LEADING ARTICLE

From comic relief to real understanding; how intestinal gas causes symptoms

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Gas content and transit appear to conspire with the motor and sensory responses of the gut to produce gas related symptoms, both in normal individuals and especially in patients with irritable bowel syndrome (IBS). In relation to gas in IBS, two questions need to be addressed: do IBS patients produce more gas and what are the relationships between intestinal gas and symptoms? The balance of evidence seems to indicate that distension is a real phenomenon in IBS and that such distension accurately reflects gas content. More problematic is extrapolation of the observations relating symptoms to gas transit and retention.

Patients with a variety of functional gastrointestinal symptoms commonly ascribe many of their varied and often distressing symptoms to "gas", firmly convinced that an excess of "gas" or "wind" is the root cause of their belching, postprandial fullness, bloating, distension, or flatulence. By the time they seek help from the gastroenterologist, many will have attempted radical dietary adjustments, as well as all that the health food store and high street pharmacy have to offer, to relieve or eradicate "gas".

These symptoms are especially prominent among those who suffer from irritable bowel syndrome (IBS), for whom bloating, distension, and flatulence are significant and sometimes overriding concerns. Long the sole preserve of the music hall and the stand up comedian, the application of serious science to the area of intestinal gas has been long overdue. It is also regrettable that these symptoms have tended to be trivialised and dismissed as imaginary by the medical practitioner; recent studies of relationships between symptoms and quality of life in IBS attest not only real but also dynamic and subject to significant diurnal variation. 1–19 Koide and his group, in the 1970s, so convincingly debunking the concept of excess gas in IBS, 13 may have inadvertently contributed to the common tenet that distension and related symptoms were imagined. The clinician was reinforced further in this opinion every time that a distressed female who insisted that she felt "three months pregnant" by the end of the day was, hey presto!, found to have a scaphoid and non-tympanitic abdomen when the great hands were laid. Prefacing his comments with a reassuring "well dearie", the learned man then proceeded to explain the phantom pregnancy in terms of "curvature of the spine", "all in the head!", and "weak abdominals". More recently, some doubts began to creep in with the publication of a number of studies employing disparate methodologies suggesting that distension was not only real but also dynamic and subject to significant diurnal variation. 15–19 Maxton et al, using plain abdominal radiographs, suggested that intestinal gas was increased in IBS. 15

If total gas content is not increased in IBS how then can these patients develop such distressing "gas related" symptoms? The studies from Levitt and his group, in the 1970s, so convincingly debunking the concept of excess gas in IBS, 13 may have inadvertently contributed to the common tenet that distension and related symptoms were imagined. The clinician was reinforced further in this opinion every time that a distressed female who insisted that she felt "three months pregnant" by the end of the day was, hey presto!, found to have a scaphoid and non-tympanitic abdomen when the great hands were laid. Prefacing his comments with a reassuring "well dearie", the learned man then proceeded to explain the phantom pregnancy in terms of "curvature of the spine", "all in the head!", and "weak abdominals". More recently, some doubts began to creep in with the publication of a number of studies employing disparate methodologies suggesting that distension was not only real but also dynamic and subject to significant diurnal variation. 15–19 Koide et al, using plain abdominal radiographs, suggested that intestinal gas was increased in IBS. 15

Abbreviations: IBS, irritable bowel syndrome

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cavity. Even the least reproducible of these methods, direct measurement of abdominal girth, demonstrated a progressive increase during the day. Lewis et al and Lea et al, using applied potential tomography, have confirmed and extended these observations. Their studies revealed considerable fluctuations in abdominal girth over a 24 hour period; girth increased following meal ingestion and was lowest during sleep. Interestingly, they and others recorded a poor correlation between measured distension and symptoms.

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A new concept emerges from these observations: changes in gas content or volume do not necessarily imply an increase in gas production and could arise within the intestine as a result of altered transit and/or expulsion, without any change in endogenous production; the patient’s much derided description of “trapped gas” may, after all, owe more to physiology than imagination!

GAS PERCEPTION

Impaired transit alone cannot however explain gas related symptoms. A divergence between measured distension on the one hand and symptoms on the other has been a consistent feature of this and other studies. In the glucagon experiments, visible distension was asymptomatic; yet in the same study a similar degree of retention and distension induced by voluntary suppression of gas evacuation resulted in an increase in symptoms. Similarly, gas retention exacerbated in IBS subjects by intraduodenal lipid infusion led to an increase in abdominal girth but no change in perception. Symptoms may well result therefore not from gas retention per se but from the local response to this stimulus; the small intestine contracts with a resultant increase in wall tension, thereby inducing symptoms; the colon relaxes and accommodates. When the small intestine is similarly relaxed by glucagon for example, gas infusion no longer produces symptoms.

“Impaired transit alone cannot however explain gas related symptoms.”

One can only speculate on what may transpire in the IBS patient; could for example the hold up in transit described by others at the ileocolonic junction result in small intestinal gas retention and thus precipitate bloating?

In summary, gas content and transit appear to conspire with the motor and sensory responses of the gut to produce gas related symptoms, both in normal individuals and especially in IBS patients. Gas retention in the small intestine, an organ poorly prepared for this event, is more likely to cause symptoms, especially in an individual who exhibits visceral hypersensitivity. Are these elegant experimental studies relevant to IBS in general? Certainly the balance of evidence does seem to indicate that distension is a real phenomenon in IBS and that such distension accurately reflects gas content. More problematic is extrapolation of the observations relating symptoms to gas transit and retention. The Barcelona group have convinced us that abnormal transit of exogenous gas is a real phenomenon in IBS; what is in question is whether these levels of gas infusion into the proximal small intestine have any relevance to IBS? Could these additional gas loads occur under non-experimental circumstances in IBS? The suggestion that bacterial overgrowth or changes in the bacterial flora may occur in IBS would provide one scenario conducive to local increases in gas content; both proposals remain however highly contentious. We know little of the relationships between...
aerophagia, the other and more likely source of excess gas, and IBS. Resolution of this issue must await studies which can simultaneously trace the movement of intrinsic or “normal” gas volumes through the intestine, detect changes in abdominal girth, and record associated symptoms.

REFERENCES


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