PediatricsinReview®

Vomiting

Karen F. Murray and Dennis L. Christie

Pediatr. Rev. 1998;19;337
DOI: 10.1542/pir.19-10-337

Vomiting

Karen F. Murray, MD* and Dennis L. Christie, MD*

IMPORTANT POINTS

1. **Because the causes of vomiting are broad, proper diagnosis requires a high index of suspicion and a thorough history and evaluation.**
2. **Bilious vomiting is an ominous sign that mandates immediate evaluation.**
3. **Vomiting may be a symptom of a systemic disease that is not primarily of the gastrointestinal tract.**
4. **The patient who has bloody emesis requires hemodynamic stabilization before diagnostic studies are performed.**
5. **Psychological causes of vomiting can be serious and difficult to treat.**

Definition

Vomiting is a generally unpleasant activity that results in the expulsion of stomach contents through the mouth. It is a physical act that has clearly associated gastrointestinal motor activity. Nausea, on the other hand, although frequently accompanying vomiting, is not universally associated with it and does not have an obvious physical mechanism. It is an uncomfortable feeling known to be relieved by vomiting.

Physiology

The ability to vomit presumably conveys a survival advantage by enabling the expulsion of toxins from the stomach. Vomiting occurs after stimulation of either the vomiting center (VC), a central "control center" in the medulla near the respiratory center, or the chemoreceptor trigger zone (CTZ) in the area postrema on the floor of the fourth ventricle (Fig. 1). These coordination centers can be stimulated through multiple pathways. Vomiting resulting from psychological stress occurs via pathways traveling through the cerebral cortex and limbic system to the VC. Anticipatory vomiting may be mediated through this mechanism. Vomiting related to motion occurs when the VC is stimulated through the vestibular or vestibulocerebellar system from the labyrinth of the inner ear. Chemical signals from the bloodstream and cerebrospinal fluid are detected by the CTZ. This mechanism has been the target of many antiemetic medications. The vagal and visceral nerves are the fourth pathway for stimulation of vomiting via gastrointestinal irritation, distention, and delayed gastric emptying.

Once the vomiting centers are stimulated, the cascade of motor events leading to the act of vomiting is the same. Nonperistaltic contractions in the small intestine increase, the gallbladder contracts, and some of the duodenal contents regurgitate into the stomach. This is followed by a large retrograde peristaltic wave that pushes small bowel contents and pancreatobiliary secretions into the stomach and suppresses gastric activity. Meanwhile, the inspiratory muscles contract against a closed glottis, resulting in esophageal dilatation. As the abdominal muscles contract, the stomach contents are forced into the distal esophagus. Relaxation of the abdominal muscles allows the esophageal contents to re-enter the stomach. The cycles of retching quicken until the esophagus no longer empties between cycles, and the contents finally are extruded. The gastrointestinal motor events of vomiting are mediated through vagal and sympathetic efferents from the VC, as are the autonomic events associated with the act of vomiting, namely, increased salivation, increased respiratory and heart rates, and pupillary dilatation.

Pathogenesis

True vomiting can be divided into two broad categories: nonbilious and bilious. Bilious vomiting occurs when bile is purged along with the gastric contents. Although some small intestinal reflux into the stomach is common with all vomiting, in nonbilious vomiting, antegrade intestinal flow is preserved, and the majority of the bile drains into the more distal portions of the intestine. If an obstruction is present, nonbilious vomiting implies that the obstruction is proximal to the ampulla of Vater. Conditions leading to bilious vomiting involve either a disorder of motility or physical blockage to this antegrade flow of

