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■ ■FURTHER READING Bunsawat K et al: Exercise intolerance in heart failure with preserved ejection fraction: Causes, consequences and the journey toward a cure. Exp Physiol 109:502, 2023. Edwards JA et al: Characteristics and treatment of exercise intolerance in patients with long COVID. J Cardiopulm Rehabil Prev 43:400, 2023. Houstis NE et al: Exercise intolerance in heart failure with preserved ejection fraction. Circulation 137:148, 2018. McCoy J et al: Pathophysiology of exercise intolerance in chronic dis eases: The role of diminished cardiac performance in mitochondrial and heart failure patients. Open Heart 4:000632, 2017. Ramirez MF et al: Obesity-related biomarkers are associated with exercise intolerance in HFpEF. Circ Heart Fail 16:010618, 2023. Shah RV et al: Metabolic cost of exercise initiation in patients with heart failure with preserved ejection fraction vs community dwelling adults. JAMA Cardiol 6:653, 2021. Section 6 Alterations in Gastrointestinal Function Ikuo Hirano*, Peter J. Kahrilas

Dysphagia Dysphagia—difficulty with swallowing—refers to problems with the transit of food or liquid from the mouth to the hypopharynx or through the esophagus. Severe dysphagia can compromise nutrition, cause aspiration, and reduce quality of life. Additional terminology pertaining to swallowing dysfunction is as follows. Aphagia (inability to swallow) typically denotes complete esophageal obstruction, most commonly encountered in the acute setting of a food bolus or foreign body impaction. Odynophagia refers to painful swallowing, typically resulting from mucosal ulceration within the oropharynx or esopha gus. It commonly is accompanied by dysphagia, but the converse is not true. Globus pharyngeus is a foreign body sensation localized in the neck that does not interfere with swallowing and sometimes is relieved by swallowing. Transfer dysphagia frequently results in nasal regurgita tion or pulmonary aspiration during swallowing and is characteristic of oropharyngeal dysphagia. Phagophobia (fear of swallowing) and refusal to swallow may be psychogenic or related to anticipatory anxiety about food bolus obstruction, odynophagia, or aspiration. ■ ■PHYSIOLOGY OF SWALLOWING Swallowing begins with a voluntary (oral) phase that includes prepara tion during which food is masticated and mixed with saliva. This is followed by a transfer phase during which the bolus is pushed into the pharynx by the tongue. Bolus entry into the hypopharynx initiates the pharyngeal swallow response, which is centrally mediated and involves a complex series of actions, the net result of which is to propel food through the pharynx into the esophagus while preventing its entry into the airway. To accomplish this, the larynx is elevated and pulled forward, actions that also facilitate upper esophageal sphincter (UES) opening. Tongue pulsion then propels the bolus through the UES, fol lowed by a peristaltic contraction that clears residue from the pharynx and through the esophagus. The lower esophageal sphincter (LES) relaxes as the food enters the esophagus and remains relaxed until the peristaltic contraction has delivered the bolus into the stomach. Peri staltic contractions elicited in response to a swallow are called primary peristalsis and involve sequenced inhibition followed by contraction of the musculature along the entire length of the esophagus. The ■ ■PATHOPHYSIOLOGY OF DYSPHAGIA Dysphagia can be subclassified both by location and by the circum stances in which it occurs. With respect to location, distinct consid erations apply to oral, pharyngeal, or esophageal dysphagia. Normal transport of an ingested bolus depends on the consistency and size of the bolus, the caliber of the lumen, the integrity of peristaltic contrac tion, and deglutitive inhibition of both the UES and the LES. Dyspha gia caused by an oversized bolus or a narrow lumen is called structural dysphagia, whereas dysphagia due to abnormalities of peristalsis or impaired sphincter relaxation after swallowing is called propulsive or motor dysphagia. More than one mechanism may be operative in a patient with dysphagia. Scleroderma commonly presents with absent peristalsis as well as a weakened LES that predisposes patients to peptic stricture formation. Likewise, radiation therapy for head and neck can cer may compound the functional deficits in the oropharyngeal swal low attributable to the tumor and cause cervical esophageal stenosis. It is worth noting that in addition to bolus transit, symptom reporting of dysphagia is dependent upon intact sensory innervation and cen tral nervous system perception. Chronicity of motor dysfunction in *Deceased.

inhibition that precedes the peristaltic contraction is called degluti tive inhibition. Local distention of the esophagus anywhere along its length, as may occur with gastroesophageal reflux, activates secondary peristalsis that begins at the point of distention and proceeds distally. Tertiary esophageal contractions are nonperistaltic, disordered esopha geal contractions that may be observed to occur spontaneously during fluoroscopic observation.

