they point is poorly correlated with the actual level of obstruction. Dysphagia may result from a mechanical obstruction of the esophagus, from inflammation of the esophageal mucosa, or from an abnormality of esophageal motility.

Odynophagia, or pain on swallowing, needs to be differentiated from dysphagia in the patient history because it can be an important clue to the cause of the swallowing disorder. Painful swallowing is most often associated with infectious esophagitis or pill-induced esophageal ulcers but is only rarely present in acid-mediated esophageal disease.

Chest pain may also be a sign of esophageal disease, most often caused by gastroesophageal reflux or esophageal dysmotility. Unfortunately for the clinician, the symptoms of cardiac and esophageal chest pain overlap because of the shared neural pathways mediating pain sensation to these organs. Typical features of angina may occur in reflux-induced chest pain, including radiation to the neck and jaw; relief with nitrates, which modulate esophageal motility; and onset of symptoms with exertion. Chest pain that awakens a patient from sleep, however, is uncommon in true cardiac disease and suggests an esophageal disorder, as does pain that is relieved with antacids or pain that lasts for several hours without associated symptoms. Esophageal chest pain is often thought to occur in response to esophageal spasm, but data suggest that gastroesophageal reflux is responsible for most cases.

## GASTROESOPHAGEAL REFLUX DISEASE

Gastroesophageal reflux disease (GERD) is the most common disorder of the esophagus. It causes occasional heartburn in almost one half of the population and daily symptoms in almost 7% of adult Americans. GERD is responsible for about $10 billion to $12 billion in direct medication costs per year, and related acid antisecretory therapies are among the most commonly prescribed drugs in the United States.

Gastroesophageal reflux is defined as the reflux of gastric contents back into the esophagus. There are three main underlying mechanisms that can result in gastroesophageal reflux. First, in both healthy subjects and those with GERD, reflux of gastric contents occurs primarily in the postprandial period (physiologic reflux) due to transient relaxation of the LES. Two additional mechanisms of gastroesophageal reflux are low baseline LES pressures and stress reflux. The latter indicates an inability to mount sufficient LES pressures to prevent reflux in response to sudden increases in intra-abdominal pressure. Other pathophysiologic mechanisms also bear a role in GERD, including esophageal dysmotility, delayed gastric emptying, duodenogastroesophageal reflux, impaired local defense mechanisms, and decreases in salivation.

Hiatal hernia is another important factor in the pathogenesis of GERD. The size of the hernia tends to correlate with the severity of the reflux. The presence of a nonreducible hiatal hernia disrupts the sphincter mechanism and prolongs esophageal clearance, leading to increased duration of esophageal acid exposure.

Patients with impaired esophageal motility have more severe reflux, slower acid clearance, worse mucosal injury, and more frequent extraesophageal manifestation of GERD. The importance of esophageal dysmotility is best illustrated by scleroderma, in which a hypotensive LES and impaired or absent esophageal peristalsis contribute to reflux that is often severe. Further, many patients with scleroderma have an associated sicca syndrome and therefore have reduced acid-neutralizing capacity as a result of the absence of saliva.

Heartburn is the cardinal clinical feature of GERD, and when it is present, the diagnosis of GERD is made easily. Complaints of bitter regurgitation or water brash add to the diagnostic accuracy, but these features are not always present. In some cases, atypical symptoms dominate in patients with no history of heartburn. Most cases of noncardiac chest pain, which can mimic angina, are thought to be caused by GERD. A significant number of additional symptoms, including chronic cough, asthma, hoarseness, chronic sore throat, and globus sensation, may also result from occult gastroesophageal reflux

### Diagnosis

When heartburn is the predominant or sole symptom, gastroesophageal reflux is the cause in at least 75% of individuals, indicating that the presence of heartburn is specific for the diagnosis of GERD. Other GERD-related symptoms include acid regurgitation, belching, water brash, dysphagia, odynophagia, chest pain, globus sensation, chronic cough, hoarseness, and asthma.

A presumptive diagnosis of GERD based on symptoms commonly leads to a trial of empiric antireflux treatment without further diagnostic testing. In first-time health care seekers, more detailed diagnostic evaluation is definitely indicated if alarm symptoms are present (i.e., dysphagia, odynophagia, anorexia, weight loss, or evidence of upper gastrointestinal bleed). Presently, most physicians accept the concept that a marked symptomatic response to antireflux treatment is highly suggestive of GERD as the underlying cause of the patient's symptoms.

Endoscopy has a low sensitivity for diagnosis of GERD, because 50% to 70% of patients with GERD-related symptoms have no evidence of esophageal mucosal injury on endoscopy. However, upper endoscopy remains the "gold standard" for diagnosis of erosive esophagitis, Barrett's esophagus (BE) (E-Fig. 35-1), and other important complications of GERD, such as ulceration and stricture. The test is indicated in patients with alarm symptoms and to exclude BE in those with long-standing symptoms of GERD. Endoscopy is also warranted if there is diagnostic uncertainty (e.g., persistence of symptoms on appropriate empiric therapy).

Presently, pH monitoring is best reserved for patients who are considered to be candidates for antireflux surgery; those who report relapse of GERD symptoms after antireflux surgery; and those with atypical or extra-esophageal symptoms that are poorly responsive to an adequate trial of antireflux therapy. Measurement of esophageal impedance and pH has a role in identifying nonacidic reflux as the case of residual GERD-related symptoms in patients being treated with proton pump inhibitors (PPIs) at least twice daily.

### Treatment

For patients with mild or infrequent symptoms, it is reasonable to use antacids and lifestyle modification such as weight loss, cessation of smoking, and elevation of the head of the bed. For patients with more severe reflux symptoms, a definitive treatment is required. Treatment of GERD is summarized in Table 35-1.