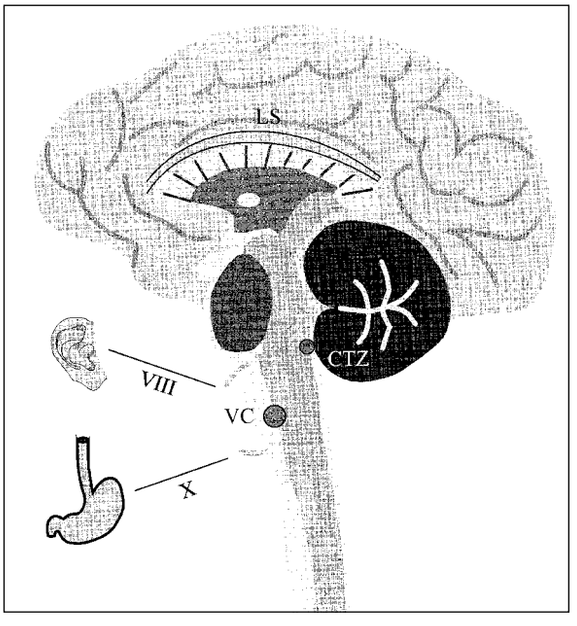

FIGURE 1. Central nervous system coordination of vomiting: LS = limbic system, CTZ = chemoreceptor trigger zone, VC = vomiting center, VIII = vestibular nerve, X = vagus nerve.

Division of Gastroenterology, Children's Hospital and Regional Medical Center, Seattle, WA.

proximal intestinal contents distal to the ligament of Treitz.

Gastroesophageal reflux (GER), although not true vomiting, frequently is included in discussions of vomiting. In contrast to the mechanism of true vomiting discussed previously, GER occurs as a result of failed normal esophageal function. Normally, the lower esophageal sphincter (LES) relaxes with swallowing and propagation of esophageal peristalsis, allowing a food bolus to enter the stomach. Its basal contraction prevents food from re-entering the esophagus from the stomach. Transient relaxation of the LES predisposes to GER and is the major mechanism in infants who have GER. The LES is aided by surrounding structures, especially the crural diaphragm, and disruption of these structures, as with a hiatal hernia, contributes to the GER in some patients. Nicotine, alcohol, caffeine, and some medications can increase relaxation of the LES, contributing to GER in some individuals. GER also is distinguished from true vomiting by its symptoms; the emesis of GER is effortless and generally not associated with retching or autonomic symptoms.

Causes of Vomiting

Although the causes of vomiting in children are many, a thorough history of the nature of the vomiting and any associated signs or symptoms as well as a complete physical examination generally helps narrow the differential diagnosis (Table 1). Targeted studies help identify the cause of the illness.

NONBILIOUS

Infectious/inflammatory

Acute gastroenteritis is the most common cause of vomiting in children. It is usually associated with diarrhea and abdominal pain, and viruses are common etiologic agents, although bacterial pathogens also must be considered. The most common viral agent in infants is rotavirus. Bacterial pathogens include *Salmonella*, *Shigella*, *Campylobacter*, and *Escherichia coli.* Bacterial infections are associated more commonly with bloody diarrhea and high fevers than are viral infections. Enterohemorrhagic *E coli* 0157:H7 can cause hemorrhagic colitis and may be complicated by the development of hemolytic-uremic syndrome. *Clostridium difficile* is a bacterial pathogen that frequently is associated with the recent use of antibiotics. It causes a pseudomembranous colitis, often with bloody diarrhea and associated with abdominal pain and vomiting. *Giardia lamblia* is a protozoan commonly associated with contaminated water and attendance at child care centers. It may cause watery diarrhea and vomiting.

Diagnosis is made with stool bacterial cultures, rotazyme analysis for rotavirus, detection of *C difficile* toxin, and detection of ova and parasites for *G lamblia.* Sepsis, central nervous system infections, urinary tract infections, and pneumonia all can present with or involve vomiting, usually in addition to other symptoms.

Labyrinthitis and pancreatitis both cause vomiting. Dizziness usually is associated with labyrinthitis and abdominal pain with pancreatitis.

Inflammatory conditions of the intestinal tract, such as inflammatory bowel disease, also tend to involve vomiting. In these conditions, the vomiting frequently is related to altered motility with abnormal or dysfunctional swallowing, gastric emptying, or peristalsis.

