# The Vomiting Patient

## Small Bowel Obstruction, Cyclic Vomiting, and Gastroparesis

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**KEYWORDS**

• Small bowel obstruction • Cyclic vomiting • Cannabinoid hyperemesis
• Gastroparesis • Prokinetic agents

**KEY POINTS**

- Small bowel obstructions represent 15% of emergency department visits for acute abdominal pain and can be associated with significant morbidity and mortality if unrecognized and untreated.
- Computed tomography scans have become the mainstay of diagnosis, and management should be designed to correct physiologic and electrolyte disturbances, allow bowel rest, and remove the source of the obstruction.
- Cyclic vomiting syndrome is a poorly understood condition characterized by recurrent episodes of intense vomiting, which is treated acutely with antiemetics, fluids, and electrolyte replacement, although, among adults, cannabinoid may represent a previously under-recognized cause.
- Gastroparesis is a chronic motility disorder of the stomach that involves delayed gastric emptying without evidence of mechanical obstruction.
- First-line therapy in the emergency department is the use of metoclopramide, but domperidone, erythromycin, and antiemetics are also often used, and interventional therapy should be reserved for refractory cases.

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## APPROACH TO VOMITING PATIENTS

Vomiting and abdominal pain are among the most common complaints for which patients present to the emergency department. Box 1 lists the differential diagnoses for vomiting. A thorough history, physical examination, and evaluation in the emergency department can help narrow the differential diagnosis for a more certain diagnosis.

**Box 1**
**Differential diagnosis for vomiting**

Abdominal causes
- Mechanical obstruction
- Motility disorders
- Acute appendicitis
- Acute cholecystitis
- Acute hepatitis
- Acute mesenteric ischemia
- Crohn disease
- Gastric and duodenal ulcer disease
- Pancreatitis and pancreatic neoplasms
- Peritonitis and peritoneal carcinomatosis
- Retroperitoneal and mesenteric disorders
- Acute cholecystitis
- Acute hepatitis
- Acute mesenteric ischemia
- Crohn disease
- Gastric and duodenal ulcer disease
- Pancreatitis and pancreatic neoplasms
- Peritonitis and peritoneal carcinomatosis
- Retroperitoneal and mesenteric disorders

Drugs

Infectious causes
- Acute gastroenteritis
- Systemic infections

Metabolic and endocrine causes
- Acute intermittent porphyria
- Addison disease
- Diabetic ketoacidosis
- Hypoparathyroidism/hyperparathyroidism
- Hyperthyroidism
- Pregnancy

Nervous system causes
- Demyelinating disorders
- Hydrocephalus
- Intracerebral lesions
- Labyrinthine disorders
- Meningitis
- Migraine headaches
- Seizure disorders

Other causes
- Anxiety and depression
- Cardiac disorders
- Collagen vascular diseases
- Paraneoplastic syndromes
- Postoperative states
- Eating disorders

This article focuses on 3 specific entities that have become increasingly prevalent: small bowel obstruction (SBO), cyclic vomiting, and gastroparesis.

## SMALL BOWEL OBSTRUCTION

### *Introduction*

SBO is one of the true emergencies encountered in the emergency department. It is defined as a failure of progression of food and bowel contents through the small intestine[1] and is secondary to functional or mechanical causes. In SBOs, the main concerns arise from systemic effects of electrolyte and fluid abnormalities and increased intestinal tract pressure.

A functional SBO is caused by an intestinal motility disorder. Typically, neurogenic causes lead to atony of intestinal muscles and malfunction of peristalsis, often referred to as adynamic or paralytic ileus.[2] The primary causes of functional SBOs are listed in **Box 2**. Although the exact cause of paralytic ileus is unknown, it is suspected to result from the synergistic effect of autonomic dysfunction, endocrine response, and inflammatory mediators.[3]

A mechanical obstruction occurs secondary to a physical impediment to the flow of intestinal matter as a result of intraluminal, intramural, and extramural causes.[1] This condition can be further classified as simple or complicated. A simple obstruction is caused by a blockage at 1 or 2 points of the intestine, without vascular compromise. A complicated or strangulated obstruction leads to intestinal ischemia. A partial obstruction occurs when gas or liquid stool is still capable of moving forward past a narrowing of the intestine.

SBOs are responsible for 15% of all emergency department visits for acute abdominal complaints.[2] Approximately 300,000 laparotomies are performed to relieve SBOs, costing the health care industry about $2.3 billion annually.[4,5] The incidence of SBOs in patients who have not had previous abdominal surgery is reported to be between 0.1% and 5% but those with previous surgery have an incidence as high as 15%.[2,6] This risk increases with each laparotomy that is performed, with recurrence rates as high as 30% at 30 years.[7]

Although 15% of partial SBOs require surgery, up to 85% of complete SBOs require surgery. The presence of ischemic bowel can increase mortality 10-fold. In the past 50 years, overall mortality from SBOs has decreased from 25% to 5%.[8]

### *Pathophysiology*

In SBOs, there is a disruption in the patency of the bowel, causing gradual accumulation of fluids. An initial increase in peristalsis produces an increase in intraluminal pressure. As the pressure approaches the systolic blood pressure, venous blood flow

