

Leading article

Functional gastroenterological disorders: the name's the thing . . .

Most of the patients, who are referred to gastroenterologists suffer not from conditions that cause identifiable changes in tissue histology or plasma biochemistry, but from disturbances in function or a disintegration of the activity of different regions of the gut. Until recently we have not had the means of identifying such disturbances of function. Patients with disease that cannot be diagnosed by routine gastroenterological screening have therefore been classified on the basis of their symptoms into categories, such as functional dyspepsia, the irritable bowel syndrome (IBS), idiopathic vomiting, idiopathic constipation, and chronic idiopathic intestinal pseudoobstruction (CIIP).

Giving a name to a disease that cannot be adequately classified creates an identity out of something that is hypothetical and insubstantial. No two doctors ever perceive such a condition in quite the same way and their perception often varies according to their speciality. For example, the surgeon may diagnose irritable bowel syndrome in patients with unexplained left iliac fossa pain, the gastroenterologist may regard the same condition as part of the differential diagnosis of diarrhoea while the general practitioner may see it as the reaction of the gut to intolerable social pressures. Once the name is accepted into our system of medical practice, its status is defended by all the devices that society uses to reinforce its own prejudices. New tests or measurements can help to establish a condition on a more scientific base, but unless such tests discriminate the disease from other conditions that may present in similar ways, they merely add a kind of intellectual varnish to a flakey diagnosis. A few years ago several groups of investigators reported that the myoelectrical activity of the sigmoid colon in patients with IBS showed a greater preponderance of low frequency oscillations than in normal subjects,^{1 2} and this finding began to assume the status of a marker for IBS. Subsequent experience has suggested that low frequency sigmoid oscillations are as common in normal subjects or in psychoneurotic patients, who have no bowel disturbance, as they are in patients with IBS³ and may, in any case, be influenced by the degree of sigmoid distension or faecal loading.⁴

Pharmaceutical companies may reinforce the 'scientific' characterisation of a functional disease in order to give credibility to their products. '.... is the drug of choice for colonic dyssynergia, because it restores the disturbed myoelectrical rhythm'. Such statements can be misleading unless the motor change clearly identifies the condition as a distinct entity and there is an established relationship between the symptoms and the alteration in electrical rhythm.

The term chronic idiopathic intestinal pseudoobstruction (CIIP) was coined by Maldonado in 1970⁵ to describe a condition, the symptoms and signs of which resembled intestinal obstruction, but without objective evidence of mechanical obstruction, or of any recognised disease such as systemic sclerosis. In addition, the five patients that he described had steatorrhoea and weight loss, which could progress to death from malnutrition. Patients with CIIP are said to present with recurrent attacks of nausea, vomiting, cramping abdominal pain, and abdominal distension, but unlike organic intestinal obstruction the presence of constipation and the absence of flatus are not prerequisites for the diagnosis.⁶⁻⁸ Distension is a common feature and may be observed as a swelling as large as a full term pregnancy.⁸ Patients may be embarrassed by loud borborygmi and splashes, as they move around. The abdominal pain is generally epigastric or periumbilical and may be partly, or completely relieved by a large emesis or the passage of flatus or faeces. Vomiting is often of large volume. Most patients have diarrhoea caused, it is thought, by an overgrowth of stagnant areas of small intestine by anaerobic bacteria. Some patients however, are constipated and others have alternating diarrhoea or constipation. Weight loss is common and may be considerable. The variability of the clinical presentation makes it difficult to discriminate CIIP from other conditions unless the features are as severe as those described in Maldonado's paper.⁵ What then would make us decide that the patient who has been attending our clinic for years with nausea, occasional bloating, abdominal pain and bowel disturbance has CIIP and not IBS? Is it a question of degree, or are there more objective criteria that can be used to identify CIIP?

The key to correct diagnosis of CIIP according to Schuffler,⁸ is 'well done radiology by an interested radiologist'. Plain films of the abdomen show small intestinal or colonic dilatations in most, but not in all patients with CIIP. The stomach is enlarged and gastric emptying is delayed in about one third of patients and contrast studies of the small bowel show either distension of the duodenum alone, or distension of ileum, or diffuse distension extending from the duodenum to the ileum. Radiology may also show areas of spasm or abnormal transit in many parts of the gastrointestinal tract. Thus the radiological features of CIIP are also variable and do not necessarily constitute precise diagnostic criteria. How many patients with irritable bowel syndrome and how many normal subjects would have similar findings when radiographed in the same way by the same interested and motivated radiologist? Although abnormalities in oesophageal manometry have been described in patients with CIIP, these are neither consistent nor discriminative. In any case, patients with CIIP do not usually complain of dysphagia or heartburn. Histology probably provides the best diagnosis, but cannot be justified unless the patient is severely ill; – full thickness biopsies of the affected areas of bowel show abnormalities of the myenteric plexus or smooth muscle in most cases of CIIP, if appropriate stains are used.^{7 8}