Metabolic/endocrinologic

Both inborn errors of metabolism and endocrinologic disorders can cause vomiting (Table 2). The inborn errors of metabolism generally present in early infancy, and the vomiting is associated with symptoms of lethargy, hypo- or hypertonia, seizures, or coma. The constellation of symptoms is similar to that seen in sepsis, necessitating a high index of suspicion in the evaluation of these patients. The presence or absence of metabolic acidosis, hypoglycemia, hyperammonemia, or ketosis and a family history that includes possible consanguinity can help to determine the diagnosis.

One endocrinologic condition associated with vomiting is diabetes mellitus. Vomiting can complicate acute ketoacidosis or occur in patients who have had long-standing diabetes and consequent gastroparesis. Slowed gastric motility usually presents after diabetes mellitus has been present for approximately 10 years. Early

TABLE 1. General Causes of Vomiting

Nonbilious
• Infectious/inflammatory
• Metabolic/endocrinologic
• Neurologic
• Psychological
• Obstructive lesion
Bilious
• Distal obstructive lesion

TABLE 2. Selected Inborn Errors of Metabolism Associated With Vomiting

Carbohydrate Metabolism Defects
• Glycogen storage disease II (Pompe disease)
• Galactosemia
• Hereditary fructose intolerance
• Pyruvate carboxylase deficiency
• Pyruvate dehydrogenase complex deficiency
Amino Acid/Organic Acid Metabolism Defects
• Urea cycle defects
• Phenylketonuria
• Maple syrup urine disease
• Propionic acidemia
• Glutaric acidemia
• Isovaleric acidemia
• Tyrosinemia type I
Lysosomal Storage Diseases
• Mucopolysaccharidoses
• Mucolipidoses
• Niemann-Pick disease
• Wolman disease
Peroxisomal Disorders
• Zellweger disease
• Adrenal leukodystrophy
Fatty Acid Oxidation Disorders
• Carnitine deficiency syndromes
• MCAD, LCAD

satiety and a sense of fullness frequently precede the onset of vomiting in diabetic gastroparesis.

Vomiting as a consequence of food-related sensitivity always should be considered. The variants encountered most commonly in pediatric patients are cow milk and soy protein intolerance, type I (IgE-mediated) food allergy, and celiac disease. Cow milk protein intolerance affects 2% to 7% of infants, with approximately 20% of these also sensitive to soy protein. In addition to vomiting, the patients usually have diarrhea that frequently is guaiac-positive. Celiac disease occurs only in children who eat gluten-containing foods. Patients typically suffer from wasting, irritability, and diarrhea, but vomiting also can occur.

Neurologic

Vomiting occurs in any neurologic condition that involves increased intracranial pressure (ICP) (Table 3). Additionally, patients who have seizures, autonomic disorders (Riley-Day syndrome), and conditions affecting the floor of the fourth ventricle without increased ICP frequently have their condition worsened with vomiting.

Cyclic vomiting is a unique entity that must be considered separately. Onset occurs typically at early school age, and it is characterized by acute-onset periodic episodes of nausea and vomiting interspersed with conspicuous periods of wellness. Approximately 77% of patients can identify precipitating events, usually intense emotional states. These patients have an increased incidence of migraine headaches and prevalence of epilepsy and irritable bowel syndrome. Because no diagnostic test or specific clinical identifying feature distinguishes cyclic vomiting syndrome from other potentially life-threatening conditions, the diagnosis can be made only after time has elapsed and upon exclusion of other diagnoses.

TABLE 3. Selected Neurologic Conditions Associated With Vomiting

Structural
• Hydrocephalus
• Congenital malformations
• Intracranial hemorrhage
• Intracranial mass lesions
Infectious
• Congenital infections
• Encephalitis and meningitis
Toxic
• Kernicterus
• Acidosis and other metabolic byproducts

Psychological

Behavioral or psychological causes of vomiting can be problematic in the pediatric age group. Some children induce vomiting to seek attention in environments in which personal attention is lacking. The extreme of this behavior is rumination. Rumination is a serious condition that occurs in infants when there is a failure in reciprocal interaction between the infant and caregiver. The purposes of rumination are self-stimulation and satisfaction of needs. Rumination also is seen in older children, especially those who are severely mentally retarded.

