TREATMENT

Treatment of dysphagia depends on both the locus and the specific etiology. Oropharyngeal dysphagia most commonly results from functional deficits caused by neurologic disorders. In such circumstances, the treatment focuses on utilizing postures or maneuvers devised to reduce pharyngeal residue and enhance airway protection learned under the direction of a trained swallow therapist. Aspiration risk may be reduced by altering the consistency of ingested food and liquid. Dysphagia resulting from a cerebrovascular accident usually, but not always, spontaneously improves within the first few weeks after the event. More severe and persistent cases may require gastrostomy and enteral feeding. Patients with myasthenia gravis (Chap. 461) and polymyositis (Chap. 388) may respond to medical treatment of the primary neuromuscular disease. Surgical intervention with cricopharyngeal myotomy is usually not helpful, with the exception of specific disorders such as the idiopathic cricopharyngeal bar, Zenker's diverticulum, and oculopharyngeal muscular dystrophy. Chronic neurologic disorders such as Parkinson's disease and amyotrophic lateral sclerosis may manifest with severe oropharyngeal dysphagia. Feeding by a nasogastric tube or an endoscopically placed gastrostomy tube may be considered for nutritional support; however, these maneuvers do not provide protection against aspiration of salivary secretions or refluxed gastric contents.

Treatment of esophageal dysphagia is covered in detail in Chap. 347. The majority of causes of esophageal dysphagia are effectively managed by means of esophageal dilatation using bougie or balloon dilators. Cancer and achalasia are often managed surgically, although endoscopic techniques are available for both palliation and primary therapy, respectively. Infectious etiologies respond to antimicrobial medications or treatment of the underlying immunosuppressive state. Finally, eosinophilic esophagitis has emerged as an important cause of dysphagia that is amenable to treatment by elimination of dietary allergens or administration of swallowed, topically acting glucocorticoids.

54 Nausea, Vomiting, and Indigestion

William L. Hasler

Nausea is the subjective feeling of a need to vomit. *Vomiting* (emesis) is the oral expulsion of gastrointestinal contents due to contractions of gut and thoracoabdominal wall musculature. Vomiting is contrasted with *regurgitation,* the effortless passage of gastric contents into the mouth. *Rumination* is the repeated regurgitation of food residue, which may be rechewed and reswallowed. In contrast to emesis, these phenomena may exhibit volitional control. *Indigestion* is a term encompassing a range of complaints including nausea, vomiting, heartburn, regurgitation, and dyspepsia (the presence of symptoms thought to originate in the gastroduodenal region). Some individuals with dyspepsia report predominantly epigastric burning, gnawing, or pain. Others experience postprandial fullness, early satiety (an inability to complete a meal due to premature fullness), bloating, eructation (belching), and anorexia.

NAUSEA AND VOMITING

MECHANISMS

Vomiting is coordinated by the brainstem and is effected by responses in the gut, pharynx, and somatic musculature. Mechanisms underlying nausea are poorly understood but likely involve the cerebral cortex, as nausea requires conscious perception. This is supported by functional brain imaging studies showing activation of a range of cerebral cortical regions during nausea.

Coordination of Emesis Brainstem nuclei—including the nucleus tractus solitarius; dorsal vagal and phrenic nuclei; medullary nuclei regulating respiration; and nuclei that control pharyngeal, facial, and tongue movements—coordinate initiation of emesis. Neurokinin NK_1, serotonin 5-HT_3, and vasopressin pathways participate in this coordination.

Somatic and visceral muscles respond stereotypically during emesis. Inspiratory thoracic and abdominal wall muscles contract, producing high intrathoracic and intraabdominal pressures that evacuate the stomach. The gastric cardia herniates above the diaphragm, and the larynx moves upward to propel the vomitus. Distally migrating gut contractions are normally regulated by an electrical phenomenon, the slow wave, which cycles at 3 cycles/min in the stomach and 11 cycles/min in the duodenum. During emesis, the slow wave is abolished and is replaced by orally propagating spikes that evoke retrograde contractions that assist in expulsion of gut contents.

Activators of Emesis Emetic stimuli act at several sites. Emesis evoked by unpleasant thoughts or smells originates in the brain, whereas cranial nerves mediate vomiting after gag reflex activation. Motion sickness and inner ear disorders act on the labyrinthine system. Gastric irritants and cytotoxic agents like cisplatin stimulate gastroduodenal vagal afferent nerves. Nongastric afferents are activated by intestinal and colonic obstruction and mesenteric ischemia. The area postrema, in the medulla, responds to bloodborne stimuli (emetogenic drugs, bacterial toxins, uremia, hypoxia, ketoacidosis) and is termed the *chemoreceptor trigger zone*.

Neurotransmitters mediating vomiting are selective for different sites. Labyrinthine disorders stimulate vestibular muscarinic M_1 and histaminergic H_1 receptors. Vagal afferent stimuli activate serotonin 5-HT_3 receptors. The area postrema is served by nerves acting on 5-HT_3, M_1, H_1, and dopamine D_2 subtypes. Cannabinoid CB_1 pathways may participate in the cerebral cortex. Optimal pharmacologic therapy of vomiting requires understanding of these pathways.

DIFFERENTIAL DIAGNOSIS

Nausea and vomiting are caused by conditions within and outside the gut as well as by drugs and circulating toxins (Table 54-1).

Intraperitoneal Disorders Visceral obstruction and inflammation of hollow and solid viscera may elicit vomiting. Gastric obstruction results from ulcers and malignancy, whereas small-bowel and colon blockage occur because of adhesions, benign or malignant tumors, volvulus, intussusception, or inflammatory diseases like Crohn's disease. The superior mesenteric artery syndrome, occurring after weight loss or prolonged bed rest, results when the duodenum is compressed by the overlying superior mesenteric artery. Abdominal irradiation impairs intestinal motor function and induces strictures. Biliary colic causes nausea by acting on local afferent nerves. Vomiting with pancreatitis, cholecystitis, and appendicitis is due to visceral irritation and induction of ileus. Enteric infections with viruses like norovirus or rotavirus or bacteria such as *Staphylococcus aureus* and *Bacillus cereus* often cause vomiting, especially in children. Opportunistic infections like cytomegalovirus or herpes simplex virus induce emesis in immunocompromised individuals.

Gut sensorimotor dysfunction often causes nausea and vomiting. *Gastroparesis* presents with symptoms of gastric retention with evidence of delayed gastric emptying and occurs after vagotomy or with pancreatic carcinoma, mesenteric vascular insufficiency, or organic diseases like diabetes, scleroderma, and amyloidosis. Idiopathic gastroparesis is the most common etiology. It occurs in the absence of systemic illness and may follow a viral illness, suggesting an infectious trigger. *Intestinal pseudoobstruction* is characterized by disrupted intestinal and colonic motor activity with retention of food residue and secretions; bacterial overgrowth; nutrient malabsorption; and symptoms of nausea, vomiting, bloating, pain, and altered defecation. Intestinal pseudoobstruction may be idiopathic, inherited as a familial visceral myopathy or neuropathy, result from systemic disease, or