The musculature of the oral cavity, pharynx, UES, and cervical esophagus is striated and directly innervated by lower motor neurons carried in cranial nerves (Fig. 47-1). Oral cavity muscles are innervated by the fifth (trigeminal) and seventh (facial) cranial nerves; the tongue, by the twelfth (hypoglossal) cranial nerve. Pharyngeal muscles are innervated by the ninth (glossopharyngeal) and tenth (vagus) cranial nerves. Dysphagia CHAPTER 47 Physiologically, the UES consists of the cricopharyngeus muscle, the adjacent inferior pharyngeal constrictor, and the proximal portion of the cervical esophagus. UES innervation is derived from the vagus nerve, whereas the innervation to the musculature acting on the UES to facilitate its opening during swallowing comes from the fifth, sev enth, and twelfth cranial nerves. The UES remains closed at rest owing to both its inherent elastic properties and neurogenically mediated contraction of the cricopharyngeus muscle. UES opening during swal lowing involves both cessation of vagal excitation to the cricopharyn geus and simultaneous contraction of the suprahyoid and geniohyoid muscles that pull open the UES in conjunction with the upward and forward displacement of the larynx. The neuromuscular apparatus for peristalsis is distinct in proximal and distal parts of the esophagus. The cervical esophagus, like the pharyngeal musculature, consists of striated muscle and is directly innervated by lower motor neurons of the vagus nerve. Peristalsis in the proximal esophagus is governed by the sequential activation of the vagal motor neurons in the nucleus ambiguus. In contrast, the distal esophagus and LES are composed of smooth muscle and are controlled by excitatory and inhibitory neurons within the esophageal myenteric plexus. Medullary preganglionic neurons from the dorsal motor nucleus of the vagus trigger peristalsis via these ganglionic neu rons during primary peristalsis. Neurotransmitters of the excitatory ganglionic neurons are acetylcholine and substance P; those of the inhibitory neurons are vasoactive intestinal peptide and nitric oxide. Peristalsis results from the patterned activation of inhibitory followed by excitatory ganglionic neurons, with progressive dominance of the inhibitory neurons distally. Similarly, LES relaxation occurs with the onset of deglutitive inhibition and persists until the peristaltic sequence is complete. At rest, the LES is contracted because of excitatory gan glionic stimulation and its intrinsic myogenic tone, a property that distinguishes it from the adjacent esophagus. The function of the LES is supplemented by the surrounding muscle of the right diaphragmatic crus, which acts as an external sphincter during inspiration, cough, or abdominal straining.

Sagittal view of the pharynx Musculature of the pharynx Hard palate Soft palate Oral pharynx Oral cavity Valeculae PART 2 Cardinal Manifestations and Presentation of Diseases Epiglottis Tongue Laryngeal pharynx (hypopharynx) Mylohyoid ms Hyoid bone Hyoid bone Esophagus Thyrohyoid membrane Vocal cord Transverse arytenoid ms. Cricothyroid membrane Cricoid cartilage FIGURE 47-1 Sagittal and diagrammatic views of the musculature involved in enacting oropharyngeal swallowing. Note the dominance of the tongue in the sagittal view and the intimate relationship between the entrance to the larynx (airway) and the esophagus. In the resting configuration illustrated, the esophageal inlet is closed. This is transiently reconfigured such that the esophageal inlet is open and the laryngeal inlet closed during swallowing. (Adapted from PJ Kahrilas, in DW Gelfand and JE Richter [eds]: Dysphagia: Diagnosis and Treatment. New York, Igaku-Shoin Medical Publishers, 1989, pp. 11–28.) combination with defects in peripheral sensory perception may reduce patient-reported dysphagia in patients with achalasia. Oral and Pharyngeal (Oropharyngeal) Dysphagia Oral-phase dysphagia is associated with poor bolus formation and control so that food has prolonged retention within the oral cavity and may seep out of the mouth. Drooling and difficulty in initiating swallowing are charac teristic signs. Poor bolus control also may lead to premature spillage of food into the hypopharynx with resultant aspiration into the trachea, evident as swallow-induced cough or regurgitation into the nasal cavity. Pharyngeal-phase dysphagia is associated with retention of food in the pharynx due to poor tongue or pharyngeal propulsion or obstruction at the UES. Signs and symptoms of concomitant hoarseness or cranial nerve dysfunction may be associated with oropharyngeal dysphagia that is associated with neurologic disorders. Oropharyngeal dysphagia may be due to neurologic, muscular, structural, iatrogenic, infectious, and metabolic causes. Iatrogenic, neurologic, and structural pathologies are most common. Iatrogenic causes include surgery and radiation, often in the setting of head and neck cancer. Neurogenic dysphagia resulting from cerebrovascular accidents, Parkinson’s disease, and amyotrophic lateral sclerosis is a major source of morbidity related to aspiration and malnutrition. Medullary nuclei directly innervate the oropharynx. Lateralization of pharyngeal dysphagia implies either a structural pharyngeal lesion or a neurologic process that selectively targets the ipsilateral brainstem nuclei or cranial nerve. Advances in functional brain imaging have elucidated an important role of the cerebral cortex in swallow func tion and dysphagia. Asymmetry in the cortical representation of the pharynx provides an explanation for the dysphagia that occurs as a consequence of unilateral cortical cerebrovascular accidents. Oropharyngeal structural lesions causing dysphagia include Zenk er’s diverticulum, cricopharyngeal bar, and neoplasia. Zenker’s diver ticulum typically is encountered in elderly patients. In addition to dysphagia, patients may present with regurgitation of particulate food debris, aspiration, and halitosis. The pathogenesis is related to stenosis of the cricopharyngeus that causes diminished opening of the UES and results in increased hypopharyngeal pressure during swallowing with development of a pulsion diverticulum immediately above the cricopharyngeus in a region of potential weakness known as Killian’s dehiscence. A cricopharyngeal bar, appearing as a prominent

