Drugs Used to Treat Gastroesophageal Reflux and Peptic Ulcer Disease

chapter 33

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Objectives

1. Describe the physiology of the stomach.
2. Cite common stomach disorders that require drug therapy.
3. Identify factors that prevent breakdown of the body's normal defense barriers resulting in ulcer formation.
4. Discuss the drug classifications and actions used to treat stomach disorders.
5. Identify interventions that incorporate pharmacologic and nonpharmacologic treatments for an individual with stomach disorders.

Key Terms

parietal cells (pă-RĪ-ĕ-tŭl) (p. 519)
hydrochloric acid (hī-drō-KLŌR-īk) (p. 519)
gastroesophageal reflux disease (GERD) (găs-trō-ēs-ŏf-ĕ-JĒ-ŭl RĒ-flŭks) (p. 519)
heartburn (HĂRT-bŭrn) (p. 519)
peptic ulcer disease (PUD) (PĔP-tĭk ŬL-sŭr) (p. 520)
Helicobacter pylori (hĕl-ĭ-kō-BĂK-tŭr pī-LŌR-ē) (p. 520)

PHYSIOLOGY OF THE STOMACH

As a major part of the gastrointestinal (GI) tract, the stomach has three primary functions: (1) storing food until it can be processed in the lower GI tract; (2) mixing food with gastric secretions until it is a partially digested, semisolid mixture known as chyme; and (3) slowly emptying the stomach at a rate that allows proper digestion and absorption of nutrients and medicine from the small intestine.

Three types of secretory cells line portions of the stomach—chief, parietal, and mucus cells. The chief cells secrete pepsinogen, an inactive enzyme. **Parietal cells** are stimulated by acetylcholine from cholinergic nerve fibers, gastrin, and histamine to secrete **hydrochloric acid,** which activates pepsinogen to pepsin and provides the optimal pH for pepsin to start protein digestion. Normal pH in the stomach ranges from 1 to 5, depending on the presence of food and medications. Hydrochloric acid also breaks down muscle fibers and connective tissue ingested as food and kills bacteria that enter the digestive tract through the mouth. The parietal cells also secrete intrinsic factor needed for absorption of vitamin B_{12}. The mucus cells secrete mucus, which coats the stomach wall. The 1-mm-thick coat is alkaline and protects the stomach wall from damage by hydrochloric acid and the digestive enzyme pepsin. It also contributes lubrication for food transport. Small amounts of other enzymes are also secreted in the stomach. Lipases digest fats, and gastric amylase digests carbohydrates. Other digestive enzymes are also carried into the stomach from swallowed saliva.

Prostaglandins play a major role in protecting the stomach walls from injury by stomach acids and enzymes. Prostaglandins are produced by cells lining the stomach and prevent injury by inhibiting gastric acid secretion, maintaining blood flow, and stimulating mucus and bicarbonate production.

COMMON STOMACH DISORDERS

Gastroesophageal reflux disease (GERD), more commonly referred to as **heartburn,** acid indigestion, or sour stomach, is a common stomach disorder. Approximately one third of the U.S. population experiences heartburn once each month, and 5% to 7% have heartburn daily. Common symptoms are a burning sensation, bloating, belching, and regurgitation. Other symptoms that are reported less frequently are nausea, a "lump in the throat," hiccups, and chest pain.

GERD is the reflux of gastric secretions, primarily pepsin and hydrochloric acid, up into the esophagus. Causes of GERD are a weakened lower esophageal sphincter, delayed gastric emptying, hiatal hernia, obesity, overeating, tight-fitting clothing, and increased acid secretion. Acid secretions are increased by smoking, alcohol, carbonated beverages, coffee, and spicy foods.

Most cases of GERD pass quickly with only mild discomfort, but frequent or prolonged bouts of acid reflux cause inflammation, tissue erosion, and ulcerations in the lower esophagus. Anyone who has recurrent or continuous symptoms of reflux, especially if the symptoms interfere with activities, should be referred to a health care provider. These symptoms may also accompany more serious conditions, such as ischemic heart disease, scleroderma, and gastric malignancy.

Clinical Pitfall

Nurses need to be aware that the symptoms of gastroesophageal reflux disease (GERD) may also accompany more serious conditions, such as ischemic heart disease, scleroderma, and gastric malignancy. It is important to do a thorough physical assessment whenever a patient presents with heartburn or acid indigestion and not simply dismiss the symptoms as gastrointestinal in origin.

Peptic ulcer disease (PUD) refers to several stomach disorders that result from an imbalance between acidic stomach contents and the body's normal defense barriers, causing ulcerations in the GI tract. The most common illnesses are gastric and duodenal ulcers. It is estimated that approximately 10% of all Americans will develop an ulcer at some time in their lives. The incidence in men and women is approximately the same. Race, economic status, and psychological stress do not correlate with the frequency of ulcer disease. Often, the only symptom reported is epigastric pain, described as burning, gnawing, or aching. Patients often report that varying degrees of pain are present for a few weeks and are then gone, only to recur a few weeks later. The pain is most often noted when the stomach is empty, such as at night or between meals, and is relieved by food or antacids. Other symptoms that cause patients to seek medical attention are bloating, nausea, vomiting, and anorexia.

Ulcers appear to be caused by a combination of acid and a breakdown in the body's defense mechanisms that protect the stomach wall. Proposed mechanisms are oversecretion of hydrochloric acid by excessive numbers of parietal cells, injury to the mucosal barrier such as that resulting from prostaglandin inhibitors (nonsteroidal anti-inflammatory drugs [NSAIDs], including aspirin), and infection of the mucosal wall by ***Helicobacter pylori***. It had been thought that no bacterium could survive in the highly acidic environment of the stomach; however, *H. pylori* was first isolated from patients with gastritis in 1983. The bacterium seems able to live below the mucus barrier, where it is protected from stomach acid and pepsin. *H. pylori* is now thought to be associated with as many as 90% of duodenal and 70% of gastric ulcers. The exact mechanism whereby *H. pylori* contributes to ulcer formation is not known, but several hypotheses are being tested.

Several risk factors increase the likelihood of peptic ulcer disease:

1. There seems to be a genetic predisposition to PUD. Some families have a much greater history of PUD than others.
2. It is a commonly held belief that stress causes ulcers, but no well-controlled studies have supported this.
3. Cigarette smoking increases acid secretion, alters blood flow in the stomach wall, and retards prostaglandin synthesis needed for defense mechanisms.
4. NSAIDs have a twofold effect: they inhibit prostaglandins that protect the mucosa and directly irritate the stomach wall. Once ulcerations have formed, NSAIDs also slow healing.
5. It is commonly thought that certain foods (e.g., spicy foods) and alcohol contribute to ulcer formation. It is true that certain foods increase acid secretion and that alcohol irritates the stomach lining, but results from studies have not corroborated this concept.