**Box 2**
**Main causes of functional SBO**

- Abdominal surgery
- Major trauma
- Shock
- Infection
- Medications
- Metabolic derangements
- Renal colic

decreases to the bowel wall and adjacent mesentery, resulting in a decrease in absorption of fluids, electrolytes, and lymphatic drainage. This process eventually leads to ischemia and necrosis of the bowel with a mounting concern for perforation.[1] In a closed-loop obstruction, in which a segment of bowel is obstructed at 2 sites and rotates around an adhesion or hernia opening, this course is more sudden.[2]

SBOs can generate significant volume depletion and electrolyte abnormalities. Dehydration is the result of the prevention of reabsorption of intestinal contents from the colon, the loss of fluids because of vomiting and reduced intake, and progressive bowel wall edema. The most common electrolyte abnormalities with SBOs are hyponatremia and hypokalemia. Initially, metabolic alkalosis develops because of volume loss, reabsorption of bicarbonate, and a loss of chloride by the renal proximal tubule. However, as the bowel becomes more ischemic, metabolic acidosis may develop.[9,10] In addition, the stasis of intestinal matter can cause an overgrowth of bacterial intestinal flora proximal to the obstruction, causing feculent emesis. As the bowel infarcts, there is a translocation of bacteria and toxins across the bowel wall and eventual perforation.[11,12]

In a functional SBO, it is suspected that there is an activation of neural reflexes involving the sympathetic nervous system that impedes intestinal motility. Hormonal factors, such as vasoactive intestinal peptide, substance P, and nitrous oxide released during the postoperative period, also have an inhibitory effect on gastrointestinal motility. In addition, increases in the levels of inflammatory mediators, such as interleukin-1 and interleukin-6, help to potentiate decreased motility.[3,13,14]

In the United States, the predominant risk factor for mechanical SBOs is previous abdominal surgeries, which causes intra-abdominal adhesions. Surgeries most frequently implicated with SBOs are colorectal and gynecologic surgeries. Other risk factors include abdominal wall or groin hernias, malignancies, inflammatory bowel disease (specifically Crohn disease), and prior radiation (**Fig. 1**, **Table 1**).[2,10]

### *Presentation and Diagnosis*

Most patients with SBO typically have abdominal pain described as episodic and crampy, lasting seconds to minutes and located in the periumbilical area or diffusely.[1,2,15] If the patient begins to describe the pain as severe and constant, this may signal worsening intestinal ischemia.[2] Vomiting is also a common feature, with bilious vomitus present in proximal obstructions and more feculent vomitus in distal obstructions. Constipation and pain relief with vomiting have the highest specificity

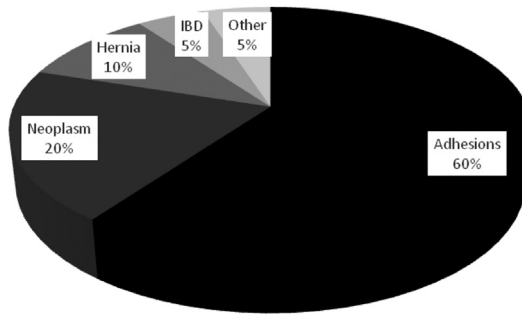


**Fig. 1.** Causes of mechanical SBOs. IBD, inflammatory bowel disease. (*Adapted from* Koch KL. Gastric neuromuscular function and neuromuscular disorders. In: Feldman M, Friedman LS, Brandt LJ, editors. Sleisenger and Fordtran's gastrointestinal and liver disease: pathophysiology/diagnosis/management. Philadelphia: Elsevier; 2010. p. 789–815.)

**Table 1**
**Causes of mechanical SBO**

| Intraluminal | Intramural | Extramural |
|---|---|---|
| Impaction | Congenital atresia | Adhesions |
| Foreign bodies/gallstones | Stricture | Hernia |
| Bezoars | Malignancy | Intussusception |

with SBOs.[16] Partial SBOs allow the passing of stool and flatus, but patients with complete SBOs may cease to have bowel movements and flatus.

In SBO, abdominal distention is the most reliable sign and can be present even early in the presentation.[16] On percussion, the abdomen may be tympanic with high-pitched bowel sounds.[1] Depending on the length of time of obstruction, the bowel sounds may be decreased. Abnormal bowel sounds are the second most reliable indicator of SBOs.[16] Tenderness on abdominal examination may vary from minimal to severe and may not be localized. The presence of localized tenderness may be a sign of ischemia or perforation, indicating more severe disease. Examination of the abdomen for any surgical scars may help to aid in the diagnosis.

The patient may also have signs of dehydration because SBO may lead to profound volume loss. These signs include tachycardia, hypotension, dry mucous membranes, and decreased urine output. A fever may also be present because of ischemia and resulting infection. A rectal examination should be performed, because it may help reveal impaction or a mass. If the examination reveals a positive guaiac stool or hematochezia, it may indicate ischemia, malignancy, or inflammation of the intestinal mucosa.