Lateral pterygoid plate Superior constrictor Stylohyoid process Digastric (post. belly) Stylohyoid ligament Buccinator Stylopharyngeus
Mylohyoid Glossopharyngeus
Styloglossus Digastric (ant. belly) Middle constrictor Hyoglossus Thyrohyoid membrane Inferior constrictor Thyroid cartilage Cricopharyngeus Cricothyroid membrane Esophagus Cricoid cartilage indentation behind the lower third of the cricoid cartilage, is related to Zenker's diverticulum in that it involves limited distensibility of the cricopharyngeus and can lead to the formation of a Zenker's diver ticulum. However, a cricopharyngeal bar is a common radiographic finding, and most patients with transient cricopharyngeal bars are asymptomatic, making it important to rule out alternative etiologies of dysphagia before treatment. Furthermore, cricopharyngeal bars may be secondary to other neuromuscular disorders that impair opening of the UES. Since the pharyngeal phase of swallowing occurs in less than a second, rapid-sequence fluoroscopy is necessary to evaluate functional abnormalities. Adequate fluoroscopic examination requires that the patient be conscious and cooperative. The study incorporates record ings of swallow sequences during ingestion of food and liquids of vary ing consistencies. The pharynx is examined to detect bolus retention, regurgitation into the nose, or aspiration into the trachea. Timing and integrity of pharyngeal contraction and opening of the UES with a swallow are analyzed to assess both aspiration risk and the potential for swallow therapy. Structural abnormalities of the oropharynx, espe cially those that may require biopsies, also should be assessed by direct laryngoscopic examination. Esophageal Dysphagia The adult esophagus measures 18–26 cm in length and is anatomically divided into the cervical esophagus, extend ing from the pharyngoesophageal junction to the suprasternal notch, and the thoracic esophagus, which continues to the diaphragmatic hiatus. When distended, the esophageal lumen has internal dimensions of about 2 cm in the anteroposterior plane and 3 cm in the lateral plane. Solid food dysphagia becomes common when the lumen is narrowed to <13 mm, but also can occur with larger diameters in the setting of poorly masticated food or motor dysfunction. Circumferential lesions are more likely to cause dysphagia than are lesions that involve only a partial circumference of the esophageal wall. The most common struc tural causes of dysphagia are Schatzki's rings, eosinophilic esophagitis, and peptic strictures. Dysphagia also occurs in the setting of gastro esophageal reflux disease without a stricture, perhaps on the basis of altered esophageal sensation, reduced esophageal mural distensibility, or motor dysfunction. Propulsive disorders leading to esophageal dysphagia result from abnormalities of peristalsis and/or deglutitive inhibition, potentially