GOALS OF TREATMENT

The goals of treatment of GERD are to relieve symptoms, decrease the frequency and duration of reflux, heal tissue injury, and prevent recurrence. The most important treatment is a change in lifestyle, which includes losing weight (if significantly over the ideal body weight), reducing or avoiding foods and beverages that increase acid production, reducing or stopping smoking, avoiding alcohol, and consuming smaller meals. Additional therapy includes remaining upright for 2 hours after meals, not eating before bedtime, and avoiding tight clothing over the abdominal area. Lozenges may be used to increase saliva production, and antacids and alginic acid therapy may provide relief for patients who experience infrequent heartburn. If the patient's symptoms do not improve within 2 to 3 weeks, or if the condition is severe, additional pharmacologic measures should be tried to reduce irritation. About 5% to 10% of patients with GERD require surgery.

The treatment of PUD and GERD is somewhat similar: relieve symptoms, promote healing, and prevent recurrence. Lifestyle changes that eliminate risk factors, such as cigarette smoking and foods (and alcohol) that increase acid secretion, should be initiated. Patients rarely need to be restricted to a bland diet. If NSAIDs are being taken, consideration should be given to switching to acetaminophen if feasible. For decades, ulcer treatment focused on reducing acid secretions (anticholinergic agents, H2 antagonists, gastric acid pump inhibitors), neutralizing acid (antacids), or coating ulcer craters to hasten healing (sucralfate). Major changes in therapy have come about because the U.S. Food and Drug Administration (FDA) has approved antibiotics to eradicate *H. pylori*. Several large studies are under way to refine the healing and reduce ulcer recurrence rate. Various combinations of antimicrobial agents (e.g., amoxicillin, tetracycline, metronidazole, clarithromycin), bismuth, and antisecretory agents (e.g., H2 antagonists, proton pump inhibitors) are used to eradicate *H. pylori*. Antibiotics are not recommended for individuals who are asymptomatic with *H. pylori* because there is concern that resistant strains of bacteria may develop.

DRUG THERAPY

ACTIONS

- Antacids neutralize gastric acid, thereby causing the gastric contents to be less acidic.
- Coating agents provide a protective covering over the ulcer crater.
- H2 antagonists decrease the volume of hydrochloric acid produced, which increases the gastric pH and thereby results in decreased irritation to the gastric mucosa.
- Proton pump inhibitors block the formation of hydrochloric acid, reducing irritation of the gastric mucosa.
- Prokinetic agents increase the lower esophageal sphincter muscle pressure and peristalsis, hastening emptying of the stomach to reduce reflux.
- Antispasmodic agents reduce the secretion of saliva, hydrochloric acid, pepsin, bile, and other enzymatic fluids necessary for digestion, and decrease GI motility and secretions.

USES

- Antacids decrease hyperacidity associated with PUD, GERD, gastritis, and hiatal hernia.
- Coating agents provide a protective barrier for the mucosal lining where hydrochloric acid may come into contact with inflamed eroded areas. They are used to treat existing ulcer craters on the gastric mucosa.
- H2 antagonists are used to treat acute gastric and duodenal ulcers and gastroesophageal disease, as well as for maintenance to prevent ulcer recurrence.
- Proton pump inhibitors are used to treat hyperacidity conditions (e.g., GERD, Zollinger-Ellison syndrome) and peptic and gastric ulcer disease.
- Prokinetic agents are used to treat GERD.
- Antispasmodic agents decrease gastric secretions by inhibiting vagal stimulation. They are used in treating GI disorders requiring decreased gastric motility or decreased gastric secretions.

❖ Nursing Implications for Agents Used for Stomach Disorders

▪ Assessment

Nutritional Assessment. Obtain patient data about current height, weight, and any recent weight gain or loss. Identify the normal pattern of eating, including snacking habits. Use a food guide such as MyPlate (see Figure 47-1) as a guide when asking questions to identify the usual foods eaten by the individual. Ask about any nutritional or cultural restrictions associated with dietary practices. Are there any food allergies (obtain details), or foods that particularly cause gastric distress when eaten? Does the individual take any nutritional supplements? How often and how much fast food is eaten?

Esophagus, Stomach. Ask patients to describe symptoms. Question in detail what is meant by the terms *indigestion, heartburn, upset stomach, nausea,* and *belching.*

Pain, Discomfort

- Ask the patient to describe the onset, duration, location, and characteristics of pain or discomfort. Determine whether there is a relationship between the ingestion of certain types of food or drinks and the onset of pain. Ask specifically about coffee, tea, cola, chocolate, and alcohol intake.
- What has the patient done to relieve the pain or discomfort? Have there been any changes in taste (e.g., bitterness, sourness)? Record pain using a rating scale before and after medications are administered.

Activity, Exercise. Ask specifically what type of work or activities the individual performs that may increase intra-abdominal pressure (e.g., lifting heavy objects, bending over frequently).

History of Diseases or Disorders

- What other diagnoses have been made for diseases or disorders (e.g., ulcer, gallbladder, liver, jaundice, irritable bowel syndrome)?
- Have there been any changes in bowel elimination or stool color, consistency, or frequency?

Medication History

- What self-medications have been tried?
- What prescribed medications are being taken?
- What is the schedule of medication administration (e.g., how frequently and when are antacids taken)?

Anxiety or Stress Level. Ask the patient to describe his or her lifestyle. What does the patient think are stressors, and how often do they occur?

Smoking. What is the frequency of smoking?

▪ Implementation

- Routine orders: Most health care providers order antacids 1 hour before meals, 2 to 3 hours after meals, and at bedtime. As-needed (PRN) medication dosages must be discussed.
- Each type of medication used to treat GERD or PUD may require somewhat different scheduling to avoid drug interactions. When developing the time frames for administration of medications on the medication administration record (MAR), schedule the other prescribed drugs 1 hour before or 2 hours after antacids.
- Changes in diet require careful planning with the patient as well as the person responsible for purchasing and cooking the meals. Schedule teaching sessions appropriately. Not only may some foods need to be altered, but also the number of meals per day may need to be increased with a smaller serving at each meal.

Patient Education and Health Promotion

Nutrition

- Implement prescribed dietary changes: eat small, more frequent meals to support optimal energy requirements and healing; avoid overdistention of the stomach; avoid any seasonings that are intolerable or that aggravate the condition; and avoid coffee, teas, colas, alcoholic beverages (including beer), carbonated beverages, peppermint, spearmint, and citric juices, which may produce discomfort in those with GERD.
- Avoid late-night snacks or meals that could result in increased gastric secretions.
- Observe for foods that aggravate the condition, and eliminate these from the diet. Drink only small amounts of fluid with the meal and drink mostly between meals. Increase protein foods and decrease fats to about 45 g/day or less; use nonfat milk.

Pain, Discomfort. Keep a written record of the onset, duration, location, and precipitating factors for any pain. Sit upright at the table when eating and do not lie down for at least 2 hours after eating. When a hiatal hernia is present, elevate the head of the bed on 6- to 8-inch blocks to prevent reflux during sleep. Have the patient keep a log of the pain including time of day, any factors that might have precipitated the pain, and degree of pain relief from medications used.