### *Evaluation in the Emergency Department*

Diagnostic strategies are needed to aid in the diagnoses, and computed tomography has become the most reliable imaging modality in the emergency department. **Table 2** describes the various imaging modalities that may be used in SBOs.[16,17]

Laboratory investigations should include a complete blood count and a basic metabolic profile. Leukocytosis may indicate translocation of bacteria, infection, or developing sepsis. If the levels are greater than 20,000/$mm^3$ or the patient has significant left shift, bowel necrosis, intra-abdominal abscess, or peritonitis should be suspected.[1] As the patient becomes more dehydrated, blood urea nitrogen and creatinine levels may become increased. Patients may also have hypokalemia or hyponatremia and may show hypochloremic metabolic alkalosis. An increased lactate level may indicate bowel ischemia. One small study of 162 patients showed that increased procalcitonin levels were predictive of bowel ischemia.[18]

### *Treatment*

Management of SBOs is 3-fold: correction of physiologic and electrolyte disturbances, bowel rest, and removing the source of the obstruction. First, resuscitation should be initiated to volume replete the patient. This resuscitation may require strict monitoring of urine output to assess the adequacy of resuscitation. Patients may need supplemental potassium because they may be significantly hypokalemic. If the patient shows fever or leukocytosis, antibiotics covering intra-abdominal flora and gram-negative and anaerobic bacteria are recommended.[2,8]

The second step to management involves bowel rest and conservative management. This step includes restricting the patient's oral intake in order to prevent further bowel distention. Gastrointestinal decompression with a nasogastric or orogastric tube may also be necessary but should be judged on a case-by-case basis. For

**Table 2**
**Imaging modalities used to diagnose SBOs**

| | Pros and Cons | Imaging Findings |
|---|---|---|
| Abdominal radiograph | • Should be initial evaluation<br>• Allows quick determination of perforation<br>• Positive predictive value of 80% in high-grade obstruction<br>• May be normal in early SBO<br>• 75% sensitivity, 66% specificity | • Dilated loops of bowel (>2.5 cm) with distal bowel collapse<br>• >2 air fluid levels<br>• Stomach may also be dilated<br>• Perforation may show free air |
| CT | • Most reliable test<br>• Optimal information with oral and intravenous contrast<br>• Can define cause and level of obstruction; ie, partial vs complete, strangulation or volvulus<br>• Can show transition point<br>• Slices 5–10 mm: 87% sensitivity, 81% specificity<br>• Increasing sensitivity and specificity with thinner slices and higher grade obstructions | • Dilated loops of bowel with distal collapse<br>• Air fluid levels<br>• Absence of contrast material in rectum<br>• Bowel wall thickening >3 mm<br>• Pneumatosis intestinalis and mesenteric fat stranding suggest necrosis and perforation |
| Contrast fluoroscopy | • Rarely performed<br>• Can help better delineate partial SBOs that are not clinically improving<br>• Water-soluble contrast may also be therapeutic in partial SBO<br>• Inferior to abdominal CT for closed-loop obstruction, ischemia, and determining cause<br>• Contraindicated if there are signs of strangulation<br>• If contrast reaches colon within 24 h, 96% sensitivity and 98% specificity in predicting resolution | • Dilated loops of proximal bowel highlighted with contrast material<br>• Diameter change at transition point<br>• No contrast distal to the obstruction |
| Ultrasonography | • Limited by poor visibility of gas-filled structures<br>• May be useful in patients who cannot have CT scans<br>• More sensitive and specific than radiographs, but cannot find grade, location, or cause<br>• In trained individuals, 75%–97% sensitivity and 75%–90% specificity for high-grade SBO | • Dilated loops of bowel<br>• Bowel wall thickening<br>• Increased intestinal contents<br>• Decreased peristalsis activity |
| MRI | • Limited by availability and time<br>• Requires a cooperative patient<br>• May be useful in patients who cannot have CT scans<br>• May better identify strictures in cases of recurrent SBOs<br>• Useful for low-grade bowel obstruction<br>• 92% sensitivity, 89% specificity | • Dilated bowel loops<br>• May show point of transition<br>• Hyperintensity of injured bowel |

*Abbreviation:* CT, computed tomography.

patients with high-grade or complete SBOs, gastrointestinal decompression may help to relieve abdominal distention and pain. It also helps to prevent further air swallowing and increased distention. In patients with a functional SBO, a nasogastric tube may not be necessary, and bowel rest is often enough.[1,2,8]

The final step involves relieving the obstruction, which often involves a trial of conservative management and depends on the cause of the obstruction. If the obstruction is thought to be secondary to adhesions, a laparoscopy or laparotomy is needed. There has been no difference in recurrence rates of SBO between laparoscopy versus laparotomy.[19] However, it is important to remember that further surgical trauma is a significant risk factor for recurrent SBOs. For an incarcerated hernia, if manual reduction is not possible, surgical intervention is needed. For malignant tumors, resection may be required. In the case of SBOs secondary to inflammatory bowel disease, bowel rest combined with high-dose steroids may help reduce the inflammation. All patients with complicated SBOs should have operative management. Surgery may be needed if patients have fever, leukocytosis, tachycardia, sepsis, lactic and metabolic acidosis, or worsening abdominal pain and peritonitis.

Conservative management is more successful in stable patients with partial SBOs. The success rate ranges from 40% to 70%.[20] However, if symptoms do not improve within 24 to 48 hours, operative management may be necessary. In one study comparing conservative versus operative treatment, patients treated operatively experienced a longer length of stay but had a lower rate of recurrence and longer time interval to recurrence.[21]

### *Summary*

SBOs remain a significant reason for emergency department visits and hospital admissions. Through early diagnosis and appropriate management, the morbidity and mortality associated with SBOs can be significantly reduced.

