Dyspepsia is a common symptom. Dyspeptic symptoms may be caused by a variety of conditions such as peptic ulcer disease, gastro-oesophageal reflux, and malignancy. Most often, however, no cause is identified and dyspepsia is deemed to be functional. While symptom severity does influence frequency of consultation, dyspeptic consultants also differ from non-consultants with respect to symptom perception and anxiety. This highlights the importance of understanding the patient’s agenda early in the course of evaluation. Patients over the age of 55 years or with alarm symptoms should be referred for prompt endoscopy. In the absence of other clinically apparent aetiologies, uninvestigated dyspeptics can be either tested and treated for *Helicobacter pylori* or empirically treated with proton pump inhibitors. Uninvestigated dyspeptics failing empiric therapy should be referred for evaluation that includes endoscopy. Further therapy with prokinetics, tricyclic antidepressants, fundal relaxants, antidepressants, or psychotherapy is guided by predominant symptoms and assessment of possible psychiatric factors.

**DEFINING THE PROBLEM**

Defining dyspepsia is like describing a close relative—easily recognised but difficult to describe. A variety of criteria exist. Most define dyspepsia as symptoms arising from the upper digestive tract unrelated to colonic function. Often symptoms are brought on or worsened by eating. The Rome II consensus report, a recent widely cited effort symptomatically categorising functional digestive disorders, defines dyspepsia as pain or discomfort centred in the upper abdomen.4 Discomfort specifically includes early satiety, fullness, upper abdominal bloating, and nausea. “Functional” dyspepsia (synonymous with “non-ulcer” dyspepsia) is defined by these same authors as 12 weeks in the past 12 months of persistent or recurrent pain or discomfort in the upper abdomen in the absence of organic disease and no association with bowel habit.

Functional dyspepsia may be further categorised by predominant symptoms into ulcer-like, dysmotility-like, and unspecified dyspepsia. Ulcer-like dyspepsia has pain as the predominant symptom while dysmotility-like dyspepsia has predominantly abdominal discomfort. A previous category of reflux-like dyspepsia is no longer included since most of these patients actually have reflux disease and should be managed as such. It is worth noting that this classification scheme was primarily developed as an aid in the design and performance of clinical trials. Its clinical utility beyond taxonomy remains unproven.

Defining dyspepsia allows for more accurate study of the problem and the problem is considerable. Studies from the United States, Great Britain, and other parts of the world have shown the prevalence of dyspepsia to be between 26% and 41%.2 While only 20%-25% of these individuals seek medical care, dyspepsia accounts for 2%-5% of all consultations in primary care.4 For gastroenterologists, dyspepsia accounts for between 20% and 40% of consultations.5 It appears that as primary care physicians have grown more comfortable with proton pump inhibitors and *Helicobacter pylori* eradication, the percentage of attendees in gastroenterology clinics with functional dyspepsia is steadily increasing.

The burden of illness with respect to quality of life and economic consequences of dyspepsia is considerable. Recent data from a large cross sectional survey in the UK suggest dyspepsia may be costing society approximately £1 billion ($1.46 billion) annually.6 Similar estimates exist for the costs of diagnosis and management of dyspepsia in the United States.7 A Swedish study estimated direct costs of dyspepsia to be approximately £26 million annually for 8 million people.8 When indirect costs were included, total costs increased almost 10-fold. This was largely attributable to the average of 26 (!) more days of lost productivity by dyspeptics. Indirect health costs are paralleled by decreased quality of life, which can be profound.9,10 Figure 1 shows Psychological General Well Being index scores for healthy controls, patients with functional dyspepsia, and gas-troparetics seen in our clinic. Clearly, the burden of dyspepsia is considerable from economic, societal, and personal vantage points.

**EARLY INVESTIGATION AND MANAGEMENT OF THE “UNINVESTIGATED DYSPEPTIC”**

Dyspepsia is a symptom and not a diagnosis. The differential diagnosis of dyspepsia is shown in...
underlying gastro-oesophageal reflux disease or peptic ulcer disease are better tolerated and effectively treated by antisecretory or prokinetic agents. Antisecretory agents are usually the first-line therapy for patients with dyspeptic symptoms. However, a significant proportion of patients with dyspepsia do not respond to these agents, and it is essential to rule out other causes of dyspepsia before starting treatment. Patients with dyspeptic symptoms who are not candidates for endoscopy can be managed empirically. If the background prevalence of H pylori and ulcer disease is high, a “test and treat” approach is reasonable. H pylori-negative patients or those not responding to eradication therapy can be given a trial of proton pump inhibitors. If there is a clinical response to either acid suppressive therapy or H pylori eradication, patients can be managed intermittently for recurrent symptoms. For patients who require additional reassurance, fail empiric therapy, or require chronic treatment, referral for further investigation including upper gastrointestinal endoscopy is indicated.
Evaluation and treatment of dyspepsia