affecting the cervical or thoracic esophagus. Since striated muscle pathol ogy usually involves both the oropharynx and the cervical esophagus, the clinical manifestations usually are dominated by oropharyngeal dysphagia. Diseases affecting smooth muscle involve both the thoracic esophagus and the LES. A dominant manifestation of this, absent peristal sis, refers to either the complete absence of swallow-induced contraction (absent contractility) or the presence of nonperistaltic, disordered con tractions. Absent peristalsis and failure of deglutitive LES relaxation are the defining features of achalasia. In distal esophageal spasm (DES), LES function is normal, with the disordered motility restricted to the esopha geal body. Absent contractility combined with severe weakness of the LES is a pattern commonly found in patients with scleroderma. APPROACH TO THE PATIENT Dysphagia Figure 47-2 shows an algorithm for the approach to a patient with dysphagia. HISTORY The patient history is extremely valuable in making a presumptive diagnosis or at least substantially limiting the differential diagnoses in most patients. Key elements of the history are the localization of dysphagia, the circumstances in which dysphagia is experienced, other symptoms associated with dysphagia, and progression. Dys phagia that localizes to the suprasternal notch may indicate either an oropharyngeal or an esophageal etiology as distal dysphagia is referred proximally about 30% of the time. Dysphagia that local izes to the chest is esophageal in origin. Nasal regurgitation and tracheobronchial aspiration manifest by coughing with swallow ing are hallmarks of oropharyngeal dysphagia. Severe cough with swallowing may also be a sign of a tracheoesophageal fistula. The presence of hoarseness may be another important diagnostic clue. When hoarseness precedes dysphagia, the primary lesion is usually laryngeal; hoarseness that occurs after the development of dyspha gia may result from compromise of the recurrent laryngeal nerve by a malignancy. The type of food causing dysphagia is an important Dysphagia localized to neck, nasal regurgitation, aspiration, associated ENT symptoms Oropharyngeal dysphagia Esophageal dysphagia Propulsive Propulsive Structural Structural Myogenic Neurogenic • Cerebral vascular accident • Parkinson's • Amyotropic lateral sclerosis • Brainstem tumor • Guillain-Barré • Huntington's chorea • Post-polio syndrome • Multiple sclerosis • Cerebral palsy • Zenker's diverticulum • Neoplasm • Cervical web • Cricopharyngeal bar • Osteophytes • Congenital abnormalities • Post head and neck surgery • Chemotherapy mucositis • Radiation • Corrosive injury • Infection • Myasthenia gravis • Polymyositis • Mixed connective tissue disorders • Oculopharyngeal muscular dystrophy • Paraneoplastic syndrome • Myotonic dystrophy • Sarcoidosis FIGURE 47-2 Approach to the patient with dysphagia. Etiologies in bold print are the most common. ENT, ear, nose, and throat; GERD, gastroesophageal reflux disease.

consideration. Intermittent dysphagia that occurs only with solid food implies structural dysphagia, whereas constant dysphagia with both liquids and solids strongly suggests an esophageal motor abnormality. Two caveats to this pattern are that despite having a motor abnormality, patients with scleroderma generally develop mild dysphagia for solids only and that patients with oropharyn geal dysphagia often have greater difficulty managing liquids than solids. Dysphagia that is progressive over the course of weeks to months raises concern for neoplasia. Episodic dysphagia to sol ids that is unchanged or slowly progressive over years indicates a benign disease process such as a Schatzki ring or eosinophilic esophagitis. Food impaction with a prolonged inability to pass an ingested bolus even with ingestion of liquid is typical of structural dysphagia. Chest pain may accompany dysphagia whether it is related to motor disorders, structural disorders, or reflux disease. A prolonged history of heartburn preceding the onset of dysphagia is suggestive of peptic stricture and, infrequently, esophageal adeno carcinoma. A history of prolonged nasogastric intubation, esopha geal or head and neck surgery, ingestion of caustic agents or pills, previous radiation or chemotherapy, or associated mucocutaneous diseases may help isolate the cause of dysphagia. With accompany ing odynophagia, which usually is indicative of ulceration, infec tious or pill-induced esophagitis should be suspected. In patients with AIDS or other immunocompromised states, esophagitis due to opportunistic infections such as Candida, herpes simplex virus, or cytomegalovirus and to tumors such as Kaposi's sarcoma and lym phoma should be considered. A history of atopy increases concerns for eosinophilic esophagitis, which is most prevalent in Caucasian male patients between the ages of 20 and 40 years. Medication use should identify agents associated with pill esophagitis and narcot ics that are associated with opioid-induced esophageal dysmotility. Dysphagia CHAPTER 47 PHYSICAL EXAMINATION Physical examination is important in the evaluation of oral and pha ryngeal dysphagia because dysphagia is usually only one of many manifestations of a more global disease process. Signs of bulbar or pseudobulbar palsy, including dysarthria, dysphonia, ptosis, and Dysphagia Dysphagia localized to chest or neck, food impaction Solid and liquid dysphagia Solid dysphagia Odynophagia Intermittent

• Schatzki ring • Esophageal web • Pill esophagitis • Infectious esophagitis • Caustic injury • Chemotherapy mucositis • Sclerotherapy • Crohn’s disease • Behcet’s syndrome • Bullous pemphygoid • Lichen planus Progressive • Neoplasm • GERD with weak peristalsis • Achalasia (primary and secondary) • Diffuse esophageal spasm • Scleroderma Variable • Peptic stricture • Eosinophilic esophagitis • Hiatal hernia • Extrinsic compression • Surgical stenosis • Radiation esophagitis • Ringed esophagus • Congenital esophageal stenosis

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