## CYCLIC VOMITING

### *Introduction*

Cyclic vomiting syndrome (CVS) has been described in children since 1882,[22] and is defined by recurrent, stereotypical episodes of vomiting with return to baseline health between episodes.[23] CVS has been increasingly recognized in adults as well,[24] and research interest accelerated with the 2004 description of cannabinoid hyperemesis (CH) as a cause of recurrent vomiting (**Fig. 2**).[25] Among children, the incidence of CVS has been estimated at 3.2 per 100,000 children per year,[26] with a prevalence of 1.9% reported in 2 separate studies.[27,28] The median age at onset is 4 to 7 years,[26–29] with an average delay in diagnosis of 3.1 years after symptom onset,[30] and a slight female predominance (55:45).[29] CVS of childhood persists into adulthood in about 30% of patients,[29,31,32] but may develop de novo in adults, with a mean age at diagnosis of 34.8 years after a delay in diagnosis of 7.9 years.[30]

### *Pathophysiology*

It is important to recognize that cyclic vomiting is a syndrome in the truest sense, defined by frequent co-occurrence of signs and symptoms rather than by shared mechanism. There is evidence that some cases of both pediatric and adult CVS may be related to undiagnosed mitochondrial dysfunction,[33,34] whereas other investigators attribute CVS to abdominal migraine because some cases are associated with migraine headaches.[32,35,36]

In the absence of a unifying pathophysiologic understanding of CVS, various professional societies have developed operational definitions and diagnostic approaches.[23,37]

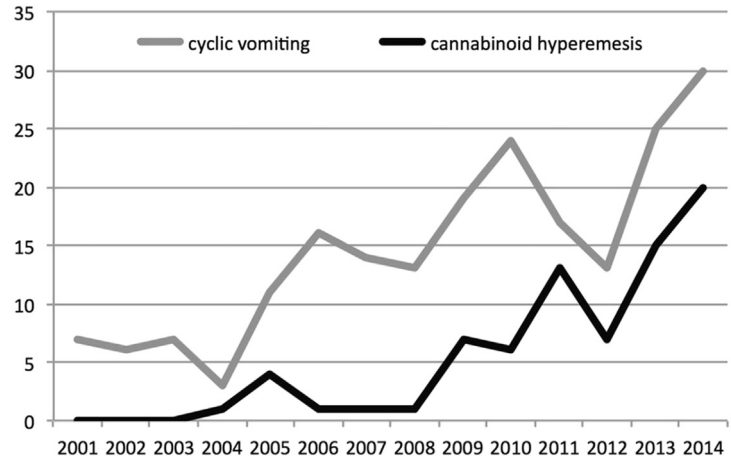


**Fig. 2.** PubMed search results by year for the terms "cyclic vomiting" and "cannabinoid hyperemesis."

Most prominently, the Rome process produced a definition and recommended management approach for CVS along with other functional gastrointestinal disorders (**Box 3**).[23] The North American Society for Pediatric Gastroenterology, Hepatology and Nutrition has published guidelines specific to the pediatric population that include recommendations concerning diagnosis, abortive and prophylactic therapies, and lifestyle modifications (**Box 4**).[37]

CH may represent a uniquely well-understood cause of adult CVS. CH is characterized by severe cyclic vomiting in the context of chronic heavy marijuana use and is associated with abdominal pain, compulsive hot water bathing behavior, and resolution of symptoms after cessation of use.[38] CH may be more common than was previously appreciated; an Internet survey of adult patients with CVS found that more than 80% used marijuana,[39] and cases have been reported in the pediatric population.[40] The prevalence of CH may increase with growing societal acceptance of cannabis use; diagnoses of CH doubled in Colorado following legalization of marijuana.[41]

### *Presentation and Diagnosis*

The typical presentation of CVS in children involves between 8 and 12 attacks per year,[26,29] lasting between 20 and 48 hours,[26,28,30,33] with complete resolution of symptoms between episodes. Vomiting is typically severe, with at least 4 emeses per

**Box 3**
**Rome III diagnostic criteria for cyclic vomiting syndrome**

Must include all of the following:
1. Stereotypical episodes of vomiting regarding onset (acute) and duration (<1 week)
2. Three or more discrete episodes in the prior year
3. Absence of nausea and vomiting between episodes

Supportive criterion: personal or family history of migraine headaches.

Criteria fulfilled for the last 3 months with symptom onset at least 6 months before diagnosis.
*Data from* Tack J, Talley NJ, Camilleri M, et al. Functional gastroduodenal disorders. Gastroenterology 2006;130:1466–79.

**Box 4**
**North American Society for Pediatric Gastroenterology, Hepatology and Nutrition (NASPGHAN) diagnostic criteria for cyclic vomiting syndrome in children**

Must include all of the following:

1. At least 5 attacks in any interval or a minimum of 3 attacks during a 6-month period
2. Episodic attacks of intense nausea and vomiting lasting 1 hour to 10 days and occurring at least 1 week apart
3. Vomiting during attacks occur at least 4 times per hour for at least 1 hour
4. Return to baseline health between episodes
5. Not attributed to another disorder

*Data from* Li BU, Lefevre F, Chelimsky GG, et al. North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition consensus statement on the diagnosis and management of cyclic vomiting syndrome. J Pediatr Gastroenterol Nutr 2008;47:379–93.

hour,[33,42] and frequently commences in the early morning hours.[29] Concurrent abdominal pain is noted among 71% of patients.[26] Emotional stress and viral infections have been identified as precipitants.[29,33] Among adults the typical presentation is similar, except that episodes are of longer median duration (2.0 vs 3.8 days).[30]

CVS is a diagnosis of exclusion. The differential diagnosis of CVS is listed in **Box 5**.