**Box 2: Potential causes of non-ulcer dyspepsia**

- Duodenogastic reflux.
- Duodenitis.
- Carbohydrate malabsorption (lactose, fructose, sorbitol).
- Cholelithiasis or choledocholithiasis.
- Chronic pancreatitis.
- Systemic disorders (diabetes, thyroid, parathyroid, hypothalamic, connective tissue disease).
- Intestinal parasites.
- Psychiatric disorders.
- Visceral hypersensitivity.
- Gastric/small intestinal dysmotility.
- Gallbladder/biliary dysmotility.

**INVESTIGATION OF DYSPEPSIA AND NON-ULCER DYSPEPSIA**

Eradication of *H pylori* and use of acid suppressive therapy will benefit those patients with dyspepsia attributable to peptic ulcer and reflux disease. There additionally appears to be some benefit of acid suppression in true functional dyspepsia. Studies of functional dyspepsia that have aggressively excluded ulcer and reflux disease have tended to show little benefit of commonly employed tests of gastric function identify therapies that reliably improve symptoms.

Much recent attention has focused on the concept that patients with functional dyspepsia have augmented perception of visceral pain or visceral hypersensitivity. Many dyspepsias will report pain at levels of balloon distension in the stomach or proximal intestine that are not perceived as adverse by controls. These observations should be interpreted cautiously. Many of these studies have used protocols prone to response bias. Borrowing from studies of visceral hypersensitivity in irritable bowel syndrome, studies using less bias-prone methods tend to not demonstrate visceral hypersensitivity. This raises the possibility that much of the coexistence of psychiatric disturbances and dyspeptic consultations is attributable to sepsia and reflux-like dyspepsia. The third double blind, placebo control trial of omeprazole 20 mg daily in 197 patients with functional dyspepsia showed omeprazole to be superior to placebo in providing complete symptom relief after two weeks.

For patients unresponsive to acid suppressive therapy or *H pylori* eradication, mechanisms of symptom generation are largely speculative. This means therapeutic interventions are largely speculative. A variety of potential causes have been proposed with varying degrees of support (box 2). While patients often complain of excess acid, there is no evidence for abnormal gastric acid secretion. The role of *H pylori* has already been discussed. It should be kept in mind that “gastritis” is neither an endoscopic diagnosis nor a cause of recognised cause of dyspepsia.

Three aetiologies deserve particular attention: dysmotility, visceral hypersensitivity, and psychiatric disorders. Abnormalities of gastric neuromuscular function can be detected by scintigraphic gastric emptying studies, electrogastrography, or antroduodenal manometry in between 30% and 60% of patients. In addition to impaired motor function, a subset of dyspeptics has impaired postprandial relaxation of the proximal stomach. Some investigators have suggested that certain symptoms are associated with altered gastric physiology. Predictors of delayed gastric emptying include female sex, excessive postprandial fullness, and severe vomiting. Impaired postprandial relaxation of the proximal stomach has been associated with early satiety. The acute administration of the interstitial serotonin receptor (5-HT3) agonist, bupropion, has been shown to improve accommodation and tolerance to balloon distension of the proximal stomach. While these observations are encouraging, most studies have failed to demonstrate a relationship between disturbed gastrointestinal motor function and symptoms. In particular, there is little evidence that abnormalities of commonly employed tests of gastric function identify therapies that reliably improve symptoms.

The coexistence of psychiatric disturbances and dyspeptic symptoms is well documented. Importantly, it appears that dyspeptic consulters do not differ from non-consulters with respect to objective symptoms, but they tend to perceive their symptoms as more severe and have greater associated anxiety. The implication is clear—in managing functional dyspepsia, answers are less likely to be found by taking an ever more microscopic view of the digestive tract and more likely to be found by taking a more macroscopic view of the entire patient. Patients with psychiatric distress have a high prevalence of digestive symptoms. Conversely, patients with longstanding unexplained digestive symptoms are vulnerable to the development of reactive psychiatric disorders. Anxiety, depression, personality disorders, and a history of physical or sexual abuse are all seen with increased frequency in this population. Understanding these issues is critical to managing patients with functional disorders. The importance of addressing patient concerns and exploring the psychosocial context of symptoms cannot be overstated, particularly in...
patients refractory to standard therapies. Frank discussions in an empathic manner or use of self-administered questionnaires such as the Beck Depression Index, Beck Anxiety Index, and Symptom Check List-90 can provide objective documentation that may help further explore these areas.