Medications

- Take prescribed medications at recommended times to promote optimal healing. See individual drug monographs for suggested scheduling.
- Avoid NSAIDs and aspirin-containing medicines that irritate the gastric mucosa. Consult the prescriber or pharmacist regarding scheduling of or discontinuation of these medications.

Lifestyle Changes

- Discuss stress and its effects on the person, and implement needed lifestyle changes.
- Encourage a significant reduction or cessation of smoking.
- Implement plans to gain sufficient rest.

Fostering Health Maintenance

- Discuss medication information and how it will benefit the course of treatment to produce an optimal response. Medications used in the treatment of hyperacidity are important measures to alleviate the irritating effects on the mucosal tissue; stress the importance of not discontinuing treatment, and the need for continued medical follow-up.
- Seek cooperation and understanding of the following points so that medication adherence is increased: name of medication, dosage, route and times of administration, and common and serious adverse effects. Stress the need to complete a full course of treatment for *H. pylori* so that the organisms are indeed killed and not only suppressed; *H. pylori* can regrow if medications are discontinued too early.

Written Record. Enlist the patient's aid in developing and maintaining a written record of monitoring parameters (e.g., a list of foods causing problems, degree of pain relief) (see Patient Self-Assessment form for Agents Affecting the Digestive System on the Evolve Web site at http://evolve.elsevier.com/Clayton). Complete the Premedication Data column for use as a baseline to track response to drug therapy. Ensure that the patient understands how to use the form and instruct the patient to bring the completed form to follow-up visits. During follow-up visits, focus on issues that will foster adherence with the therapeutic interventions prescribed.

DRUG CLASS: ANTACIDS

ACTIONS

Antacids lower the acidity of gastric secretions by buffering the hydrochloric acid (normal pH is 1 to 2) to a lower hydrogen ion concentration. Buffering hydrochloric acid to a pH of 3 to 4 is highly desired because the proteolytic action of pepsin is reduced and the gastric juice loses its corrosive effect.

USES

Antacid products account for one of the largest sales volumes (more than $1 billion annually) of over-the-counter medication. Antacids are commonly used for heartburn, excessive eating and drinking, and PUD. However, nurses and patients must be aware that not all antacids are alike. They should be used judiciously, particularly by certain types of patients (e.g., those with heart failure, hypertension, renal failure). Long-term self-treatment with antacids may also mask symptoms of serious underlying diseases, such as a bleeding ulcer.

The most effective antacids available are combinations of aluminum hydroxide, magnesium oxide or hydroxide, magnesium trisilicate, and calcium carbonate. All act by neutralizing gastric acid. Combinations of these ingredients must be used because any compound used alone in therapeutic quantities may produce severe systemic adverse effects. Other ingredients found in antacid combination products include simethicone, alginic acid, and bismuth. Simethicone is a defoaming agent that breaks up gas bubbles in the stomach, reducing stomach distention and heartburn. It is effective in patients who have overeaten or who have heartburn, but it is not effective in treating PUD. Alginic acid produces a highly viscous solution of sodium alginate that floats on top of the gastric contents. It may be effective only for the patient being treated for GERD or hiatal hernia and should not be used in the patient with acute gastritis or PUD. Bismuth compounds have

little acid-neutralizing capacity and are therefore poor antacids.

The following principles should be considered when antacid therapy is planned:

- For indigestion, antacids should not be administered for more than 2 weeks. If after this time the patient is still experiencing discomfort, a health care provider should be contacted.
- Patients with edema, heart failure, hypertension, renal failure, pregnancy, or salt-restricted diets should use low-sodium antacids, such as Riopan, Maalox, or Mylanta II. Therapy should continue only on the recommendation of a health care provider.
- Antacid tablets should be used only for the patient with occasional indigestion or heartburn. Tablets *do not* contain enough antacid to be effective in treating PUD.
- A common complaint of patients consuming large quantities of calcium carbonate or aluminum hydroxide is constipation. Excess magnesium results in diarrhea. If a patient experiences these symptoms and still has stomach discomfort, a health care provider should be consulted.
- Effective management of acute ulcer disease requires large volumes of antacids. The selection of an antacid and the quantity to be taken depend on its neutralizing capacity. Any patient with coffee-ground emesis, bloody stools, or recurrent abdominal pain should seek medical attention immediately and not attempt to self-treat the disorder.
- Calcium carbonate and sodium bicarbonate may cause rebound hyperacidity.
- Patients with renal failure should not use large quantities of antacids containing magnesium. The magnesium ions cannot be excreted and may produce hypermagnesemia and toxicity.
- Most antacids have similar ingredients. Selection of an antacid for occasional use should be determined by quantity of each ingredient, cost, taste, and frequency of adverse effects. Patients may need to try more than one product and weigh the advantages and disadvantages of each.

THERAPEUTIC OUTCOMES

The primary therapeutic outcomes expected from antacid therapy are relief of discomfort, reduced frequency of heartburn, and healing of irritated tissues.

Nursing Implications for Antacids

Premedication Assessment

1. Check renal function test results to ensure that renal function is normal. When renal failure is present, patients should not take large quantities of antacids containing magnesium. Monitor the patient's renal function tests, including BUN (blood urea nitrogen), creatinine, and serum electrolyte levels, including magnesium and potassium. Magnesium and potassium ions cannot be excreted and may produce hypermagnesemia, hyperkalemia, and toxicity.
2. Check the pattern of bowel elimination for diarrhea or constipation.
3. Record the pattern of gastric pain being experienced; report coffee-ground emesis, bloody stools, or recurrent abdominal pain to the health care provider for prompt attention.
4. If the patient is pregnant or has edema, heart failure, hypertension, or salt restrictions, ensure that a low-sodium antacid has been prescribed.
5. Schedule other prescribed medications to be taken 1 hour before or 2 hours after antacids are to be administered.

Life Span Considerations

Antacids

Those older than 65 years are the most common purchasers of antacid products. Gastrointestinal disorders such as PUD, NSAID-induced ulcers, and GERD occur more often in this age-group. Magnesium-containing antacids are often used as laxatives. Although the primary symptom of ulcer disease in a younger person is usually burning epigastric pain, the symptoms in an older person, if present at all, usually include complaints of vague abdominal discomfort, anorexia, and weight loss.

Availability

See Table 33-1.

- Liquid forms of antacid preparations should be used for treatment of PUD because tablets do not contain enough of the active ingredients to be effective.
- Antacid tablets may be used for occasional episodes of heartburn. They should be well chewed before swallowing for a more rapid onset of action.

Monitoring

Common Adverse Effects

Chalky Taste. A chalky taste is a common problem with antacids. Suggest a change in brands or flavors. Suggest using a liquid instead of tablets.

Serious Adverse Effects

Gastrointestinal

Diarrhea, Constipation. Diarrhea or constipation is a common problem when antacids are used in therapeutic dosages to treat ulcers. Alternating between calcium- or aluminum-containing compounds and magnesium-containing compounds should help alleviate the problem.

