What is irritable bowel syndrome?

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The term irritable bowel syndrome (IBS) has been used for many years to describe a collection of symptoms consisting of abdominal pain which is often associated with bloating and accompanied by some form of bowel dysfunction which is usually classified as constipation, diarrhoea or an alternation between the two. Characteristically, the stools can be pellety, stringy or loose and following defecation there is often a feeling of incomplete evacuation. In this classic form, and in the absence of alarm features, the diagnosis is usually obvious to the experienced functional gastroenterologist familiar with the nonverbal clues that can help to substantiate the clinical impression. With this positive approach to diagnosis, investigation should be aimed at excluding realistic alternatives rather than being exhaustive and there is evidence to suggest that significant disease is seldom missed by following such a course of action [Vanner *et al.* 1999; Jellema *et al.* 2009; Ishihara *et al.* 2012]. However, very reasonably, others feel extremely uncomfortable with this symptom-based style of diagnosis and think that a more scientific, pathophysiology-based approach should be developed. A good example in support of this view is the fact that when, for instance, diarrhoea-type IBS is investigated with a SeHCAT test a significant proportion of patients turn out to have bile acid malabsorption and not IBS [Wedlake *et al.* 2009; Kurien *et al.* 2011; Gracie *et al.* 2012; Wong *et al.* 2012].

It does seem somewhat paradoxical that the same name is currently applied to, for example, patients with abdominal pain in whom the bowels are open once a week as well as others who constantly suffer from diarrhoea. From these and other observations it seems reasonable to conclude that it is unlikely that IBS, as we define it today, has a single cause and that it probably results from a variety of distinct pathophysiological processes. However, as these various entities are searched for and identified in patients with presumed IBS, it is possible that we may still be left with patients who are suffering from ‘pure’ IBS.

Table 1. Factors considered to contribute to the expression of symptoms in patients with irritable bowel syndrome.

Inheritance
Disordered motility
Visceral hypersensitivity
Abnormal central processing of noxious peripheral stimuli
Bacterial imbalance
Inflammation
Dietary factors
Psychological influences

What could ‘pure’ IBS look like? From research to date, the concept of it just being a disorder of motility is now untenable and the evidence points to symptoms being associated with a whole variety of factors some of which are listed in Table 1. It is likely that an individual has to be initially genetically predisposed to the condition and that in such a person symptoms may or may not become manifest depending on a particular combination of different triggers, both medical and environmental, and their ‘dose’. This forms the basis of the biopsychosocial model of disease [Engel, 1977] of which IBS is held up to be a prime example [Drossman, 1996]. Thus, we have on the one hand a unidimensional (pathophysiology-based) concept of IBS where all patients will eventually be found to have a specific cause or, on the other hand, a multidimensional (biopsychosocial) model of the disorder although these two explanations may not necessarily be mutually exclusive. In this issue of the journal we have juxtaposed two papers written by internationally recognized authorities [Camilleri, 2012; Sperber and Drossman, 2012] arguing the case in support of the model to which they feel the medical community should be adhering. However, while this discussion rumbles on, two notes of caution need to be sounded.

First, we need to learn from the history of duodenal ulceration where until the discovery of

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Helicobacter pylori [Marshall and Warren, 1984] much of the research and thinking about this condition was very similar to that which now applies to IBS. Therefore, as is now the case for duodenal ulceration, there is just a chance that a single cause for the majority of cases of IBS might be found. Second, we need to think about the patient. There are not many things that are more important to the sufferer than to be given a diagnosis and the term IBS, however imprecise, currently serves that purpose. We therefore need to bear this in mind when thinking about how to advance diagnostic accuracy in patients with symptoms that we currently classify as being indicative of IBS. As we go through the process of ticking off the various conditions that can lead to an IBS-like syndrome, do we really want to go back to telling those who cannot yet be classified that we do not know what is wrong with them, as we used to do in the past, when IBS was considered to be a diagnosis of exclusion rather than a positive diagnosis?

References

- Camilleri, M. (2012) Irritable bowel syndrome: how useful is the term and the 'diagnosis'? *Therapeutic Advances in Gastroenterology* 27 March (epub ahead of print).
- Drossman, D. (1996) Gastrointestinal illness and the biopsychosocial model. *J Clin Gastroenterol* 22: 252–254.
- Engel, G. (1977) The need for a new medical model: a challenge for biomedicine. *Science* 196: 129–136.
- Gracie, D., Kane, J., Mumtaz, S., Scarsbrook, A., Chowdhury, F. and Ford, A. (2012) Prevalence of, and predictors of, bile acid malabsorption in outpatients with chronic diarrhea. *Neurogastroenterol Motil*, in press.
- Ishihara, S., Yashima, K., Kushiya, Y., Izumi, A., Kawashima, K., Fujishiro, H. *et al.* (2012) Prevalence of organic colonic lesions in patients meeting Rome III criteria for diagnosis of IBS: a prospective multi-center study utilizing colonoscopy. *J Gastroenterol*, in press.
- Jellema, P., Van Der Windt, D., Schellevis, F. and Van Der Horst, H. (2009) Systematic review: accuracy of symptom-based criteria for diagnosis of irritable bowel syndrome in primary Care. *Aliment Pharmacol Ther* 30: 695–706.
- Kurien, M., Evans, K., Leeds, J., Hopper, A., Harris, A. and Sanders, D. (2011) Bile acid malabsorption: an under-investigated differential diagnosis in patients presenting with diarrhea predominant irritable bowel syndrome type symptoms. *Scand J Gastroenterol* 46: 818–822.
- Marshall, B. and Warren, J. (1984) Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration. *Lancet* 1: 1311–1315.
- Sperber, A. and Drossman, D. (2012) Irritable bowel syndrome: a multidimensional disorder cannot be understood or treated from a unidimensional perspective. *Therapeutic Advances in Gastroenterology* (epub ahead of print).
- Vanner, S., Depew, W., Paterson, W., Dacosta, L., Groll, A., Simon, J. *et al.* (1999) Predictive value of the Rome criteria for diagnosing the irritable bowel syndrome. *Am J Gastroenterol* 94: 2912–2917.
- Wedlake, L., A'hern, R., Russell, D., Thomas, K., Walters, J. and Andreyev, H. (2009) Systematic review: the prevalence of idiopathic bile acid malabsorption as diagnosed by SeHCAT scanning in patients with diarrhoea-predominant irritable bowel syndrome. *Aliment Pharmacol Therapeut* 30: 707–717.
- Wong, B., Camilleri, M., Carlson, P., McKinzie, S., Busciglio, I., Bondar, O. *et al.* (2012) Increased bile acid biosynthesis is associated with irritable bowel syndrome with diarrhea. *Clin Gastroenterol Hepatol*, in press.