Classically, the infant (older than 3 months) learns to bring up gastric contents into the mouth, frequently by inserting a hand into the back of the throat or simply through rhythmic contractions of the pharynx, tongue, and abdominal muscles. The oral contents then are reswallowed, although spillage does occur. Not only do these patients suffer social deprivation, but the chronic emesis can lead to inanition and growth failure. The failure to thrive does not improve with traditional medical intervention, but rather requires sensitive and interactive nurturing. A mother-substitute who is empathic with and observant of both the parent's and infant's needs is required to help the parent establish more nurturing skills. More formal psychotherapeutic help frequently is needed for the parents.

Bulimia, characterized by secretive binge-eating episodes followed by self-induced vomiting, is a cause of vomiting especially among teenagers.

Anatomic

The anatomic and, thus, the generally surgical causes of nonbilious vomiting are those that affect the intestinal tract proximal to the point of bilious drainage (ampulla of Vater), which is proximal to the ligament of Treitz (Table 4). Whereas congenital anomalies usually present in the newborn period, acquired lesions can present at any age. Any infant who exhibits persistent nonbilious vomiting, with or without feeding, in the immediate newborn period must be suspected of having an intestinal atresia or a luminally obstructing lesion (pyloric stenosis, luminal band, web) proximal to the point of bile drainage (ampulla of Vater).

An easy and rapid test to evaluate possible esophageal atresia is the ability to pass a nasogastric tube easily into the stomach. After the tube has been passed, it is important to obtain a radiograph to assure that the tube is in the stomach and not coiled in an atretic esophagus. Any resistance to passage of the tube is an indication for evaluation by contrast radiograph for an obstruction. If an obstruction is present, nasoesophageal tube drainage is important to prevent aspiration of pooled esophageal secretions. Contrast studies are the standard for the diagnosis of these conditions (Figs 2-4).

TABLE 4. Physical Conditions of the Gastrointestinal Tract That Cause Nonbilious Vomiting

Structural
• Foreign body
• Esophageal/gastric atresia
• Esophageal/gastric stenosis
• Stricture
• Duplication/diverticulum/ choledochal cyst
• Pyloric stenosis
• Annular pancreas
• Web
• Peptic disease
Disorders of Motility
• Achalasia
• Ileus
• Scleroderma
• Gastroparesis
• Appendicitis
• Pseudo-obstruction

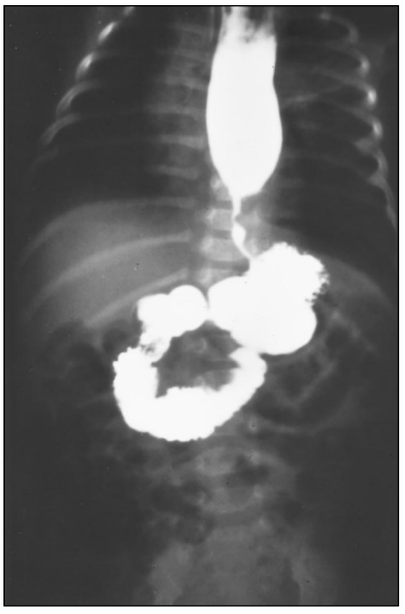

FIGURE 2. Barium study showing a distal esophageal stricture with proximal esophageal dilatation.

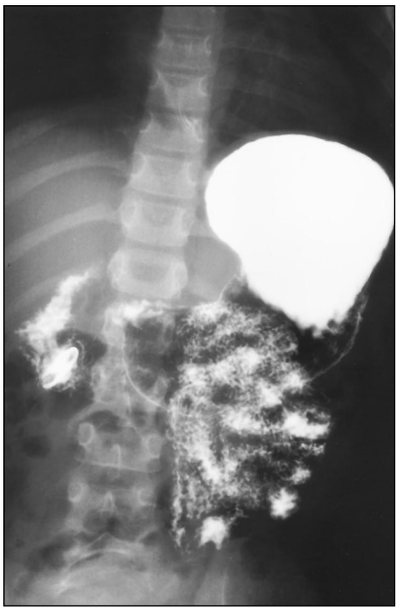

FIGURE 3. Barium study showing a coin partially obstructing the duodenum.