**Box 5**
**Differential diagnosis of cyclic vomiting syndrome (CVS) among children and adults**

Among children the differential diagnosis for CVS includes:

1. Primary gastrointestinal disorders (7%)
   a. Delayed presentation of intermittent volvulus associated with malrotation
   b. Gallbladder, liver, or pancreas disorders
2. Extra-abdominal disorders (5%)
   a. Intermittent hydronephrosis
   b. Diabetes mellitus
   c. Acute intermittent porphyria
   d. Intracranial neoplasms/masses/hydrocephalus
   e. Metabolic conditions, including disorders of mitochondrial function, fatty acid oxidation, the urea cycle, or organic and amino acids

These conditions may also exist in adults. Additional consideration should be given to:

1. Primary gastrointestinal disorders
   a. Gastroparesis
   b. Peptic ulcer disease
   c. Intermittent SBO
   d. Gallbladder disorder
   e. Pancreatitis
   f. Hepatitis
2. Extra-abdominal disorders
   a. Nephrolithiasis
   b. Intracranial neoplasms/masses/hydrocephalus
   c. Adrenal insufficiency

*Data from* Li BU, Lefevre F, Chelimsky GG, et al. North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition consensus statement on the diagnosis and management of cyclic vomiting syndrome. J Pediatr Gastroenterol Nutr 2008;47:379–93; and Abell T, Adams K, Boles R, et al. Cyclic vomiting syndrome in adults. Neurogastroenterol Motil 2008;20:269–84.

### *Emergency Department Evaluation*

As with all patients who present with abdominal pain, a careful history and physical examination are vital. Red flags include bilious emesis, abdominal tenderness, severe pain, or hematemesis. Additional testing in these patients may include urinalysis and abdominal ultrasonography to rule out ureteropelvic junction obstruction among children or nephrolithiasis in adults; acute abdominal series to rule out bowel obstruction; measurement of lipase levels, liver function tests, and gamma-glutamyl transferase levels to screen for pancreatitis, hepatitis, and gallbladder disease; with consideration of abdominal computed tomography scanning in lieu of or in addition to other imaging.

Among children, rare inborn errors of metabolism must be considered if fasting; if there is other illness or a high-protein meal is noted to provoke attacks; or if severe anion gap metabolic acidosis, altered mental status, or a peculiar odor are present.[37] Samples of blood and urine should be obtained during the attack before administration of carbohydrate-containing intravenous fluids. Serum ammonia level should be measured because urea cycle disorders are associated with increased serum ammonia levels while symptoms are present. Patients with possible delayed presentation of an inborn error of metabolism require hospitalization, because of the catastrophic outcome of these disorders if untreated.

Among children and adults, increased intracranial pressure secondary to obstructive hydrocephalus or intracranial mass may produce repetitive vomiting. Brain MRI is the test of choice given the limitations of computed tomography for evaluation of the posterior fossa. Rarely, temporal lobe epilepsy or other seizure disorders may cause cyclic vomiting, and thus electroencephalography and neurology consultation are appropriate in certain situations.

### *Treatment*

Emergency department treatment of CVS and CH is directed at controlling symptoms of nausea and vomiting, addressing volume depletion and electrolyte abnormalities, and determining need for inpatient management or subspecialist consultation. Antianxiety and analgesic medications may also play a role depending on the severity of anxiety and pain.

Patients should be placed in a darkened, quiet room. Intravenous 5-hydroxytryptamine (5-$HT_3$ [serotonin]) receptor antagonists, such as ondansetron, are the cornerstone of symptomatic treatment during acute attacks.[37] Promethazine and prochlorpromazine are less effective.[41,43] Other antiemetic agents have also been reported to be effective, including prokinetic agents such as metoclopramide and erythromycin,[44] although evidence is limited. Although ondansetron and prokinetic agents provide temporary relief, triptans (5-$HT_{1B/1D}$ agonists) have the potential to terminate an attack in migraine-associated cases.[37]

Initial resuscitation should be provided with isotonic crystalloid boluses until euvolemia is achieved and should be followed by dextrose-containing hypotonic maintenance fluids until the patient is able to tolerate oral intake. **Box 6** lists proposed indications for admission.[37] Some investigators advocate deep sedation and induced sleep with intravenous benzodiazepines,[45] although such an aggressive approach seems best reserved for refractory cases.

Acute kidney injury has been reported as a common complication of CH, perhaps in part caused by volume depletion associated with compulsive hot water bathing.[46] In addition to volume resuscitation and routine supportive care, haloperidol has been reported to improve symptoms among patients with CH who did not respond to

**Box 6**
**Proposed indications for hospitalization for patients with CVS**

Loss of greater than 5% of intravascular volume

Anuria for greater than 12 hours

Serum sodium level less than 130 mEq/L, anion gap greater than 18 mEq/L

Inability to control emesis

*Data from* Li BU, Lefevre F, Chelimsky GG, et al. North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition consensus statement on the diagnosis and management of cyclic vomiting syndrome. J Pediatr Gastroenterol Nutr 2008;47:379–93.

ondansetron and other antiemetics.[47] Patients should be counseled to abstain from cannabis use.

