remove concomitant reflux as an aggravating factor. When healing results in stricture formation, dilation is indicated.

FOREIGN BODIES AND FOOD IMPACTION

Food or foreign bodies may lodge in the esophagus causing complete obstruction, which in turn can cause an inability to handle secretions (foaming at the mouth) and severe chest pain. Food impaction may occur due to stricture, carcinoma, Schatzki ring, eosinophilic esophagitis, or simply inattentive eating. If it does not spontaneously resolve, impacted food can be dislodged endoscopically. Use of meat tenderizer enzymes to facilitate passage of a meat bolus is discouraged because of potential esophageal injury. Glucagon (1 mg IV) is sometimes tried before endoscopic dislodgement. After emergent treatment, patients should be evaluated for potential causes of the impaction with treatment rendered as indicated.

ESOPHAGEAL MANIFESTATIONS OF SYSTEMIC DISEASE

SCLERODERMA AND COLLAGEN VASCULAR DISEASES

Scleroderma esophagus (hypotensive LES and absent esophageal peristalsis) was initially described as a manifestation of scleroderma or other collagen vascular diseases and thought to be specific for these disorders. However, this nomenclature subsequently proved unfortunate and has been discarded because an estimated half of qualifying patients do not have an identifiable systemic disease, and reflux disease is often the only identifiable association. When scleroderma esophagus occurs as a manifestation of a collagen vascular disease, the histopathologic findings are of infiltration and destruction of the esophageal muscularis propria with collagen deposition and fibrosis. The pathogenesis of absent peristalsis and LES hypotension in the absence of a collagen vascular disease is unknown. Regardless of the underlying cause, the manometric abnormalities predispose patients to severe GERD due to inadequate LES barrier function combined with poor esophageal clearance of refluxed acid. Dysphagia may also be manifest but is generally mild and alleviated by eating in an upright position and using liquids to facilitate solid emptying.

DERMATOLOGIC DISEASES

A host of dermatologic disorders (pemphigus vulgaris, bullous pemphigoid, cicatricial pemphigoid, Behçet's syndrome, and epidermolysis bullosa) can affect the oropharynx and esophagus, particularly the proximal esophagus with blisters, bullae, webs, and strictures. Glucocorticoid treatment is usually effective. Erosive lichen planus, Stevens-Johnson syndrome, and graft-versus-host disease can also involve the esophagus. Esophageal dilatation may be necessary to treat strictures.

348 Peptic Ulcer Disease and Related Disorders

John Del Valle

PEPTIC ULCER DISEASE

Burning epigastric pain exacerbated by fasting and improved with meals is a symptom complex associated with peptic ulcer disease (PUD). An *ulcer* is defined as disruption of the mucosal integrity of the stomach and/or duodenum leading to a local defect or excavation due to active inflammation. Ulcers occur within the stomach and/or duodenum and are often chronic in nature. Acid peptic disorders are very common in the United States, with 4 million individuals (new cases and recurrences) affected per year. Lifetime prevalence of PUD in the United States is ~12% in men and 10% in women. PUD significantly affects quality of life by impairing overall patient well-being and contributing substantially to work absenteeism. Moreover, an estimated 15,000 deaths per year occur as a consequence of complicated PUD. The financial impact of these common disorders has been substantial, with an estimated burden on direct and indirect health care costs of ~$6 billion per year in the United States, with $3 billion spent on hospitalizations, $2 billion on physician office visits, and $1 billion in decreased productivity and days lost from work.

GASTRIC PHYSIOLOGY

Despite the constant attack on the gastroduodenal mucosa by a host of noxious agents (acid, pepsin, bile acids, pancreatic enzymes, drugs, and bacteria), integrity is maintained by an intricate system that provides mucosal defense and repair.

Gastric Anatomy The gastric epithelial lining consists of rugae that contain microscopic gastric pits, each branching into four or five gastric glands made up of highly specialized epithelial cells. The makeup of gastric glands varies with their anatomic location. Glands within the gastric cardia comprise <5% of the gastric gland area and contain mucous and endocrine cells. The 75% of gastric glands are found within the oxyntic mucosa and contain mucous neck, parietal, chief, endocrine, enterochromaffin, and enterochromaffin-like (ECL) cells (Fig. 348-1). Pyloric glands contain mucous and endocrine cells (including gastrin cells) and are found in the antrum.

The parietal cell, also known as the oxyntic cell, is usually found in the neck, or isthmus, or in the oxyntic gland. The resting, or unstimulated, parietal cell has prominent cytoplasmic tubulovesicles and intracellular canaliculi containing short microvilli along its apical surface (Fig. 348-2). H^+,K^+-adenosine triphosphatase (ATPase) is expressed in the tubulovesicle membrane; upon cell stimulation, this membrane, along with apical membranes, transforms into a dense network of apical intracellular canaliculi containing long microvilli. Acid secretion, a

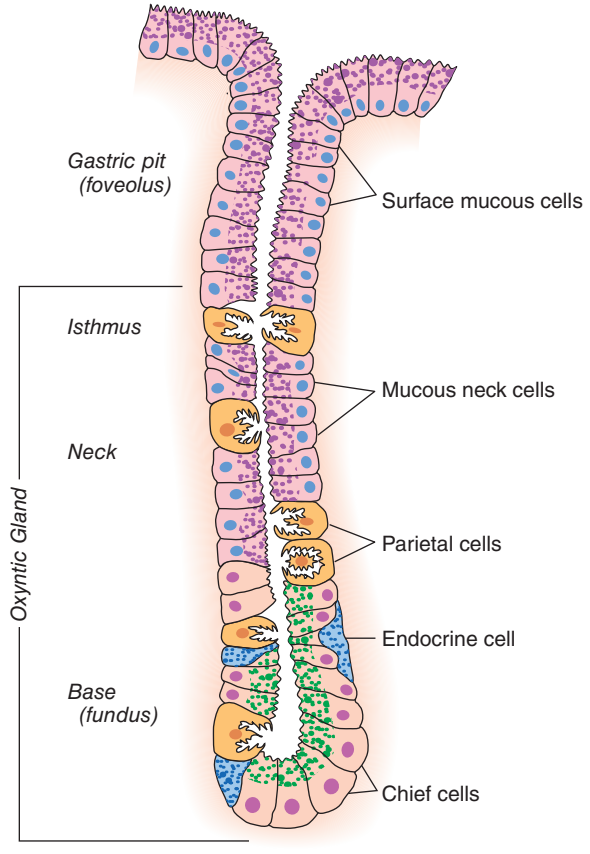

FIGURE 348-1 Diagrammatic representation of the oxyntic gastric gland. *(Adapted from S Ito, RJ Winchester: J Cell Biol 16:541, 1963. doi:10.1083/jcb.16.3.541. © 1963 Ito and Winchester.)*