During the 1970s, some reports described sensory disturbances of the gut in patients with irritable bowel syndrome (IBS) and related syndromes (1,2). For instance, Ritchie (1) reported increased pain from distension of the pelvic colon by inflating a balloon in patients with IBS; this observation was subsequently confirmed by other studies (3). Unfortunately, these studies remained largely ignored, until more recent data suggested that the symptoms in patients with functional gut disorders, specifically those with IBS, may be related to altered sensory function (4).
EXTENSION OF THE SENSORY DYSFUNCTIONS
The sensory dysfunction in patients with IBS has been investigated and characterized. First, it has been well established that the dysfunction affects exclusively visceral sensitivity. It has been shown that IBS patients with heightened perception of gut stimuli have normal or even increased tolerance of somatic pain (5). The latter has been explained on the basis of the pain reporting behaviour characteristics of many chronic painful conditions (5).

Visceral sensitivity in IBS has been further investigated to define the regions of the gut and the specific pathways affected. Assessment of visceral sensitivity requires probing stimuli that activate afferent pathways and induce perception. The most common stimulus applied in the gut has been distension (6). Distensions can be produced by manual inflation of intraluminal balloons by using a syringe, or with more sophisticated methods, such as the barostat, which applies fixed intraluminal pressures, or more recently the tensostat, which applies fixed tension levels to the gut wall (7). By applying distending stimuli to IBS patients, rectal as well as colonic hypersensitivity has been well documented (8,9), but it seems that more proximal portions of the gut, such as the jejunum, and even the esophagus, are also affected (10,11).

MECHANISMS OF HYPERSENSITIVITY
Increased sensitivity to mechanical stimuli may arise from reduced compliance of the gut wall; however, this hypersensitivity mechanism was ruled out in IBS patients because intestinal compliance was shown to be normal in most studies (8). However, distension tests alone did not provide further insight into the possible cause of hypersensitivity or the level of the dysfunction.

In analogy to somatosensory testing, which involves a variety of stimulation techniques to activate specific pathways, alternative stimuli to distension have been tested in the gut (6). Transmucosal electrical nerve stimulation has been applied in the gut via intraluminal electrodes mounted over a tube. Whereas distending stimuli activate sensory pathways and induce perception by specific stimulation of mechanoreceptors on the gut wall, transmucosal nerve stimulation induces similar perception by nonspecific stimulation of afferent pathways, that is, without relying on any specific receptor (12). Differential stimulation by a combination of techniques allowed a more precise characterization of sensory dysfunctions in IBS patients. Studies using both mechanical stimuli and transmucosal nerve stimulation have shown that patients with IBS have hypersensitivity of the small bowel, which selectively affects mechanosensitive afferents, with normal perception of electrical stimulation (8). Furthermore, these data indicate that a response bias can be reasonably excluded in IBS patients because transmucosal electrical nerve stimulation induces normal perception, even though electrical and mechanical stimuli produce similar sensations in most tests (8). These data suggest that small bowel hypersensitivity in IBS patients is related to a selective alteration of mechanosensitive pathways. The level of the afferent dysfunction in IBS has not been established. Normally, perception of gut stimuli is modulated by a series of mechanisms located at different states of the brain-gut axis (6). Theoretically, a dysfunction of these modulatory mechanisms could produce exaggerated gut perception and symptoms in response to physiological, otherwise unperceived stimuli.

A series of studies from the United Kingdom showed that patients with IBS and functional abdominal pain have a distorted referral pattern of gut sensations and perceive intestinal distensions more diffusely over the abdomen than healthy controls (13). Interestingly, normally perceived electrical stimuli also have expanded referral. Visceral and somatic afferents converge onto the same sensory neurons in the spinal cord, and sensitization of these neurons by noxious visceral input produces an expansion of their somatic receptive fields (14). Peripheral hypersensitivity of visceral sensory pathways may produce a secondary sensitization of spinal neurons, which may explain the expanded referral area of gut stimuli in IBS (15).

CLINICAL RELEVANCE
The clinical relevance of visceral sensory disturbances in patients with IBS is still unclear. It has been reported that patients with IBS may have significant discomfort during sigmoidoscopy, but when specifically tested they may exhibit normal thresholds to rectal balloon distension (15). This inconsistency has been addressed by exploring whether the IBS dysfunction affects exclusively sensory pathways or whether reflex pathways involved in the regulation of motility are also affected. Despite that gross motor abnormalities cannot be detected by manometry in IBS (10), some clinical data, such as the characteristic alteration of bowel habit, suggest that motility regulation may also be distorted (16). Indeed, it has been shown that IBS patients display exaggerated reflex responses to gut stimuli (3). For instance, rectal hypersensitivity in these patients is associated with rectal motor hyperactivity in response to distension and to ingestion of a meal. Both hypersensitivity and hyperreactivity may contribute to perception of rectal tenesmus and fecal urgency, which are common symptoms of IBS. Recent studies using a new methodology to evaluate intestinal gas dynamics further substantiate the role of combined sensory-reflex disturbances. These studies have shown that gas symptoms in IBS patients may be related to impaired intestinal handling of gas and retention, to gut hypersensitivity and poor gas tolerance, or to both (17).

CONCLUSIONS
Firm evidence collected over the past few years indicates that altered visceral sensory function may be implicated in the pathogenesis of IBS. However, the putative causes and the clinical implications of these disturbances remain to be established. Furthermore, altered conscious perception of gut stimuli seems to be associated with altered reflex activity. Sensory and reflex dysfunctions may combine to different degrees, and their interaction may explain the development of clinical symptoms.
REFERENCES