TABLE 5. Conditions That Can Cause Bilious Vomiting in Children

• Intestinal atresia and stenosis
• Malrotation with or without volvulus
• Ileus from any cause
• Intussusception
• Intestinal duplication
• Compressing or obstructing mass lesion
• Incarcerated inguinal hernia
• Superior mesenteric artery syndrome
• Appendicitis
• Peritoneal adhesions
• Pseudo-obstruction

FIGURE 4A. Barium study illustrating a malrotation with volvulus.

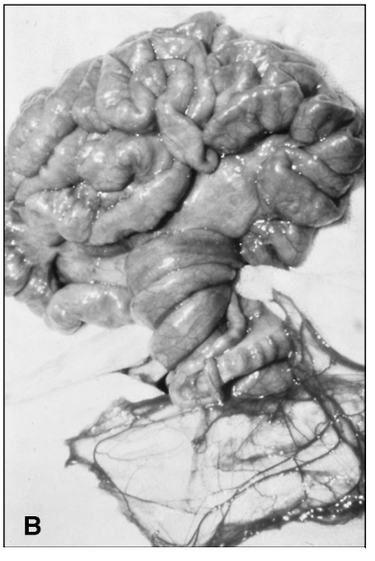

FIGURE 4B. Surgical photo of an intestinal malrotation with volvulus.

TABLE 6. Some Causes of Upper Gastrointestinal Bleeding

• Esophagitis/gastritis
• Peptic ulcer disease (gastric/duodenal)
• Mallory-Weiss tear
• Bleeding varices
• Dieulafoy lesion

BILIOUS

Although not absolute, anatomic conditions causing luminal obstruction distal to the ligament of Treitz usually cause bilious vomiting. Bilious vomiting is an ominous sign that mandates immediate evaluation (Table 5).

In the newborn period, intestinal atresias and stenosis and malrotation with or without volvulus need to be ruled out immediately. In the older child, malrotation with volvulus also is a surgical emergency that is diagnosed relatively easily by gastrointestinal contrast study. After the diagnosis has been established radiographically, the gastrointestinal tract should be decompressed with a nasogastric tube, food and drink withheld, and the patient supported with intravenous fluids until definitive surgical intervention can be undertaken.

Vomiting of Blood

Bright red blood in emesis implies active bleeding in the esophagus, stomach, or proximal duodenum. Coffee-ground color (darker oxidized blood), on the other hand, implies a recent history of bleeding. Fortunately, gastrointestinal bleeding is relatively rare in children. Children who experience massive gastrointestinal bleeding frequently have predisposing conditions, such as esophageal varices from chronic liver disease (Table 6). When such bleeding is encountered, initial therapy always is stabilization and resuscitation of the patient. Minimal bleeding that does not result in any change in hemodynamics or hematocrit frequently can be treated with histamine-2 blockers or antacids. Larger hemorrhages, however, require further intervention. The rate and volume of bleeding should be measured early, and if significant, a nasogastric tube left in place for

continuous monitoring and removal of the gastric blood. After stabilization, the patient should be transported to a facility where there are endoscopists and surgeons skilled in pediatric care and management of gastrointestinal bleeding.

SUGGESTED READING

Caty MG, Azizkhan RG. Acute surgical conditions of the abdomen. *Pediatr Ann.* 1994; 4:192–201

Cohen R. Metabolic and infectious disorders associated with emesis in infants. *Semin Pediatr Surg.* 1995;4:136–146

Fleisher DR. Functional vomiting disorders in infancy: innocent vomiting, nervous vomiting, and infant rumination syndrome. *J Pediatr.* 1994;125(suppl):S84–S94

Fleisher DR. The cyclic vomiting syndrome described. *J Pediatr Gastroenterol Nutr.* 1995;21(suppl):S1–S5.