Table 33-1 Ingredients of Commonly Used Antacids

PRODUCT	FORM	CALCIUM CARBONATE	ALUMINUM HYDROXIDE	MAGNESIUM OXIDE OR HYDROXIDE	SODIUM BICARBONATE	SIMETHICONE	OTHER INGREDIENTS
Aludrox	Tablet, suspension	—	X	X	—	X	—
Baking soda	Powder	—	—	—	X	—	—
Di-Gel	Tablet, liquid	—	X	X	—	X	—
Gelusil	Tablet	—	X	X	—	X	—
Maalox Maximum Strength	Suspension	—	X	X	—	X	—
Mylanta	Tablet, suspension	—	X	X	—	X	—
Mylanta Extra Strength	Suspension	—	X	X	—	X	—
Mylanta Supreme	Liquid	X	—	X	—	—	—
Phillip's Milk of Magnesia	Tablet, suspension	—	—	X	—	—	—
Riopan	Suspension	—	—	—	—	—	Magaldrate
Riopan Plus	Tablet, suspension	—	—	—	—	X	Magaldrate
Titralac Plus	Tablet, suspension	X	—	—	—	X	—
Tums	Tablet	X	—	—	—	—	—

Drug Interactions

Tetracycline Antibiotics, Fluoroquinolone Antibiotics, Phenytoin, Phenothiazines, Captopril, Ketoconazole, Corticosteroids, Digoxin, Iron Supplements. The absorption of these medicines is inhibited by antacids. These medications should be administered 1 hour before or 2 to 3 hours after antacids.

Levodopa, Valproic Acid. The absorption of levodopa is increased by magnesium-aluminum antacids. When antacid therapy is added, toxicity may result in the patient with Parkinson's disease whose condition is well controlled on a certain dosage of levodopa. If the patient's parkinsonism symptoms are well controlled on levodopa *and* antacid therapy, withdrawal of antacids may result in a recurrence of parkinsonian symptoms.

Quinidine, Amphetamines. Frequent use of sodium bicarbonate–containing antacid therapy may result in increased urinary pH. Renal excretion of quinidine and amphetamines may be inhibited, and toxicity may occur.

DRUG CLASS: HISTAMINE-2 RECEPTOR ANTAGONISTS

ACTIONS

One of the primary mechanisms of hydrochloric acid secretion has to do with histamine stimulation of histamine-2 (H2) receptors on the stomach's parietal cells. The H2 antagonists act by blocking the H2 receptors, resulting in a decrease in the volume of acid secreted. The pH of the stomach contents rises as a consequence of a reduction in acid.

USES

The H2 antagonists (cimetidine, ranitidine, nizatidine, famotidine) are used to treat GERD, duodenal ulcers, and pathologic hypersecretory conditions, such as Zollinger-Ellison syndrome, and for preventing and treating stress ulcers in critically ill patients. Unapproved uses include prevention of aspiration pneumonitis, acute upper GI bleeding, and hyperparathyroidism.

Cimetidine was the first approved H2 antagonist but differs from the other agents by its extensive liver metabolism and antiandrogenic effect that may result in gynecomastia. It also has many drug-drug interactions that are not seen with the others. Consequently, cimetidine is used less frequently than the other H2 antagonists.

Famotidine is similar in action and use to cimetidine but has the advantages of one dose daily, fewer drug interactions, and no antiandrogenic effect. A parenteral dosage form is available.

Ranitidine is similar in action and use to cimetidine but has the advantages of twice-daily dosing, fewer drug interactions, and no antiandrogenic effect. A parenteral dosage form is also available.

Nizatidine is similar to ranitidine and famotidine but, in contrast to the other agents, is not available in a parenteral dosage form.

THERAPEUTIC OUTCOMES

The primary therapeutic outcomes expected from H2 antagonist therapy are relief of discomfort, reduced frequency of heartburn, and healing of irritated tissues.

Table 33-2 Histamine-2 (H2) Receptor Antagonists

GENERIC NAME	BRAND NAME	AVAILABILITY	DOSAGE RANGE
cimetidine	Tagamet, Tagamet HB Novo-Cimetidine 🍁	Tablets: 200, 300, 400, 800 mg Suspension: 300 mg/5 mL Injection: 300 mg/2 mL	*Duodenal and gastric ulcers* PO: 800-1600 mg at bedtime, 400 mg twice daily, or 300 mg four times daily IM, IV: 300 mg q6-8h *GERD* PO: 800 mg twice daily or 400 mg four times daily
famotidine ⇄ *Do not confuse famotidine with fluoxetine.*	Pepcid, Pepcid AC ⇄ Do not confuse Pepcid with Prevacid. Nu-Famotidine 🍁	Tablets: 10, 20, 40 mg Tablets, chewable: 10 mg Tablets, orally disintegrating: 20, 40 mg Suspension: 40 mg/5 mL Injection: 10 mg/mL	*Duodenal and gastric ulcers* PO: 40 mg once daily at bedtime or 20 mg twice daily *GERD* PO: 20 mg twice daily
nizatidine ⇄ *Do not confuse nizatidine with tizanidine.*	Axid Apo-Nizatidine 🍁	Capsules: 150, 300 mg Tablets: 75 mg Oral solution: 15 mg/mL	*Duodenal and gastric ulcers* PO: 300 mg at bedtime or 150 mg twice daily *GERD* PO: 150 mg twice daily
ranitidine ⇄ *Do not confuse ranitidine with amantadine or rimantidine.*	Zantac; Zantac 75, 150 ⇄ *Do not confuse Zantac with Xanax or Zyrtec.* PHL-Ranitidine 🍁	Tablets: 75, 150, 300 mg Efferdose tablets: 25 mg Syrup: 15 mg/mL Solution: 15 mg/mL Injection: 1, 25 mg/mL	*Duodenal and gastric ulcers* PO: 300 mg at bedtime or 150 mg twice daily *GERD* PO: 150 mg twice daily

🍁 Available in Canada.

Nursing Implications for H2 Antagonists

Premedication Assessment

Perform a baseline assessment of the patient's mental status for comparison with subsequent mental status evaluations to detect central nervous system (CNS) alterations that may occur, particularly with cimetidine therapy.

Availability, Dosage, and Administration

See Table 33-2.

- All may be administered with or without food.
- Because antacid therapy is often continued during early therapy of PUD, administer 1 hour before or 2 hours after the H2-antagonist dose.

Monitoring

Common Adverse Effects. Approximately 1% to 3% of patients develop these adverse effects. They are usually mild and resolve with continued therapy. Encourage patients not to discontinue therapy without first consulting the health care provider.

Neurologic

Dizziness, Headache, Somnolence. Provide for patient safety during episodes of dizziness. If the patient develops somnolence and lethargy, encourage the use of caution when working around machinery, driving a car, or performing other duties that require mental alertness.

Gastrointestinal

Diarrhea, Constipation. Maintain the patient's state of hydration, and obtain an order for stool softeners or bulk-forming laxatives if necessary. Encourage the inclusion of sufficient roughage (fresh fruits, vegetables, whole-grain products) in the diet.