TREATMENT OF NON-ULCER DYSPEPSIA

Therapy in functional dyspepsia is symptom driven. There are few data to suggest that studies of gastric neuromuscular function allow implementation of therapies that reliably improve symptoms. Patients with functional dyspepsia who have predominant symptoms of upper abdominal pain (ulcer-like) can be initially treated with proton pump inhibitors. Patients with unexplained upper abdominal pain who have failed proton pump inhibitors may be treated with tricyclic antidepressants, although good supporting data are lacking.

Those with dysmotility-like symptoms can be treated initially with either acid suppressive therapy, prokinetic agents, or 5-HT3, agonists. Both metoclopramide and domperidone have been shown to be superior to placebo in the treatment of functional dyspepsia. References 1-5 Available literature suggests 5-HT3 agonists may be efficacious in patients with impaired accommodation. Since formal assessment of accommodation is not widely available, it is reasonable to use these agents in patients with excessive early satiety. The lack of supporting data and the adverse reactions associated with sumatriptan argue against the use of this agent. Buspirone has comparable effects, fewer adverse events, and may provide additional anxiolytic benefits in a non-confrontational way.

For all patients, the psychosocial context of symptoms should be assessed. Patients with a history of psychiatric distress, multiple unexplained physical symptoms, or symptoms refractory to standard therapies should be evaluated for concomitant psychopathology. If identified, appropriate therapy is offered. At present there are no data to support the use of selective serotonin reuptake inhibitors in functional dyspepsia in the absence of disorders for which these medications are otherwise indicated.

SUMMARY

Dyspepsia is a common symptom and is most often functional. Importantly, dyspeptic consulters differ from non-consulters more in terms of symptom perception and anxiety than objective symptom measures. This highlights the importance of understanding the patient’s agenda early in the course of evaluation. Patients over the age of 55 years or with alarm symptoms should be referred for prompt endoscopy. In the absence of other clinically apparent actiology, uninvestigated dyspepsias can be either tested and treated for H pylori or empirically treated with proton pump inhibitors.

Uninvestigated dyspepsias failing empiric therapy should be referred for evaluation that includes endoscopy. Further therapy with prokinetics, tricyclic antidepressants, fundal relaxants, antidepressants, or psychotherapy is guided by predominant symptoms and assessment of possible psychiatric factors.
Perichondritis: a complication of piercing auricular cartilage

A 20 year old woman presented to the ear, nose, and throat clinic with auricular perichondritis two days after piercing the helix of her left ear with the aid of a piercing gun. Two thirds of the upper part of her auricle was swollen, red, and tender. The lobule (which does not contain cartilage) remained intact, which indicated that the infection was perichondritis and not simply cellulitis.

The use of guns for piercing cartilage presents an additional risk of perichondritis. The gun applies shear forces to the perichondrium, which may slip off the cartilage. An avascular cartilage (which is normally nourished by the perichondrium), may then become necrotic. Abscess formation and loss of cartilage are potential complications that often require surgical intervention. Despite timely treatment, including antibiotic therapy, drainage, and debridement, an unsightly deformity (“cauliflower ear”) may result.

The treatment of choice for auricular perichondritis is fluoroquinoline antibiotics, such as ciprofloxacin. Although multiple species of Staphylococcus and Pseudomonas aeruginosa are the most common bacteria, they also penetrate well into the cartilage. The treatment of choice for auricular perichondritis is fluoroquinoline antibiotics, such as ciprofloxacin. Since they show good antipseudomonal activity in addition to their effect against staphylococci, they also penetrate well into the cartilage. The treatment of choice for auricular perichondritis is fluoroquinoline antibiotics, such as ciprofloxacin, since they show good antipseudomonal activity in addition to their effect against staphylococci. They also penetrate well into the cartilage. However, their use is limited to patients who are more than 18 years old because of their potential damage to young developing cartilage.

Although our treatment was successful, this case demonstrates the potential hazards of piercing cartilage, mainly in the ear and nose. Perichondritis can end with a very unsightly ear or nose, which may eventually turn out to be beyond repair. Physicians as well as the clientele of body piercers should be aware of this risk.

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