Anatomic studies may be indicated if initial testing is nondiagnostic. Upper endoscopy detects ulcers, malignancy, and retained gastric food residue in gastroparesis. Small-bowel barium radiography or computed tomography (CT) diagnoses partial bowel obstruction. Colonoscopy or contrast enema radiography detects colonic obstruction. Ultrasound or CT defines intraperitoneal inflammation; CT and magnetic resonance imaging (MRI) enterography provide superior definition of inflammation in Crohn's disease. CT or MRI of the head can delineate intracranial disease. Mesenteric angiography, CT, or MRI is useful for suspected ischemia.

Gastrointestinal motility testing may detect an underlying motor disorder when anatomic abnormalities are absent. Gastroparesis commonly is diagnosed by gastric scintigraphy, by which emptying of a radiolabeled meal is measured. Isotopic breath tests and wireless motility capsule methods are alternatives tests to define gastroparesis in different regions of the world. Intestinal pseudoobstruction often is suggested by abnormal barium transit and luminal dilation on small-bowel contrast radiography. Delayed small-bowel transit also may be detected by wireless capsule techniques. Small-intestinal manometry can confirm the diagnosis and further characterize the motor abnormality as neuropathic or myopathic based on contractile patterns. Such investigation can obviate the need for surgical intestinal biopsy to evaluate for smooth muscle or neuronal degeneration. Combined ambulatory esophageal pH/impedance testing and high-resolution manometry can facilitate diagnosis of rumination syndrome.

TREATMENT NAUSEA AND VOMITING

GENERAL PRINCIPLES

Therapy of vomiting is tailored to correcting remediable abnormalities if possible. Hospitalization is considered for severe dehydration, especially if oral fluid replenishment cannot be sustained. Once oral intake is tolerated, nutrients are restarted with low-fat liquids, because lipids delay gastric emptying. Foods high in indigestible residue are avoided because these prolong gastric retention. Controlling blood glucose in poorly controlled diabetics can reduce hospitalizations in gastroparesis.

ANTIEMETIC MEDICATIONS

The most commonly used antiemetic agents act on central nervous system sites (Table 54-2). Antihistamines like dimenhydrinate and meclizine and anticholinergics like scopolamine act on labyrinthine pathways to treat motion sickness and inner ear disorders. Dopamine D_2 antagonists treat emesis evoked by area postrema stimuli and are used for medication, toxic, and metabolic etiologies. Dopamine antagonists cross the blood-brain barrier and cause anxiety, movement disorders, and hyperprolactinemic effects (galactorrhea, sexual dysfunction).

Other classes exhibit antiemetic properties. 5-HT_3 antagonists such as ondansetron and granisetron can prevent postoperative vomiting, radiation therapy–induced symptoms, and cancer chemotherapy–induced emesis, but also are used for other causes of emesis with limited evidence for efficacy. Tricyclic antidepressant agents provide symptomatic benefit in patients with chronic idiopathic nausea and functional vomiting as well as in long-standing diabetic patients with nausea and vomiting. Other antidepressants such as mirtazapine and olanzapine also may exhibit antiemetic effects.

GASTROINTESTINAL MOTOR STIMULANTS

Drugs that stimulate gastric emptying are used for gastroparesis (Table 54-2). Metoclopramide, a combined 5-HT_4 agonist and D_2 antagonist, is effective in gastroparesis, but antidopaminergic side effects, such as dystonias and mood and sleep disturbances, limit use in ~25% of cases. Erythromycin increases gastroduodenal motility by action on receptors for motilin, an endogenous stimulant of fasting motor activity. Intravenous erythromycin is useful for inpatients with refractory gastroparesis, but oral forms have some utility. Domperidone, a D_2 antagonist not available in the United States, exhibits prokinetic and antiemetic effects but does not cross into most brain regions; thus, anxiety and dystonic reactions are rare. The main side effects of domperidone relate to induction of

TABLE 54-2 TREATMENT OF NAUSEA AND VOMITING

Treatment	Mechanism	Examples	Clinical Indications
Antiemetic agents	Antihistaminergic	Dimenhydrinate, meclizine	Motion sickness, inner ear disease
	Anticholinergic	Scopolamine	Motion sickness, inner ear disease
	Antidopaminergic	Prochlorperazine, thiethylperazine	Medication-, toxin-, or metabolic-induced emesis
	5-HT_3 antagonist	Ondansetron, granisetron	Chemotherapy- and radiation-induced emesis, postoperative emesis
	NK_1 antagonist	Aprepitant	Chemotherapy-induced nausea and vomiting
	Tricyclic antidepressant	Amitriptyline, nortriptyline	Functional vomiting, chronic idiopathic nausea, cyclic vomiting syndrome, ?gastroparesis
	Other antidepressant	Mirtazapine, olanzapine	?Functional vomiting, ?chronic idiopathic nausea, ?gastroparesis
Prokinetic agents	5-HT_4 agonist and antidopaminergic	Metoclopramide	Gastroparesis
	Motilin agonist	Erythromycin	Gastroparesis, ?intestinal pseudoobstruction
	Peripheral antidopaminergic	Domperidone	Gastroparesis
	Somatostatin analogue	Octreotide	Intestinal pseudoobstruction
	Acetylcholinesterase inhibitor	Pyridostigmine	?Small-intestinal dysmotility/pseudoobstruction
Special settings	Benzodiazepines	Lorazepam	Anticipatory nausea and vomiting with chemotherapy
	Glucocorticoids	Methylprednisolone, dexamethasone	Chemotherapy-induced emesis
	Cannabinoids	Tetrahydrocannabinol	Chemotherapy-induced emesis

Note: ?, indication is uncertain.