Vomiting is the reflexive, forceful expulsion of part or all of the contents of the stomach and proximal small intestines through the mouth. It involves a series of involuntary spasmic movements requiring visceral, diaphragmatic, and abdominal muscular contractions. The act of vomiting is usually preceded by a number of stereotypical prodromal clinical signs, including excessive salivation, repeated swallowing, retching, and marked contractions of the abdominal musculature. Vomiting must be differentiated from regurgitation, dysphagia (difficulty swallowing), and various forms of esophageal dysfunction. Regurgitation is a passive process whereby partially or completely undigested food or liquid is released from the esophagus and/or stomach through the mouth without effort or muscular contractions (ie, through gravity and body position). Although dysphagia involves active but ineffective muscular contractions and may produce movements that closely resemble the retching associated with vomiting, the process represents a dysfunctional movement of liquid and/or food into, not out of, the GI tract.

**Etiology, Pathophysiology, and Clinical Findings**

The reflex act of vomiting is initiated by the vomiting (or emetic) center, located in the reticular formation of the medulla. The vomiting center is responsive to input from 4 major sources: 1) afferent receptors from a variety of peripheral structures (the GI tract, pancreas, heart, liver, urogenital tract, and peritoneum) carried by the vagus and sympathetic nerves; 2) the chemoreceptor trigger zone (CRTZ) of the area postrema of the medulla; 3) the cerebral cortex and limbic system; and 4) the vestibular apparatus. In this way, the vomiting center is responsive to neural, humoral, and chemical input. Stimulation of the receptors in the semicircular canals of the vestibular system, increases in intracranial pressure, distention of the duodenum (eg, foreign body obstruction, gas and ileus secondary to inflammation) or colon (eg, constipation), organ capsule distention or inflammation (eg, acute liver failure or pyelonephritis), bloodborne toxins (eg, exogenous chemotherapeutics or endogenous uremic toxins) are examples of the varied inputs that may activate the vomiting reflex and demonstrate why the list of potential causes for a vomiting patient is so extensive.

Some additional causes of vomiting include toxins (pesticides, zinc, xylitol, mycotoxins), envenomation, parasites (heartworm in cats, *Spirocerca lupi*, *Physaloptera*, *Ollulanus tricuspis*), infection (*Helicobacter*) and inflammation (pancreatitis), drugs (acyclovir, cisplatin, piperazine, zolpidem, antibiotics), obstruction (intussusception, foreign body, obstipation), food allergy, motility disorders (bilious vomiting syndrome, hiatal hernia), metabolic and electrolyte derangements, neurologic disease (dysautonomia, limbic epilepsy), neoplasia, enterotoxemia, and septicemia.

Anxiety, depression, hypersalivation, and repeated swallowing accompanied by relaxation of the gastroesophageal sphincter are followed by retching. The proximal small intestine and gastric antrum contract, propelling their contents into the body of the stomach where movement is inhibited. The gastroesophageal sphincter moves into the thoracic cavity, rendering it incompetent and facilitating gastroesophageal reflux. Esophageal and pharyngoesophageal sphincter motility is repressed, and the nasopharynx closes to prevent nasal regurgitation. Forceful contractions of the abdominal muscles and diaphragm against a closed glottis combined with increases in intra-abdominal pressure force expulsion of food, fluid, or debris.

**Diagnosis**

Diagnosis begins with accurate and complete identification of the problem. This includes differentiating vomiting from regurgitation or dysphagia, and then characterizing the duration and character of the vomiting. Although the complete list of potential causes for vomiting is extensive and potentially relevant for any vomiting patient, the rank-order of that differential list is significantly affected by a variety of historical factors. The problem should be defined as acute or chronic, persistent or intermittent, static, progressive, or recurrent. The problem of vomiting can be further defined in terms of the frequency and time of day it occurs, character of the vomitus, changes in dietary or environmental circumstances that preceded the problem, and general condition of the patient (eg, attitude, activity, appetite, appearance) between bouts. Both history and physical examination should reveal potential systemic causes or consequences of the problem, such as depression, dehydration, fever, halitosis, weight loss, dyspnea (aspiration), abdominal pain, masses, thickened or distended loops of bowel, excessive or absent borborygmus, or melena.

Following a complete history and physical examination, primary and secondary GI differential diagnoses should be considered. Plain film abdominal radiographs should be part of the initial diagnostic work-up of any vomiting patient; an undiagnosed GI foreign body in an acutely vomiting patient or an undiagnosed splenic mass in a chronically vomiting patient are both potentially fatal causes of vomiting whose identification would be delayed by symptomatic treatment in the absence of appropriate diagnostic tests, to the detriment of the patient. A minimum data base, including a CBC, biochemical profile, urinalysis, and fecal examination, are also appropriate initial diagnostics, although they are less likely to identify underlying causes that would require immediate intervention.

Chronic vomiting and vomiting that has systemic manifestations such as hematemesis, abdominal pain, dehydration, fever, or weight loss should be approached aggressively. An expanding array of diagnostic tests is available to diagnose both primary and secondary causes of vomiting. Noninvasive imaging modalities in addition to plain abdominal films include contrast studies, abdominal ultrasonography, and abdominal CT or MRI. Fecal float, smear, wet mount, *Giardia* ELISA, PCR, electron microscopy, and assay of α-antiprotease levels (for protein-losing enteropathies) are available to help define GI disease. Pancreatic-lipase immunoreactivity (PLI) has replaced measures of serum amylase and lipase as a much more sensitive test of pancreatic inflammation. Laparoscopy and endoscopy are powerful diagnostic tools for both secondary and primary causes of vomiting, and they entail much less morbidity than exploratory abdominal laparotomy.

**Treatment and Control**

The primary goal of treatment in a vomiting patient is to identify and treat the underlying cause of the vomiting. Obtaining a definitive diagnosis prior to instituting symptomatic treatment is almost always a prudent and frequently cost-effective approach in any vomiting patient. Antiemetic therapy is an essential component of the plan for many vomiting patients, but with administration of an antiemetic, the clinician losses the ability to correlate the cessation of vomiting to any other specific parameter (ie, rehydration, resolution of a partial obstruction, effective treatment of an underlying disease process, the natural progression of simple gastritis).

Symptomatic therapy for acute vomiting includes withholding food for 24 hr. Water should never be withheld unless the animal is receiving SC or IV fluid support. Predictable consequences of vomiting include dehydration, electrolyte imbalances, and acid-base disturbances; withholding water without appropriately addressing the patient's hydration status will exacerbate and compound these abnormalities. Some animals show a positive response to this 24-hr intervention, and small volumes of oral liquid, and eventually food, can be slowly reintroduced without further vomiting episodes.

<http://www.merckmanuals.com/vet/digestive_system/vomiting/overview_of_vomiting.html>