Serious Adverse Effects

Neurologic

Confusion, Disorientation, Hallucinations. If high dosages (particularly of cimetidine) are used in patients with liver or renal disease or in patients older than 50 years, mental confusion, slurred speech, disorientation, and hallucinations may occur. These adverse effects dissipate over 3 or 4 days after therapy has been discontinued. Perform a baseline assessment of the patient's degree of alertness and orientation to name, place, and time before starting therapy. Make regularly scheduled subsequent mental status evaluations, and compare findings. Report alterations.

Endocrine

Gynecomastia. Mild bilateral gynecomastia and breast soreness may occur with long-term use (longer than 1 month) of cimetidine but will resolve after discontinuing therapy. Report for further observation and possible laboratory tests.

Gastrointestinal

Hepatotoxicity. Although rare, hepatotoxicity in patients taking H2 antagonists has been reported. The symptoms of hepatotoxicity are anorexia, nausea, vomiting, jaundice, hepatomegaly, splenomegaly, and abnormal liver function test results (elevated bilirubin, aspartate aminotransferase [AST], alanine aminotransferase [ALT], gamma-glutamyl transferase [GGT], alkaline phosphatase levels, increased prothrombin time).

Drug Interactions

Benzodiazepines. Cimetidine inhibits the metabolism or excretion of the following benzodiazepines: alprazolam, chlordiazepoxide, diazepam, clorazepate, flurazepam, halazepam, prazepam, and triazolam.

Patients taking cimetidine and a benzodiazepine concurrently should be observed for increased sedation; a reduction in dosage of the benzodiazepine may be required. The metabolism of oxazepam, temazepam, and lorazepam does not appear to be affected.

Theophylline Derivatives. Cimetidine inhibits the metabolism or excretion of the following xanthine derivatives: aminophylline, dyphylline, and theophylline. Patients at greater risk include those receiving larger doses of theophylline and those with liver disease. Observe for restlessness, vomiting, dizziness, and cardiac dysrhythmias. The dosage of theophylline may need to be reduced.

Beta-Adrenergic Blocking Agents. Beta-adrenergic blocking agents (e.g., propranolol, labetalol, metoprolol) may accumulate as a result of inhibited metabolism. Monitor for signs of toxicity, such as hypotension and bradycardia.

Phenytoin. Cimetidine inhibits the metabolism of phenytoin. Monitor patients with concurrent therapy for signs of phenytoin toxicity: nystagmus, sedation, and lethargy. Serum levels may be ordered, and a reduced dosage of phenytoin may be required.

Lidocaine, Quinidine, Procainamide. Cimetidine may inhibit the metabolism of these agents. Monitor for signs of toxicity (e.g., bradycardia, additional dysrhythmias, hyperactivity, sedation), and reduce the dosage if necessary.

Antacids. Administer 1 hour before or 2 hours after administration of cimetidine.

Warfarin. Cimetidine may enhance the anticoagulant effects of warfarin. Observe for the development of petechiae, ecchymoses, nosebleeds, bleeding gums, dark tarry stools, and bright red or coffee-ground hematemesis. Monitor the prothrombin time (international normalized ratio [INR]), and reduce the dosage of warfarin if necessary.

Calcium Antagonists. Cimetidine may inhibit the metabolism of diltiazem, nifedipine, and verapamil. Patients should be monitored for increased effects from the calcium antagonists (e.g., bradycardia, hypotension, dysrhythmias, fatigue).

Tricyclic Antidepressants. Cimetidine may inhibit the excretion of imipramine, desipramine, and nortriptyline, usually within 3 to 5 days after the start of cimetidine therapy. If anticholinergic effects or toxicity becomes apparent, a decreased dosage of the antidepressant may be required. If cimetidine is discontinued, the patient should be monitored for a decreased response to the antidepressant.

Famotidine, Nizatidine, Ranitidine. In general, there appear to be only minor interactions with these H2 antagonists. There are conflicting data, however. Studies indicate that patients receiving higher doses of ranitidine may be more susceptible to drug interactions with ranitidine and other drugs. When used concurrently, monitor for toxic effects of warfarin, theophylline, procainamide, and glipizide.

DRUG CLASS: GASTROINTESTINAL PROSTAGLANDIN

misoprostol (mĭs-ō-PRŎS-tŏl)
Cytotec (SĪ-tō-tĕk)

ACTIONS

Misoprostol is the first of a new synthetic prostaglandin E series to be used to treat GI disorders. Prostaglandins are normally present in the GI tract to inhibit gastric acid and pepsin secretion to protect the stomach and duodenal lining against ulceration. The prostaglandin E analogues may also induce uterine contractions.

USES

Misoprostol is used to prevent and treat gastric ulcers caused by NSAIDs, including aspirin. Whereas prostaglandin inhibition is effective in reducing pain and inflammation, especially in arthritis, prostaglandin inhibition in the stomach makes the patient more predisposed to peptic ulcers.

THERAPEUTIC OUTCOMES

The primary therapeutic outcomes expected from misoprostol therapy are relief of discomfort and healing of irritated tissues.

Nursing Implications for Misoprostol Therapy

Premedication Assessment

1. Determine if the patient is pregnant. This drug is a uterine stimulant and may induce miscarriage.
2. Check the pattern of bowel elimination; misoprostol may induce diarrhea.

Availability

100- and 200-mcg tablets.

Medication Safety Alert

Misoprostol is contraindicated during pregnancy and in women at risk of becoming pregnant. As a uterine stimulant, it may induce miscarriage.

Dosage and Administration

Adult: *PO:* 100- to 200-mcg tablets four times daily with food during NSAID therapy.

Monitoring

Common Adverse Effects

Gastrointestinal

Diarrhea. Diarrhea associated with misoprostol therapy is dose-related and usually develops after approximately 2 weeks of therapy. It often resolves after about 8 days, but a few patients require discontinuation of misoprostol therapy. Diarrhea can be minimized by taking misoprostol with meals and at bedtime and avoiding magnesium-containing antacids (e.g., Maalox, Mylanta). Encourage the patient not to discontinue therapy without first consulting the health care provider.

Encourage the patient to include sufficient roughage (fresh fruits, vegetables, whole-grain products) in the diet.

Contraindication

Pregnancy. It is crucial that therapy be discontinued if the patient is pregnant. Misoprostol is a pregnancy category X drug that may cause spontaneous abortion. The patient must receive attention from the health care provider who prescribed the misoprostol, as well as an obstetrician. The question of alternative therapies to NSAIDs must also be considered.

Drug Interactions

No significant drug interactions have been reported.

DRUG CLASS: PROTON PUMP INHIBITORS

ACTIONS

Proton pump inhibitors (PPIs) inhibit gastric secretion by inhibiting the gastric acid pump of the stomach's parietal cells. These inhibitors have no anticholinergic or H2 receptor antagonist actions.

