The autonomic nervous system in functional bowel disorders

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In recent years, there has been a substantial shift in the conceptual definitions of what constitutes functional bowel disorders and the irritable bowel syndrome (IBS). In general, these conditions are viewed as a group of disorders or clinical entities characterized by the presence, to a varying degree, of chronic pain, discomfort and disordered function.

While this type of clinical definition corresponds to how patients present to physicians, it has proved difficult to apply in the search for the pathophysiological factor(s) potentially involved in these conditions. While much remains to be done in that regard, it is increasingly accepted that these conditions are multifactorial and that the symptoms experi-
enanced by two individual patients, although somewhat simi-
lar, may result from a number of different etiologies.

While motility abnormalities are identified in many pa-
tients with functional disorders, the majority of patients
have no demonstrable motility dysfunction, and the motor
abnormalities that have been identified vary from study to
study; each abnormality is present in a small and very spe-
cific group of patients who do not represent the majority
of those seen in clinical practice. The concept that these
conditions are primarily motility disorders is also generally
viewed as failing to account for many of the other character-
isitics that are present in these patients, in particular the dis-
comfort and pain often reported in the presence of an
apparently normal gastrointestinal motility.

In many patients with functional disorders, the presence
of an altered visceral perception to various stimuli, such as
distention, has been identified. This issue of altered visceral
perception in IBS is discussed extensively elsewhere, but it is
important to point out that, while a primary disorder of vis-
ceral sensory perception provides a plausible explanation for
many of the symptoms reported in functional bowel disor-
ders, it remains a largely unproved concept at the clinical
level. In specific situations, such as the development of
chronic symptoms following an acute inflammatory or infec-
tious event (such as postinfectious IBS), altered function of
the normal sensory mechanisms, either within the gut wall
or along visceral afferent pathways, is conceivable, at least in
the short term. It is more difficult at this point to implicate
altered visceral sensory pathways in the pathogenesis of
functional symptoms such as diarrhea or increased gas that
are present in many postinfectious IBS patients but also in
patients in whom there is no history of any acute inflamm-
atory or infectious precipitant before the development of
symptoms. Similarly, the hypothesis of an altered visceral
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in functional disorders fails to account for the very high
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ders. In these conditions, it is essential to remember the
homeostatic nature of autonomic function. While many local
gastrointestinal stimuli elicit responses and reflexes whose
involvement is essentially limited to the immediate region
that was primarily stimulated, when more intense or poten-
tially noxious stimuli occur, other systems including the
central nervous system, in addition to the gastro-
intestinal tract, are involved. The transmission of the
information to the central nervous system allows for the
elaboration of an integrated homeostatic response that
may include a behavioural as well as a physiological response.
In this type of response, both visceral and autonomic
nervous pathways are essential.

Many of the systemic responses elicited by visceral stimuli
are produced through autonomic reflexes. These reflexes,
which occur primarily through the brainstem, may not be
associated with a conscious perception of the sensory stimulus.
However, other visceral sensory stimuli are sufficiently in-
tense to be felt as well.

The type and degree of the autonomic reflex response to a
specified visceral stimulus depend on the location, type and
intensity of the stimulus. If prior sensitization of the visceral
afferent pathways has resulted in a state of hyperalgesia or
allodynia, the reflex response may be exaggerated, while a
peripheral sensory neuropathy may be associated with a de-
creased reflex response. Similarly, psychological factors such
as increased vigilance, anxiety and depression are also likely
to affect autonomic reflexes (1).

ASSESSING AUTONOMIC FUNCTION
A number of methods have been developed to assess specific
aspects of autonomic nervous function. Some of the older
approaches, which were very cumbersome and involved
rather complex measurements, have been largely aban-
doned. In recent years, the development of techniques based
on the autonomic modulation of heart rate function have
largely replaced other methods because of their simplicity and validity as markers of vagal as well as sympathetic function. The use of techniques such as power spectral analysis of heart rate variability provides a simple and very accurate measure of the respective outflow of the vagal and sympathetic branches of the autonomic nervous system (2). Because the method requires only a surface electrocardiogram, it can be done in almost any setting.

**AUTONOMIC FUNCTION AND BOWEL DISORDERS**

In recent years, there have been a number of reports suggesting that functional bowel disorders are associated with autonomic disturbances (3). However, none of these studies establishes anything more than an association, and their results should not be interpreted to indicate that altered autonomic function is causally related with functional disorders. In animals, surgical ablation of celiac or mesenteric ganglia has profound effects on gastrointestinal function (4). Furthermore, tumour invasion of the sympathetic ganglia can result in intestinal and colonic pseudo-obstruction in patients with metastatic cancer (5). Conditions affecting autonomic function, such as Parkinson’s disease and autonomic neuropathies, also have a profound effect on gut function as do the degenerative neuropathies associated with diabetes and amyloidosis (6). However, these alterations are clearly not the direct cause of symptoms in the majority of patients with functional symptoms.

Altered autonomic function may also be involved in conditions such as gastroesophageal reflux disease and neuroopathic upper gastrointestinal motility disorders (7,8). More interestingly, such vagal abnormalities have also been identified in patients with functional gut disorders including functional dyspepsia and colonic inertia (9,10).

Recently Aggarwal et al (11) convincingly showed that a subgroup of patients with IBS had various autonomic abnormalities. However, the findings were varied and certainly were not present in all patients from their cohort. Patients with vagal dysfunction tended to have constipation, whereas diarrhea-prone patients primarily had increased sympathetic activity. Another group has shown that functional abdominal pain without any motility abnormalities was associated with an increased basal parasympathetic activity and a lower sympathetic activity (12). In a patient with noncardiac chest pain, the opposite effect was found; patients with increased visceral sensitivity to esophageal acid infusion had a higher resting sympathetic tone and a decreased vagal activity, suggesting that the abnormalities may differ according to the gut region involved (foregut versus hindgut) (13).

However, it is difficult from these associations to ascribe a definite causative role to any type of autonomic dysfunction in functional disorders until studies aimed at restoring a more normal basal autonomic balance are shown to change the altered visceral perception and function that is present in these patients. Central neural and emotional factors, which are well known to be associated with functional gut symptoms, are also capable of altering autonomic balance. It would be naive not to consider that, in a large number of cases where central neural as well as autonomic factors are involved, the effects of the former lead to the occurrence of the latter and of gastrointestinal symptoms.

**CONCLUSIONS**

While there is increasing agreement that autonomic abnormalities are often associated with functional disorders of the gut, until we gain a better understanding of the mechanisms responsible for the symptoms, and of their fate with restoration of autonomic function, the exact role of altered autonomic function in the pathogenesis of functional disorders will remain as poorly defined as that of the other putative mechanisms involved in these disorders. Once more, the need for studies focusing on the mechanisms of disease rather than its clinical manifestation, and on pathophysiology rather than symptomatology, is apparent.

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**REFERENCES**