In a provocative and in many ways excellent paper, published in this edition,⁹ Stanghellini and his colleagues, working at the Mayo Clinic, have described abnormal patterns of pressure activity in multilumen manometric recordings in 42 patients said to have CIIP. All of these patients showed one or more of four 'abnormal' manometric features. These were:

(i) aberrant propagation and/or configuration of the interdigestive motor complexes, (ii) prolonged bursts of non-propagated pressure activity in the fasting and fed states, (iii) sustained and incoordinated phasic pressure activity, and (iv) inability of an ingested meal to convert a fasting state into a fed pattern of motility. If these patients really did have CIIP, this is the first systematic study of small bowel manometric patterns in this condition. But did they have CIIP? Although symptomatic presentation was compatible with the diagnosis, one is left with the impression that the selection criteria were perhaps softer than those used by other workers. Eighty three per cent of their patients presented with episodic nausea or vomiting, but only 57% had distension and diarrhoea was a feature in only 29% of patients. Radiology, the keystone of the diagnosis, showed distended small bowel loops or fluid levels in 57% of patients. Full thickness biopsies of the stomach, small bowel and colon were studied in 15 patients, but abnormalities were detected in only three of these.

Thirty six of the 42 patients in Stanghellini's study had previous abdominal surgery, and 10 had gastric resection and/or vagotomy. Although we are told that the surgical procedure did not affect their symptoms, the manometric findings could well have been influenced either by the vagotomy, or by the increased delivery of fluid to the small intestine that would follow partial gastrectomy, or even by postoperative adhesions and kinking of bowel loops. Abnormal reactions of the small intestinal motor activity to food have been described in patients after vagotomy¹⁰ and we have recorded prolonged bursts of non-propagated pressure activity during infusion of plasma-like solutions at 30 ml/min into the small bowel of normal volunteers.

Knowledge of the range of small bowel motor patterns in normal human subjects is still fragmentary. I remember several years ago observing frequent non-propagated bursts of motor activity in recordings carried out on myself while I was nauseated and had a severe headache. Some may doubt the normality of my mental state to carry out such studies, but I am pretty certain I do not have chronic idiopathic intestinal pseudoobstruction. Only further experience in small bowel manometry will tell us the degree to which the manometric findings described in Stanghellini's paper are abnormal and typical of CIIP. Without more evidence of the discriminative power of the observations, it is premature to state that 'the abnormalities can help us to establish the correct diagnosis'. In any case CIIP defines a heterogeneous group of propulsive disorders, some of which, such as hollow visceral myopathy, familial visceral neuropathy and megaduodenum,⁷ possess features that would allow them to be characterised as separate diseases. It would be interesting to know how the manometric features described in this paper relate to those harder diagnostic categories.

My comments are in no way meant to undermine the value of the data presented in the paper by Stanghellini and his colleagues. The authors have described a range of abnormalities in small bowel pressure activity in patients who have no demonstrable pathological condition in the small intestine. As such the data are unique and exciting and should generate enthusiasm for the clinical application of intestinal manometry. Whether the abnormalities constitute a distinct diagnosis, whether they contribute to the patient symptoms and whether they can be used to guide therapy are

crucial questions that must be answered by further studies carried out in similar patients.

The importance of the study is that it indicates for the first time that small intestinal manometry is feasible and useful in clinical investigation. The accessibility of techniques for recording gastrointestinal motor activity in patients and the rapid development of the science of gastrointestinal motility may well alter our perception of many of the conditions that we observe commonly in our clinics. The next 20 or 30 years could witness a proliferation of terms describing subsets of the irritable bowel syndrome, or functional dyspepsia. To be useful, these terms must be restricted to phenomena that can be clearly discriminated from other conditions. Failure of investigators to do this will cause misconception and disillusion in the doctors treating such conditions. If the nature of the condition defies proper characterisation, we must be alert to the possibility that the insubstantial terms we invent to help us understand the phenomena we perceive could actually obstruct further understanding and progress. A recent example of this is the term dietary fibre, which came in to popular usage at a time when we had little conception of what it was, yet alone how it worked. The growth in our knowledge of the chemistry and the physiology of fibre has made the term an encumbrance and nutritionists are at present devoting an inappropriate amount of energy to agonising over the definition of an effete term, in order to satisfy the demands to the food industry and the public. But that's another story!

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