USES

Proton pump inhibitors are used to treat severe esophagitis, GERD, gastric and duodenal ulcers, and hypersecretory disorders, such as Zollinger-Ellison syndrome. They may also be used in combination with antibiotics (e.g., ampicillin, amoxicillin, clarithromycin) to eradicate *H. pylori*, a common cause of PUD.

THERAPEUTIC OUTCOMES

The primary therapeutic outcomes expected from proton pump inhibitors are relief of discomfort, reduced frequency of heartburn, and healing of irritated tissues.

Nursing Implications for Proton Pump Inhibitor Therapy

Premedication Assessment

Check pattern of bowel elimination; the pump inhibitors may induce diarrhea.

Availability, Dosage, and Administration

See Table 33-3. Omeprazole, lansoprazole, and esomeprazole need to be taken before a meal. Capsules and tablets should be swallowed whole; instruct the patient not to open, chew, or crush.

Monitoring

Common Adverse Effects. The following symptoms are relatively mild and rarely result in the discontinuation of therapy. Encourage the patient not to discontinue therapy without first consulting the health care provider.

Gastrointestinal

Diarrhea. Maintain the patient's state of hydration. Encourage the inclusion of sufficient roughage (fresh fruits, vegetables, whole-grain products) in the diet.

Neurologic. Headache, fatigue.

Musculoskeletal. Muscle pain.

Serious Adverse Effects

Skeletal

Risk of Fractures. Patients who are over the age of 50 years who received high doses or used them for more than 1 year are at greater risk for fractures of the hip, wrist, and spine. Patients who continue to receive PPIs and who are at risk for osteoporosis should receive vitamin D and calcium supplementation and have their bone status monitored. Patients should not stop taking their proton pump inhibitor unless told to do so by their health care provider. Short term, low-dose use of over-the-counter products is not likely to cause an increased risk of fractures.

Electrolytes

Hypomagnesemia. Hypomagnesemia has been reported in as little as 3 months of proton pump inhibitor therapy, but it more commonly occurs in patients receiving PPIs for more than 1 year. Hypomagnesemia can cause serious adverse events, including tetany, tremors, seizures, QT prolongation, and cardiac arrhythmias. Health care providers should consider obtaining serum magnesium levels prior to beginning long-term PPI therapy, especially in patients receiving digoxin or patients receiving diuretics or other

Table 33-3 Proton Pump Inhibitors

GENERIC NAME	BRAND NAME	AVAILABILITY	DOSAGE RANGE
dexlansoprazole	Dexilant	Capsules: 30, 60 mg	PO: Initial: 60 mg once daily for 4-8 wk Maintenance: 30 mg once daily
esomeprazole ⇄ *Do not confuse esomeprazole with omeprazole.*	Nexium Apo-Esomeprazole 🍁	Capsules: 20, 40 mg Powder for suspension: 10-, 20-, 40-mg unit dose IV: 20, 40 mg	IV, PO: Initial: 20-40 mg once daily for 4-8 wk Maintenance: 20 mg daily
lansoprazole	Prevacid ⇄ *Do not confuse Prevacid with Prinivil, Pepcid, Pravachol, Premarin, or Prilosec.* Teva-Lansoprazole 🍁	Capsules: 15, 30 mg Tablets: 15, 30 mg	PO: Initial: 15-30 mg once daily 30 min before a meal for 4 wk Maintenance: 15 mg once daily Maximum: 30 mg once daily before a meal
omeprazole ⇄ *Do not confuse omeprazole with esomeprazole.*	Prilosec ⇄ *Do not confuse Prilosec with Prinivil, Prednisone, Prevacid, Prinivil, or Prozac.* Losec 🍁	Capsules: 10, 20, 40 mg Tablets: 20 mg Packet: 2.5, 10 mg	PO: Initial: 20 mg once daily for 4 wk Maintenance: 20 mg daily Maximum: 120 mg three times daily for Zollinger-Ellison syndrome
pantoprazole	Protonix ⇄ *Do not confuse Protonix with Lotronex.* Pantoloc 🍁	Tablets: 20, 40 mg Powder for oral suspension: 40 mg IV: 40 mg/vial	PO: Initial: 40 mg once daily for 8 wk Maintenance: 40 mg daily IV: Initial: 40 mg once daily for up to 7-10 days; switch to oral dosages
rabeprazole	Aciphex ⇄ *Do not confuse Aciphex with Accupril, Adipex-P, Aricept, or Vioxx.* Pariet 🍁	Tablets: 20 mg	PO: Initial: 20 mg daily after morning meal for up to 4 wk Maintenance: 20 mg once daily Maximum: 60 mg twice daily

🍁 Available in Canada.

medicines known to cause hypomagnesemia. Magnesium supplementation may resolve the hypomagnesemia, but discontinuation of the PPI may be necessary. Magnesium levels return to normal within about a week of discontinuing PPI therapy. Patients should not stop PPI therapy without first discussing it with their health care provider. Use of over-the-counter PPIs taken according to directions and for a limited duration have not been associated with hypomagnesemia.

Integumentary

Rash. Persistent vesicular rash from omeprazole may be cause for discontinuing therapy. Report to the health care provider for further observation and possible laboratory tests.

Drug Interactions

Diazepam, Triazolam, Flurazepam. Omeprazole and esomeprazole significantly increases the half-life of diazepam, triazolam, and flurazepam by inhibiting its metabolism. Observe patients for increased sedative effects from these medicines. Caution against hazardous tasks, such as driving and operating machinery. The dosages of diazepam, triazolam, and flurazepam may have to be reduced.

Phenytoin. Omeprazole slows the metabolism of phenytoin. Observe for nystagmus, sedation, and lethargy. The dosage of phenytoin may have to be reduced.

Clopidogrel. There has been a controversy in the literature as to whether PPIs prevent conversion of clopidogrel to its active, therapeutic metabolite. A

consensus statement by the American Cardiology Foundation, the American Heart Association, and the American College of Gastroenterology reports that clopidogrel alone and aspirin alone and their combination are associated with an increased risk of gastrointestinal (GI) bleeding; the risk of GI bleeding increases as the number of risk factors increase (such as prior GI bleeding, advanced age, concurrent use of anticoagulants); PPIs are appropriate in patients with multiple risk factors for GI bleeding who are also receiving antiplatelet therapy such as clopidogrel; a clinically significant interaction cannot be excluded in subgroups who are poor metabolizers of clopidogrel; until solid evidence exists to support staggering PPIs with clopidogrel, the dosing of PPIs should not be altered.

Warfarin. Omeprazole may reduce the rate of metabolism of warfarin. Monitor the patient closely for signs of bleeding tendencies, and monitor the prothrombin time (INR) closely. Reduction of warfarin dosage may be required.

Sucralfate. Sucralfate inhibits the absorption of proton pump inhibitors. Administer the pump inhibitors at least 30 minutes before sucralfate.

Altered Absorption. The reduction in gastric acid secretion may alter absorption of food and drugs as follows:

- *Ketoconazole, ampicillin, iron:* These medicines require an acid medium for absorption. They should be administered at least 30 to 45 minutes before lansoprazole therapy.
- *Insulin:* The absorption of food may be altered, and an adjustment in timing or dosage of insulin in patients with diabetes may be required.

