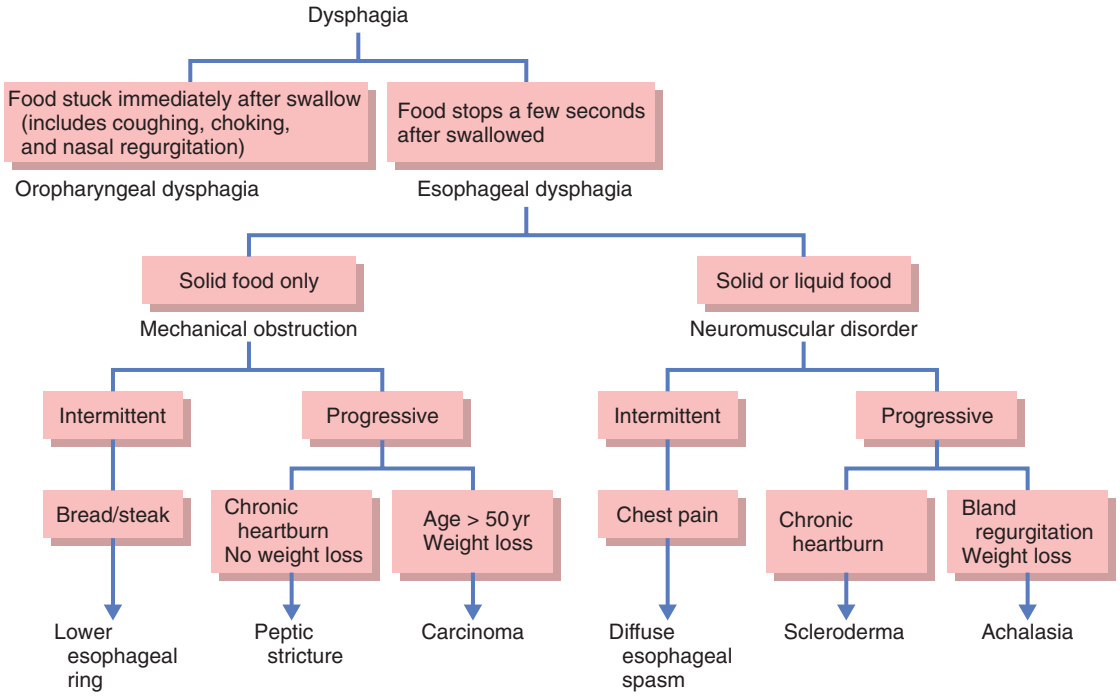

FIGURE 35-1 Algorithm for the differential diagnosis of dysphagia.

the airway, requires a fine coordination of events happening at a very rapid rate. Disruption of this phase of swallowing may be caused by structural defects or, more commonly, by neuromuscular dysfunction. Structural abnormalities that may be encountered in the hypopharynx include cervical osteophytes, hypopharyngeal diverticulum (Zenker's diverticulum), head and neck tumors, radiation injury, and postcricoid webs. In these settings, patients may have difficulty when a solid bolus leaves the mouth and enters the tubular esophagus.

Much more commonly, symptoms of transfer dysphagia result from neuromuscular injury causing disruption of this finely coordinated act of swallowing. In these situations, problems are much more commonly associated with attempts to swallow liquids. Sensory or motor injury may lead to an inability to accomplish the transfer of a bolus from the mouth to the esophagus. Stroke (particularly involving the brain stem) is one of the most common causes of oropharyngeal dysphagia. This scenario is associated with a higher patient mortality rate because of a higher risk of aspiration pneumonia and dehydration.

Essentially, any disease process that affects the brain can result in dysphagia. The more common associations are amyotrophic lateral sclerosis, Parkinson's disease, and brain tumors. Primary muscular diseases may also result in oropharyngeal dysphagia. These include oculopharyngeal muscular dystrophy, myotonic dystrophy, myasthenia gravis, and tardive dyskinesia. Patients with oropharyngeal dysphagia commonly complain that food gets "stuck" immediately upon swallowing; this sensation may be associated with choking, coughing, or nasal regurgitation.

Esophageal Dysphagia

Once the food bolus enters the esophagus, passage may be impeded by structural abnormalities or alterations in esophageal motility. These alterations range from congenital abnormalities to acquired conditions, and they constitute a rather large differential diagnosis. Solid food dysphagia alone is more commonly seen in patients with mechanical obstruction, whereas solid and liquid dysphagia is typically encountered in esophageal motility disorders such as achalasia. Patients with esophageal dysphagia usually report the onset of symptoms several seconds after initiation of a swallow.

ESOPHAGEAL MOTILITY DISORDERS

The more common disorders of esophageal motility are achalasia, diffuse esophageal spasm, and scleroderma, and their key features are outlined in Table 35-2.

Primary Motility Disorders

The term *primary motility disorders* refers to a number of conditions in which there is some disruption in the neuromuscular control of esophageal peristalsis.

Achalasia

The prototype of esophageal dysmotility is achalasia. This condition, characterized by loss of esophageal peristalsis and failure of LES relaxation, is the most widely recognized primary motility disorder. A variety of changes have been described in patients with this condition: loss of ganglion cells from the myenteric plexus; degenerative changes in the vagus nerve; degenerative changes in the dorsal motor nucleus of the vagus, including occasional evidence of intracytoplasmic inclusions (Lewy bodies); loss of small intramuscular nerve fibers; and reduction in vesicles of small nerve fibers. The initial site of injury is unknown, and 98% of cases are idiopathic. Achalasia may occur at any age from childhood to late adulthood. The peak occurrence is between the ages of 30 and 60 years.

The hallmark symptom of achalasia is dysphagia, typically for both solids and liquids. Other commonly noted symptoms include regurgitation, chest pain, and what patients describe as "heartburn." Symptoms typically have been present for years before diagnosis, and this may especially be true if patients are