In conclusion, the results of this study are in keeping with the published literature on the subject in addition to recently published work by Accarino et al. [15] Patients with FGIDs do not have increased IGV over those with organic disorders despite bloating being one of the most common symptoms of those with FGIDs. Accarino showed patients with IBS to have total volumes no different from healthy controls but have abnormal gas distribution during symptomatic episodes (caudo-ventral redistribution). In light of this study's results and recent published work, total intestinal gas volume would not seem to be an adequate therapeutic target for patients with functional gastro-intestinal disorders.

**References**


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