DRUG CLASS: COATING AGENTS

sucralfate (sū-KRĂL-fāt)
Carafate (KĂR-ă-fāt)

ACTIONS

When swallowed, sucralfate forms a complex that adheres to the crater of an ulcer, thereby protecting it from aggravators such as acid, pepsin, and bile salts. However, sucralfate does not inhibit gastric secretions (as do the H2 antagonists) or alter gastric pH (as do antacids).

USES

Sucralfate is used to treat duodenal ulcers, particularly in those patients who do not tolerate other forms of therapy.

THERAPEUTIC OUTCOMES

The primary therapeutic outcomes expected from sucralfate therapy are relief of discomfort and healing of irritated tissues.

Nursing Implications for Sucralfate Therapy

Premedication Assessment

Check pattern of bowel elimination; sucralfate may induce constipation.

Availability

PO: 1-g tablets and 1 g/10 mL suspension.

Dosage and Administration

Adult: *PO:* 1 tablet 1 hour before each meal and at bedtime, all on an empty stomach. Because antacid therapy is often continued during early therapy of ulcer disease, administer antacids at least 30 minutes before or after sucralfate.

Monitoring

Common Adverse Effects. The following adverse effects are usually mild and tend to resolve with continued therapy. Encourage the patient not to discontinue therapy without first consulting the health care provider.

Gastrointestinal

Constipation, Dry Mouth. Measures to alleviate dry mouth include sucking on ice chips or hard candy. Avoid mouthwashes that contain alcohol because they cause further drying and irritation. Maintain the patient's state of hydration, and obtain an order for stool softeners or bulk-forming laxatives if necessary. Encourage the inclusion of sufficient roughage (e.g., fresh fruits, vegetables, whole-grain products) in the diet.

Neurologic

Dizziness. Provide patient safety during episodes of dizziness.

Drug Interactions

Tetracyclines. Sucralfate may interfere with the absorption of tetracycline. Administer tetracyclines 1 hour before or 2 hours after sucralfate.

Omeprazole, Lansoprazole. Sucralfate inhibits the absorption of omeprazole and lansoprazole. Administer omeprazole or lansoprazole at least 30 minutes before sucralfate.

DRUG CLASS: PROKINETIC AGENTS

metoclopramide (mĕt-ō-KLŌ-pră-mīd)
Reglan (RĒG-lăn)

ACTIONS

Metoclopramide is a gastric stimulant whose mechanisms of action are not fully known. It increases lower esophageal sphincter pressure, thereby reducing reflux; increases stomach contractions; relaxes the

pyloric valve; and increases peristalsis in the GI tract, resulting in an increased rate of gastric emptying and intestinal transit. Metoclopramide is an antiemetic that blocks dopamine in the chemoreceptor trigger zone. It inhibits serotonin (5-HT3) when administered in higher dosages.

USES

Metoclopramide is used to relieve the symptoms of gastric reflux esophagitis and diabetic gastroparesis, as an aid in small bowel intubation, and to stimulate gastric emptying and intestinal transit of barium after radiologic examination of the upper GI tract. It is also given as an antiemetic to patients undergoing cancer chemotherapy.

THERAPEUTIC OUTCOMES

The primary therapeutic outcomes expected from metoclopramide therapy are relief of discomfort, reduced frequency of heartburn, and healing of irritated tissues.

Nursing Implications for Metoclopramide Therapy

Premedication Assessment

1. Determine if other drugs being taken may induce extrapyramidal symptoms; do not administer drug concurrently.
2. Check for a history of epilepsy. If present, check with the health care provider before starting drug therapy.
3. Do not administer to an individual with symptoms of GI perforation, mechanical obstruction, or hemorrhage.
4. For diabetic patients, food absorption may be altered and more frequent monitoring for hypoglycemia may be required.

Availability

PO: 5- and 10-mg tablets and 5 mg/5 mL syrup. *Injection:* 5 mg/mL in 2-, 10-, and 30-mL ampules.

Caution

Approximately 1 in 500 patients may develop extrapyramidal symptoms manifested by restlessness, involuntary movements, facial grimacing, and possibly oculogyric crisis, torticollis, or rhythmic tongue protrusion. Children and young adults are most susceptible, as are patients receiving higher doses of metoclopramide as an antiemetic. Metoclopramide should not be used in patients with epilepsy or those receiving drugs likely to cause extrapyramidal reactions (e.g., phenothiazines) because the frequency and severity of seizures or extrapyramidal reactions may be increased. Metoclopramide must not be used in patients when increased gastric motility may be dangerous, such as with GI perforation, mechanical obstruction, or hemorrhage.

Dosage and Administration

Adult

- *PO:* Diabetic gastroparesis: 10 mg 30 minutes before each meal and at bedtime. Duration of therapy depends on response and continued well-being after discontinuation of therapy.
- *IV:* Antiemesis: Initial two doses, 2 mg/kg. If vomiting is suppressed, follow with 1 mg/kg. Dilute the dose in 50 mL of parenteral solution (D5W, normal saline [0.9%], D5/0.45 saline, Ringer's solution, or lactated Ringer's solution). Infuse over at least 15 minutes, 30 minutes before beginning chemotherapy. Repeat every 2 hours for two doses, followed by one dose every 3 hours for three doses.

Medication Safety Alert

Rapid IV infusion of metoclopramide (Reglan) may cause sudden, intense anxiety and restlessness, followed by drowsiness. If extrapyramidal symptoms should develop, treat with diphenhydramine.

Monitoring

Common Adverse Effects. The following adverse effects are usually mild and tend to resolve with continued therapy. Encourage the patient not to discontinue therapy without first consulting the health care provider.

Neurologic

Drowsiness, Fatigue, Lethargy, Dizziness. People who work around machinery, drive a car, or perform other duties that require mental alertness should be particularly cautious. Provide patient safety during episodes of dizziness.

Gastrointestinal. Nausea.

Serious Adverse Effects

Extrapyramidal Symptoms. Provide patient safety, and then report extrapyramidal symptoms to the health care provider immediately.

Drug Interactions

Drugs That Increase Sedative Effects. Antihistamine, alcohol, analgesics, phenothiazines, and sedative-hypnotics increase the sedative effects of metoclopramide. Monitor the patient for excessive sedation, and reduce dosage if necessary.

Drugs That Decrease Therapeutic Effects. Anticholinergic agents (e.g., atropine, benztropine, antihistamines, dicyclomine) and narcotic analgesics (e.g., meperidine, morphine, oxycodone, and others) decrease the therapeutic effects of metoclopramide. Instruct the patient to avoid taking these agents while using metoclopramide.