Long-term therapy for CVS is directed at identifying and avoiding precipitating factors, pharmacologic prophylaxis, migraine-specific therapies for migraine-associated CVS, and psychological support.[45] Tricyclic antidepressants, amitriptyline in particular, are the mainstay of pharmacologic prophylaxis.[48] Among children, β-blockers, such as propranolol, have also been used.[45,49]

### *Summary*

CVS is a condition of uncertain cause, defined by recurrent, stereotypical episodes of vomiting with return to baseline health between episodes. CH may account for a significant proportion of adult cases. Emergency department evaluation must be designed to identify red flags and rule out life-threatening alternative diagnoses. Treatment of acute episodes is primarily directed at symptom control, volume and electrolyte repletion, and arranging appropriate specialist follow-up.

## GASTROPARESIS

### *Introduction*

Gastroparesis is a chronic neuromuscular disorder of the upper gastrointestinal tract. It is characterized by chronic upper gastrointestinal symptoms, with objective evidence of delay in gastric emptying, in the absence of mechanical gastric outlet obstruction.[50] Characteristic symptoms of gastroparesis are described in **Table 3**.

Gastroparesis is estimated to affect up to 4% of the population and may produce mild, intermittent symptoms with little impairment of daily function to relentless vomiting with total disability and frequent hospitalizations.[51] A population-based study

**Table 3**
**Symptom profile of patients with gastroparesis**

| Symptom | Percentage of Patients |
|---|---|
| Nausea | 92 |
| Vomiting | 84 |
| Bloating | 75 |
| Early satiety | 60 |
| Abdominal pain | 46 |

*Data from* Parkman HP, Hasler WL, Fisher RS. American Gastroenterological Association technical review on the diagnosis and treatment of gastroparesis. Gastroenterology 2004;127:1592–622.

estimated that the age-adjusted incidence per 100,000 person-years for definite gastroparesis was 2.5 for men and 9.8 for women. The age-adjusted prevalence per 100,000 persons was 9.6 for men and 37.8 for women.[52] Hospitalizations with gastroparesis as a diagnosis more than doubled from 1995 to 2004, highlighting the importance of identifying and appropriately treating these patients when they present to the emergency department.[53]

### *Pathophysiology*

Gastroparesis is a consequence of many systemic illnesses; it may complicate selected surgical procedures, or it may be idiopathic. In one case series, 29% of cases had underlying diabetes, 13% occurred after gastric surgery, and 36% were idiopathic (**Table 4**).[54] Of the idiopathic cases, postinfectious gastroparesis represented 21% of cases.[55]

Disruption of the normal physiology of gastric emptying (**Fig. 3**) caused by an abnormality in the smooth muscles, the enteric nervous system, the interstitial cells of Cajal, and the extrinsic innervation from the autonomic nervous system leads to delays in gastric emptying and the development of gastroparesis.[56]

### *Presentation and Diagnosis*

Patients with suspected gastroparesis usually present with several nonspecific abdominal complaints. Symptoms typically include nausea, vomiting, bloating, early satiety, and abdominal pain.[54] The abdominal pain is often described as burning, vague, or crampy, with some patients localizing it to the epigastrium.[57] Sharp, well-localized pain is not characteristic, and other causes need to be ruled out in these situations.[58] Initial laboratory testing is not generally useful in diagnosing patients with gastroparesis. However, routine blood tests can help rule out other diagnoses. Diagnostic evaluation generally requires an esophagogastroduodenoscopy initially to rule out mechanical obstruction. If endoscopy is negative, patients require additional testing to assess their rate of gastric emptying.

Gastric emptying scintigraphy is currently the gold standard for measuring motility of the stomach.[52] Consensus standards for gastric emptying scintigraphy have been published by multiple societies.[59] Delayed gastric emptying is present if there is greater than 90% gastric retention at 1 hour, greater than 60% at 2 hours, and greater

**Table 4**
**Causes of gastroparesis**

| Major Causes (%) | Less Common Causes |
|---|---|
| Diabetes mellitus (29) | Connective tissue disease |
| Post-surgical (13) | Ischemia |
| Idiopathic (36) | Cancer |
| | Neurologic disease (eg, Parkinson) |
| | Eating disorders |
| | Metabolic/endocrine conditions |
| | Medications (eg, anticholinergics, calcium channel blockers, and opiates) |
| | Critical illness |

*Data from* Soykan I, Sivri B, Sarosiek I, et al. Demography, clinical characteristics, psychological and abuse profiles, treatment, and long-term follow-up of patients with gastroparesis. Dig Dis Sci 1998;43:2398–404; and Bityutskiy LP, Soykan I, McCallum RW. Viral gastroparesis: a subgroup of idiopathic gastroparesis–clinical characteristics and long-term outcomes. Am J Gastroenterol 1997;92:1501–4.

