surgery, especially those having had preoperative gastric outlet obstruction as a complication of PUD, are also commonly affected by gastroparesis. Finally, Parkinson's disease, rheumatologic disorders, hypothyroidism or hyperthyroidism, chronic intestinal pseudo-obstruction, and a variety of paraneoplastic syndromes can also produce gastroparesis.

The diagnostic evaluation of delayed gastric emptying should focus on excluding structural and metabolic abnormalities. Endoscopy is the preferred initial test to rule out mechanical gastric outlet obstruction, and a CT enterography or capsule endocopic study may be useful to exclude small bowel lesions. Serum electrolytes, blood cell counts, and thyroid studies should also be performed. When these studies are negative, radionuclide scintigraphy (gastric-emptying scan) using a mixed solid-liquid meal can quantitate delayed gastric emptying. Assessment of solid emptying is more clinically relevant than liquid emptying. In especially difficult cases, GI manometry and electrogastrography may help in the diagnosis.

Managing gastroparesis begins with identifying and treating potentially correctable causes. Medications that reduce gastric emptying, such as narcotics, anticholinergics, and tricyclic antidepressants, should be avoided. Because liquids empty easier than solids, and because liquid emptying is often preserved in patients with gastroparesis, simple dietary modifications may be helpful in treatment. The diet should be modified to include blenderized foods and liquid supplements. High-fat and fiber-rich foods should be avoided because they inhibit gastric emptying under normal conditions and are less likely to empty. Medical options are limited and involve the use of prokinetic drugs, which are agents that improve transit in the GI tract.

Metoclopramide is a dopamine-2 receptor antagonist that also facilitates the release of acetylcholine from cholinergic nerve terminals in the gut, thereby accelerating gastric emptying. The efficacy of metoclopramide is inconsistent, and adverse effects and the development of tolerance complicate long-term therapy. Adverse effects occur in up to 20% of patients and include drowsiness, anxiety, fatigue, insomnia, restlessness, agitation, extrapyramidal effects, galactorrhea, and menstrual irregularities. The typical dosage is 10 mg, 20 to 30 minutes before meals and at bedtime, although doses as high as 80 mg or as low as 20 mg may be used daily. Doses should be reduced for patients with renal failure. Domperidone, another dopamine receptor antagonist with prokinetic properties, has similar efficacy to metoclopramide in the treatment of delayed gastric emptying but is currently not available in the United States.

Erythromycin is a macrolide antibiotic that stimulates smooth muscle motilin receptors located at all levels of the GI tract. The prokinetic effects of erythromycin are related to its ability to mimic the effect of the GI peptide motilin to stimulate smooth muscle contraction, which accounts for the acceleration of solid and liquid gastric emptying. Erythromycin may dramatically improve gastric emptying in patients with severe diabetic gastroparesis when given acutely at an intravenous dose of 1 to 3 mg/kg every 8 hours. Long term use of the drug at a dose of 250 to 500 mg orally every 8 hours in patients with gastric stasis is of limited efficacy because of tachyphylaxis and side effects.

Endoscopic botulinum toxin A injection into the pyloric sphincter has also been reported in the treatment of delayed gastric emptying in small studies, but larger clinical trials have not shown this procedure to be effective.

In patients who are refractory to these measures, surgical placement of a jejunal tube, with or without a venting gastrostomy, may be necessary. Total parenteral nutrition is rarely indicated. Surgical gastrectomy should only be considered in patients with refractory postsurgical gastric stasis. Gastric pacemakers and other prokinetics, specifically new serotonin-receptor agonists, are under investigation and may be options in the future.

RAPID GASTRIC EMPTYING

Rapid gastric emptying is a far less common clinical problem than delayed gastric emptying. *Dumping syndrome* describes the alimentary and systemic manifestations of early delivery of large amounts of osmotically active food to the small intestine. Dumping syndrome is usually seen when the normal reservoir, grinding, and sieving properties of the stomach are disrupted, most commonly following surgery for obesity (Roux-en-Y gastric bypass) or PUD. The accelerated emptying of hypertonic boluses of nutrient material into the small intestine results in splanchnic vasodilation and release of vasoactive peptides. Early dumping symptoms, occurring about 30 minutes after a meal, include epigastric fullness and pain, nausea, vomiting, early satiety, and vasomotor features such as flushing, palpitations, and diaphoresis. Later symptoms, such as diaphoresis, tremulousness, and weakness, occur about 2 hours after a meal and may be caused by hypoglycemia from rebound hyperinsulinemia. Treatment of dumping syndrome involves dietary manipulation to decrease the volume and osmotic load emptied into the intestine. Frequent small feedings of meals low in carbohydrates, separation of liquid and solid intake, and avoidance of hypertonic fluids and lactose are usually helpful. When these measures fail, administration of octreotide at a dose of 25 to 50 mcg subcutaneously 30 minutes before meals may be helpful. Octreotide acts by slowing gastric emptying and intestinal transit as well as by inhibiting the release of insulin. Surgical procedures to slow gastric emptying have limited success.

GASTRIC VOLVULUS

Gastric volvulus occurs when the stomach rotates within the abdominal or thoracic cavity, compromising its emptying and its vascular supply. This event may be transient, producing few if any symptoms, or may lead to obstruction or even ischemia and necrosis. *Primary gastric volvulus*, seen in one third of the patients, occurs below the diaphragm when the stabilizing ligaments are too lax as a result of congenital or acquired causes. *Secondary gastric volvulus* occurs above the diaphragm in association with paraesophageal hernias or other diaphragmatic defects. Acute gastric volvulus produces sudden, severe pain of the upper abdomen or chest, and persistent retching producing scant vomitus; this is often associated with the inability to pass a nasogastric tube by medical personnel. This combination of symptoms, also known as Borchardt triad, should lead to a strong clinical suggestion of acute gastric volvulus. Chronic gastric volvulus may be associated with mild and nonspecific symptoms, such as epigastric discomfort, heartburn, abdominal fullness or bloating, and borborygmi, especially after meals. The diagnosis of gastric volvulus is made by upper GI series demonstrating an