Altered Absorption. The GI stimulatory effects of metoclopramide may alter absorption of food and drugs as follows:

- *Digoxin:* Monitor for decreased activity (e.g., return of edema, weight gain, heart failure).
- *Levodopa:* Monitor for increased activity (e.g., restlessness, nightmares, hallucinations, and additional involuntary movements such as bobbing of head and neck, facial grimacing, and active tongue movements).
- *Alcohol:* Monitor for signs of sedation and intoxication with smaller amounts of alcohol.
- *Insulin:* The absorption of food may be altered, and an adjustment in timing or dosage of insulin in patients with diabetes mellitus may be required.

DRUG CLASS: ANTISPASMODIC AGENTS

ACTIONS

Drugs used as antispasmodic agents are actually anticholinergics. The GI tract is heavily innervated by the cholinergic branch of the autonomic nervous system. Cholinergic fibers stimulate the GI tract, causing secretion of saliva, hydrochloric acid, pepsin, bile, and other enzymatic fluids necessary for digestion, relaxation of sphincter muscles, and peristalsis to move the contents of the stomach and bowel through the GI tract. The antispasmodic agents act by preventing acetylcholine from attaching to the cholinergic receptors in the GI tract. The extent of reduction of cholinergic activity depends on the amount of anticholinergic medication blocking the receptors. Inhibition of cholinergic nerve conduction results in decreased GI motility and reduced secretions.

Because cholinergic fibers innervate the entire body and these agents are not selective in their actions to the GI tract, the effects of blocking this system are seen throughout the body. To provide adequate dosages to inhibit GI motility and secretions, the following effects may also occur: reduced perspiration and oral and bronchial secretions, mydriasis (dilation of the pupils) with blurring of vision, constipation, urinary hesitancy or retention, tachycardia (possibly with palpitations), and mild transient postural hypotension. Psychiatric disturbances, such as mental confusion, delusions, nightmares, euphoria, paranoia, and hallucinations, may be indications of overdosage.

USES

Antispasmodic agents are used to treat irritable bowel syndrome, biliary spasm, mild ulcerative colitis, diverticulitis, pancreatitis, infant colic, and, in conjunction with diet and antacids, PUD. Since the advent of the H2 antagonists, antispasmodic agents are used much less frequently in treating ulcers.

THERAPEUTIC OUTCOMES

The primary therapeutic outcomes expected from antispasmodic therapy are relief of discomfort, reduced frequency of heartburn, and healing of irritated tissues.

Nursing Implications for Antispasmodic Therapy

Premedication Assessment

1. Check the patient's history to screen for presence of closed-angle glaucoma; use of antispasmodic therapy in a patient with this condition could initiate an acute attack.
2. Perform a baseline mental status examination for future comparison of subsequent findings; these medicines may cause confusion, depression, nightmares, or hallucinations. These symptoms should be reported.
3. Obtain a baseline blood pressure and plan to monitor blood pressure for orthostatic hypotension, a common drug adverse effect.
4. Use these drugs with caution in older patients and in patients with any condition in which GI transit time is compromised, because anticholinergic agents slow peristalsis.

Availability, Dosage, and Administration

See Table 33-4. Administer with food or milk to minimize gastric irritation.

Medication Safety Alert

All patients should be screened for closed-angle glaucoma before initiating antispasmodic agents. Patients with open-angle glaucoma can safely use anticholinergic agents. Intraocular pressure should be monitored regularly.

Monitoring

Common Adverse Effects

Anticholinergic

Blurred Vision; Constipation; Urinary Retention; Dryness of the Mouth, Nose, and Throat. These are the anticholinergic effects produced by antispasmodic agents. Patients taking these medications should be monitored for these adverse effects. Mucosa dryness may be alleviated by sucking hard candy or ice chips or by chewing gum. If patients develop urinary hesitancy, assess for bladder distention. Report to the health care provider for further evaluation. Give stool softeners as prescribed. Encourage adequate fluid intake and foods that provide sufficient bulk. Caution the patient that blurred vision may occur, and make appropriate suggestions for patient safety.

Serious Adverse Effects

Neurologic

Confusion, Depression, Nightmares, Hallucinations. Perform a baseline assessment of the patient's degree of alertness

Table 33-4 Antispasmodic Agents

GENERIC NAME	BRAND NAME	AVAILABILITY	CLINICAL USES	INITIAL RANGE
atropine ⇄ *Do not confuse atropine with Akarpine.*	Atropine Sulfate	Injection: 0.05, 0.1, 0.4, 0.5, 0.8, 1, 2 mg/mL Tablets: 0.4 mg	Treatment of pylorospasm and spastic conditions of GI tract	PO: 0.4-0.6 mg
dicyclomine ⇄ *Do not confuse dicyclomine with demeclocycline, diphenhydramine, or doxycycline.*	Bentyl ⇄ *Do not confuse Bentyl with Benadryl, Bumex, or Proventil.* Bentylol 🍁	Tablets: 20 mg Capsules: 10, 20 mg Syrup: 10 mg/5 mL Injection: 10 mg/mL	Irritable bowel syndrome Infant colic	Adults: PO: 20-40 mg three or four times daily Infants: PO: 5 mg three or four times daily
glycopyrrolate	Robinul, Cuvposa	Tablets: 1, 2 mg Injection: 0.2 mg/mL Oral solution: 1 mg/5 mL	Peptic ulcer disease, preanesthetic	PO: 1 mg three times daily IM: 0.004 mg/kg 30-60 min before surgery
mepenzolate	Cantil	Tablets: 25 mg	Peptic ulcer disease	PO: 25-50 mg four times daily
methscopolamine	Pamine	Tablets: 2.5, 5 mg	Peptic ulcer disease	PO: 2.5 mg 30 min before meals and 2.5-5 mg at bedtime
propantheline	—	Tablets: 15 mg	Peptic ulcer disease	PO: 15 mg before meals and 30 mg at bedtime
scopolamine	Scopace, Scopolamine, Transderm Scōp, Transderm-V 🍁	Tablets: 0.4 mg Injection:, 0.4, mg/mL Transdermal patch: 1.5 mg	GI hypermotility, pylorospasm, irritable colon syndrome	PO: 0.4 to 0.8 mg Preoperative: Subcutaneously or IM: 0.32-0.65 mg Patch: 1.5 mg, 72 hr duration

🍁 Available in Canada.

and orientation to name, place, and time **before** initiating therapy. Make regularly scheduled subsequent mental status evaluations and compare findings. Report alterations. Provide patient safety during these episodes. Reducing the daily dosage may control these adverse effects.

Cardiovascular

Orthostatic Hypotension. All antispasmodic agents may cause some degree of orthostatic hypotension, although it is infrequent and generally mild. It is manifested by dizziness and weakness, particularly when therapy is initiated. Monitor the blood pressure daily in both the supine and standing positions. Anticipate the development of postural hypotension, and take measures to prevent it. Teach the patient to rise slowly from a supine or sitting position. Encourage the patient to sit or lie down if feeling faint.

Palpitations, Dysrhythmias. Report to health care provider for further evaluation.

Drug Interactions

Amantadine, Tricyclic Antidepressants, Phenothiazines. These agents may potentiate the anticholinergic adverse effects. Confusion and hallucinations characterize excessive anticholinergic activity.