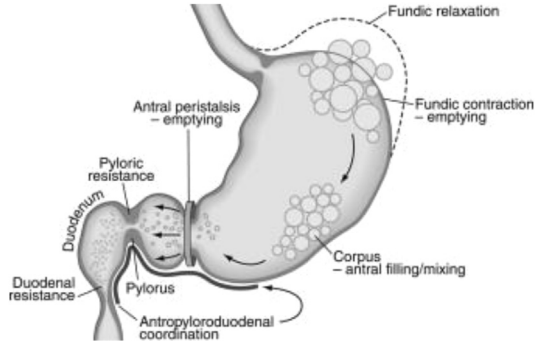


**Fig. 3.** Normal neuromuscular activity of the stomach in response to ingestion of food. (*Data from* Koch KL, Calles-Escandon J. Diabetic gastroparesis. Gastroenterol Clin North Am 2015;44:41; with permission.)

than 10% at 4 hours.[59,60] Other tests that measure delayed gastric emptying include wireless capsule motility, antroduodenal manometry, breath testing, transabdominal ultrasonography, and MRI.[61]

### *Emergency Department Evaluation*

Although the diagnosis of gastroparesis is not typically made in the emergency department, an emergency department evaluation can help identify the severity of the disease and associated complications. Patients with mild gastroparesis often present with intermittent symptoms that worsen after a large solid meal. In severe cases, patients often complain of progressive nausea, distention, and pain that is relieved by vomiting old food residue. The history should also elicit existing comorbidities (eg, diabetes or scleroderma), prior gastric surgery, abdominal irradiation, or a recent viral illness.[51]

The physical examination in these patients can serve 2 purposes: to assess the severity of the presenting complaints and to facilitate diagnosis. Poor skin turgor, sunken eyes, dry mucous membranes, and orthostatic vital signs mandate prompt fluid resuscitation. Patients can also have abdominal distention and tympany on examination with or without abdominal tenderness. The clinician may also be able to elicit a succussion splash by gently rocking the patient from side to side.

Selected laboratory tests and radiologic studies may help direct further management of patients with presumed gastroparesis. Serum electrolyte levels can be used to assess for hypokalemia and contraction alkalosis. Tests for diabetes, uremia, and thyroid or parathyroid dysfunction are indicated in certain cases. Abdominal radiographs help rule out entities such as an SBO. If available, endoscopy may reveal mucosal lesions, such as reflux or candida esophagitis, which require treatments other than those typically used for treatment of gastroparesis.[62]

### *Treatment*

Treatment of gastroparesis often includes dietary modifications, pharmacotherapy, and interventional therapy. Dietary modifications generally involve altering the meal content and frequency. Patients should be encouraged to eat more liquid-based meals, because they often have intact gastric emptying of liquids. Intake of fats and nondigestible fibers should be reduced, because they retard gastric emptying through various mechanisms.[63]

Management typically involves the use of pharmacotherapy. Nonetheless, treatment options should also include hydration with correction of electrolyte abnormalities and identification and treatment of the underlying disorder. However, the main aim of treatment is to alleviate the symptoms with medications (**Table 5**).

***Metoclopramide***

At present, metoclopramide is the only medication that is approved for the use of gastroparesis in the United States and should be considered the first-line treatment. Metoclopramide is a dopamine receptor antagonist, thereby stimulating the cholinergic receptors. This stimulation results in a reduction of esophageal sphincter and gastric tone, increased intragastric pressure, improved antroduodenal coordination, and accelerated gastric emptying.[64] Metoclopramide improved gastric emptying by 56% in patients with gastroparesis, compared with 37% in the placebo group.[65] However, multiple short-term studies generally showed a poor correlation of acceleration of gastric emptying with symptom improvement.[64,66–70] In addition, the US Food and Drug Administration has placed a black box warning on the use of metoclopramide. Acute dystonic reactions can occur with use of this medication, as well as irreversible tardive dyskinesia.[71]

***Domperidone***

Domperidone is approved only on an investigational basis in the United States. It is also a dopamine receptor antagonist. However, because it does not cross the blood-brain barrier, the central nervous system side effects are less evident. In addition, domperidone and metoclopramide have been shown to be equally efficacious in improving symptoms of gastroparesis.[67,72]

***Erythromycin***

Erythromycin, a macrolide antibiotic, acts as a motilin receptor agonist. It mimics the effects of motilin, a polypeptide involved in gastric smooth muscle contractions, and promotes gastric emptying.[73,74] Long-term use of erythromycin leads to tachyphylaxis and other complications of prolonged antibiotic use,[75,76] and thus there are an inadequate number of clinical trials evaluating the use of erythromycin for long-term treatment of gastroparesis.

***Antiemetic agents***

Given the lack of correlation between symptoms and gastric emptying, it is reasonable to propose that the primary goal of treating gastroparesis should focus on symptom relief. Because nausea and vomiting are the most common symptoms, medications targeted for symptomatic relief of these symptoms are often used. These medications work through a variety of peripheral and central pathways. **Box 7** lists these agents. To date, there have not been any controlled clinical studies formally evaluating nonprokinetic antiemetics in gastroparesis.[56]

***Interventional therapy***

Patients who fail medical therapy and are unable to meet their nutritional requirements should be considered for endoscopic and surgical options.[50] Endoscopic treatment involves onabotulinumtoxinA injections into the pyloric sphincter. Although earlier studies revealed a temporary improvement in symptoms, later studies have not been as promising.[77–80] Placement of a jejunostomy tube can be performed in patients with severe refractory gastroparesis. In a retrospective study, 39% of patients reported fewer symptoms, 52% reported fewer hospitalizations, 56% reported better nutritional status, and 83% reported overall improvement in their health.[81] The Food and Drug Administration approved gastric electrical stimulation as a surgical option

**Table 5**
**Primary prokinetic agents used in the treatment of gastroparesis**

| Medication | Main Mechanism | Starting Oral Dose (mg) | Main Adverse Effects | Comments |
|---|---|---|---|---|
| Metoclopramide | Central and peripheral dopamine-2 receptor antagonist | 10 TID and at bedtime | Extrapyramidal movement disorders (ie, tardive dyskinesia), hyperprolactinemia | Only FDA-approved drug for gastroparesis |
| Domperidone | Peripheral dopamine-2 receptor antagonist | 10 TID and at bedtime | Hyperprolactinemia | Only available through investigational program in the United States |
| Erythromycin | Motilin receptor agonist | 125 BID | Gastrointestinal upset, arrhythmias, and drug interactions | Macrolide antibiotic with antimicrobial properties |

*Abbreviations:* BID, twice daily; FDA, US Food and Drug Administration; TID, 3 times daily.
*Data from* Tang DM, Friedenberg FK. Gastroparesis: approach, diagnostic evaluation, and management. Dis Mon 2011;57:74–101.