Forbes D. Differential diagnosis of cyclic vomiting syndrome. *J Pediatr Gastroenterol Nutr.* 1995;21(suppl):S11–S14

Gordon N. Recurrent vomiting in childhood, especially of neurological origin. *Dev Med Child Neurol.* 1994;36:463–470

Johns DW. Disorders of the central and autonomic nervous systems as a cause for emesis in infants. *Semin Pediatr Surg.* 1995;4:152–156

Keller VE. Management of nausea and vomiting in children. *J Pediatr Nurs.* 1995;10: 280–286

Mehler PS. Eating disorders: 2. bulimia nervosa. *Hosp Pract.* 1996;31:107–126.

Orenstein SR. Gastroesophageal reflux. *Pediatr Rev.* 1992;13:174–182

Sondheimer JM. Vomiting. In: Walker WA, Durie PR, Hamilton JR, Walker-Smith LA, Watkins JB, eds. *Pediatric Gastrointestinal Disease, Pathophysiology, Diagnosis, Management.* Vol. 1. St. Louis, Mo: Mosby; 1996:195–203

PIR QUIZ

6. A newborn male spits up his first feeding and develops bilious emesis with subsequent feedings. On physical examination, he appears ill, has a scaphoid abdomen, and has absent bowel sounds. A plain radiograph reveals air in the proximal small bowel but a paucity of air in the distal digestive tract. Of the following, the *most* likely cause for this infant's vomiting and clinical findings is:
   A. Antral web.
   B. Choledochal cyst.
   C. Hirschsprung disease.
   D. Tracheoesophageal fistula.
   E. Volvulus.

7. A 13-month-old girl is referred because of nonbilious vomiting, failure to thrive, and chronic diarrhea. She had done well on a cow milk formula until 6 months of age, but difficulties developed when solid foods were introduced. Physical examination reveals a wasted, irritable toddler who has a protuberant abdomen and wasted extremities. The *most* likely cause for this child's vomiting and clinical symptoms is:
   A. Celiac disease.
   B. Cow milk/soy protein allergy.
   C. Hiatal hernia.
   D. Intussusception.
   E. Urinary tract infection.

8. A 6-year-old girl has had abdominal pain and nonbilious vomiting for 8 hours. History reveals cough and fever for the past 3 days. Findings on physical examination include temperature of 39˚C (102.2˚F); tachypnea; toxic appearance; diffuse, voluntary guarding; and quiet bowel sounds. The examination *most* likely to confirm the etiology of the abdominal pain and fever in this patient is a(n):
   A. Abdominal radiograph.
   B. Chest radiograph.
   C. Complete blood count.
   D. Rectal examination.
   E. Upper gastrointestinal series.

9. A 7-year-old girl has been having recurrent bouts of nonbilious emesis for 18 months. The vomiting episodes occur every 3 to 4 weeks, last 48 hours, and often require intravenous fluids to prevent dehydration. The patient is otherwise well between vomiting episodes. Results of evaluation, including blood chemistries, complete blood count, urinalysis, upper endoscopy, abdominal computed tomography, barium swallow, and head magnetic resonance imaging, have been normal. The *most* likely cause for this child's vomiting is
   A. Cyclic vomiting syndrome.
   B. Glycogen storage disease II.
   C. Hydrocephalus.
   D. Peptic ulcer disease.
   E. Recurrent intussusception.

10. A 12-year-old boy with alpha$_1$-antitrypsin deficiency presents vomiting bright red blood. Physical examination reveals an anxious, diaphoretic child who is inconsolable. He has a firm, enlarged liver palpable 2 cm below the right costal margin; splenomegaly; and a prominent vascular pattern over the abdomen. Of the following, the *most* important first step in management is to:
    A. Order an acute abdominal radiographic series.
    B. Perform a tagged red cell study.
    C. Prepare for variceal sclerotherapy.
    D. Schedule for an upper endoscopy.
    E. Start fluid resuscitation.

Vomiting

Karen F. Murray and Dennis L. Christie

Pediatr. Rev. 1998;19;337
DOI: 10.1542/pir.19-10-337