**Box 7**
**Primary antiemetic agents used in gastroparesis**

*Phenothiazine derivatives*

Prochlorperazine

*Serotonin (5-hydroxytryptamine [5-$HT_3$]) receptor antagonists*

Ondansetron

*Dopamine receptor antagonists*

Metoclopramide

Domperidone

*Histamine H1 receptor antagonists*

Diphenhydramine

Promethazine

Meclizine

*Benzodiazepines*

Lorazepam

*Data from* Tang DM, Friedenberg FK. Gastroparesis: approach, diagnostic evaluation, and management. Dis Mon 2011;57:74–101.

in 2000 for the treatment of refractory gastroparesis. It has been shown to significantly decrease gastrointestinal symptoms and improve quality of life, even over the long term.[82,83]

### *Summary*

Gastroparesis is a chronic motility disorder. The most common causes include diabetes, postsurgical causes, and postinfectious causes. Fig. 4 shows the general approach to these patients. They often present to the emergency department complaining of nausea, vomiting, bloating, early satiety, and abdominal pain. Evaluation should be designed to assess the severity of their symptoms. After restoration of fluid and electrolyte disturbances and glucose control, the mainstay of therapy is the use of the prokinetic agent metoclopramide. Treatment with domperidone, erythromycin, and antiemetics is also often used. Patients who have refractory gastroparesis should be considered for hospitalization to evaluate for interventional therapy.

## SUMMARY: PEARLS AND PITFALLS

### *Small Bowel Obstruction*

- Through early diagnosis and appropriate management, the morbidity and mortality associated with SBOs can be significantly reduced.
- Computed tomography has become the most reliable imaging modality in the emergency department.
- Management of SBOs is 3-fold: correction of physiologic and electrolyte disturbances, bowel rest, and removing the source of the obstruction.

### *Cyclic Vomiting*

- CH may represent a uniquely well-understood cause of adult CVS.
- CVS is a diagnosis of exclusion, and emergency department evaluation must be designed to identify red flags and rule out life-threatening alternative diagnoses.

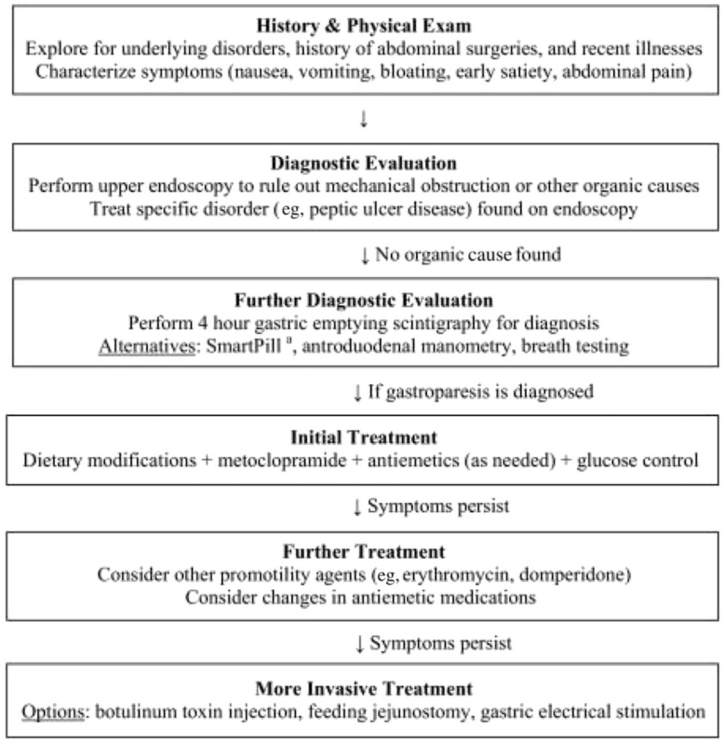


**Fig. 4.** General approach to gastroparesis. [a] SmartPill Corporation, Buffalo, NY. (*From* Tang DM, Friedenberg FK. Gastroparesis: approach, diagnostic evaluation, and management. Dis Mon 2011;57:86; with permission.)

- Treatment of acute episodes is primarily directed at symptom control, volume and electrolyte repletion, and arranging appropriate specialist follow-up.

### *Gastroparesis*

- Gastroparesis is a chronic motility disorder, often associated with diabetic patients, postsurgical patients, and postinfectious patients.
- Evaluation should be designed to assess the severity of the patient's symptoms. After restoration of fluid and electrolyte disturbances and glucose control, the mainstay of therapy is the use of the prokinetic agent, metoclopramide.
- Patients with refractory gastroparesis should be considered for interventional therapy.

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