Most cases of diarrhea are self-limited and require only supportive care. Conversely, patients with more serious infection and associated comorbidity may have life-threatening dehydration and shock. Associated sepsis and septic shock can be complications of severe diarrheal disease. Numerous but relatively rare, noninfectious causes of diarrhea should be considered.

**Incidence**

Worldwide, diarrhea remains a major health problem, accounting for approximately 4% of all deaths each year, which is estimated by the World Health Organization (WHO) to be 2.2 million victims. A large proportion of these deaths occur in small children in developing countries. Diarrheal disease kills 1.5 million children every year. Globally, there are about 2 billion cases of diarrheal disease every year.

Rotavirus causes 25 to 65% of childhood-associated diarrheal illnesses (3.5 million cases per year in the United States), whereas adults experience 74 million episodes of diarrhea annually. In the United States, 90% of diarrheal illnesses are caused by noroviruses (caliciviruses), of which more than 100 different strains are recognized. Patients at the extremes of age, those with significant comorbidity, those who are immunologically compromised, and those with iatrogenic illness are most vulnerable to significant morbidity and mortality. An estimated 60% of patients infected with human immunodeficiency virus (HIV) experience significant diarrhea during the course of their illness.

Natural disasters, environmental changes, and wars or political uprisings can result in mass migrations of refugees to neighboring countries. This can lead to starvation and thirst in areas with limited resources, a setup for water contamination and epidemic diarrhea. As witnessed in Haiti after the 2010 earthquake, lack of clean water and poor sanitation led to an epidemic of a deadly form of *Vibrio cholera*. Most adults experience diarrhea many times during their lifetime. Diarrheal illnesses are the primary cause of many hospitalizations and hours of lost work.

**Definition and Categorization**

The term *diarrhea* is derived from the Greek words *dia* (“through”) and *rhein* (“to flow”). The two main categories of diarrheal-associated illness are infectious and noninfectious. Infectious causes represent about 85% of cases, whereas noninfectious causes account for only 15% of the total. Infectious diarrhea may be divided into viral, bacterial, and parasitic causes (Box 31-1), with estimates of their relative contributions being 70% for viral, 24% for bacterial, and 6% for parasitic infections.

Definitions for diarrhea have been proposed to standardize nomenclature, help the clinician determine a probable cause, and direct empirical therapy if indicated:

*Acute diarrhea* is defined as lasting for 14 days or less.
*Persistent diarrhea* lasts for longer than 14 days.
*Chronic diarrhea* lasts 30 days or longer.

Acute diarrhea presentations usually will be infectious. A majority of these cases are self-limited and caused by viral and bacterial pathogens. Persistent diarrhea suggests an enteric pathogen other than viral, such as bacterial or protozoan. Chronic diarrhea usually is associated with noninfectious causes and requires further testing to determine the cause.

Normally, the small and large bowels absorb 99% of gastrointestinal tract secretions produced and liquids ingested each day. Any pathologic state that reduces water absorption by 1% can cause diarrhea. Diarrhea results from one or more of four different pathologic processes that are characteristic of the primary cause and that contribute to the decreased absorption of the gut.

*Secretory diarrhea* is caused by pathogens that produce cytoxicins that increase cellular permeability and cause the oversecretion of water and electrolytes. Most cases of diarrhea encountered in the emergency department (ED) are secretory. Noninfectious causes of secretory diarrhea include medications, toxic substances, endocrine disorders, and neoplasias (Box 31-2).

*Inflammatory diarrhea*, also described as *invasive or severe diarrhea*, or dysentery, results from cellular damage to the intestinal mucosa, leading to the hypersecretion of water, electrolytes, blood, mucus, and plasma proteins. Inflammatory diarrhea most commonly is caused by invasive bacterial and parasitic pathogens that produce dysenteric illnesses (see Box 31-1). Some noninfectious causes of inflammatory diarrhea include chemotherapy, radiation therapy, hypersensitivity reactions, autoimmune disorders, ischemic colitis, and inflammatory bowel disease. With inflammatory diarrhea, fecal leukocytes and erythrocytes typically are present, as are systemic symptoms, and the diarrhea continues despite fasting.

*Osmotic diarrhea* occurs with the ingestion or malabsorption of osmotically active solutes. These solutes cause the osmotic movement of water into the intestinal lumen, which then overwhelms the gut’s ability to reabsorb it. Examples include the effects of osmotic laxatives and carbohydrate malabsorption. Steatorrhea results from osmotic effects of lipids not absorbed in malabsorption and malabsorption syndromes.

*Abnormal motility* generally is seen in patients with chronic diarrhea but is also a component of acute diarrhea. Hypermotility decreases contact time between luminal contents and the absorbing mucosa, limiting water and electrolyte absorption.
Causative Agents of Infectious Diarrhea

**Viral (60% of Cases)**
- Astrovirus
- Calicivirus
- Coronavirus
- Cytomegalovirus* 
- Enteric adenovirus
- Hepatitis A through G
- Herpes simplex virus
- HIV enteropathy
- Norwalk-like agents
- Norwalk virus
- Pararotavirus
- Picornavirus
- Rotavirus
- Small round viruses

**Bacterial (20% of Cases)**
- *Invasive* 
  - Aeromonas species
  - Campylobacter species
  - Clostridium difficile
  - Enteroinvasive *Escherichia coli*
  - Mycobacterium species
  - Plesiomonas shigelloides
  - Salmonella species
  - Shigella species
  - *Vibrio fluvialis*
  - *Vibrio parahaemolyticus*
  - *Vibrio vulnificus*
  - *Yersinia enterocolitica*
  - *Yersinia pseudotuberculosis*

**Toxigenic**
- Food poisoning with preformed toxins
  - Bacillus cereus
  - Clostridium botulinum
  - Staphylococcus aureus
  - Clostridium perfringens
  - Enterohemorrhagic *E. coli* O157:H7
  - Enterotoxigenic *E. coli*
  - Klebsiella pneumoniae
  - *Shigella* species
  - *Vibrio cholerae*

**Other Bacteria**
- Parasitic (5% of Cases)
  - Protozoa
    - *Balantidium coli* 
    - *Blastocystis hominis*
    - Cryptosporidium
    - Cyclospora
    - Entamoeba histolytica*
    - Entamoeba polecki
    - Enteromonas hominis
    - Giardia lamblia
    - Isospora belli
    - Microsporida
    - Sarcocystis hominis
  - Helminths
    - Angiostrongylus costaricensis
    - Anisakiasis
    - *Ascaris lumbricoides*
    - Diphyllolothrium latum
    - Enterobius vermicularis
    - Hookworms
    - *Schistosoma* species
    - Strongyloides stercoralis
    - *Taenia* species
    - *Trichinella spiralis*
    - *Trichuris trichiura*

**HIV,** human immunodeficiency virus.
* Associated with fever, abdominal pain, and fecal red blood cells or white blood cells. % indicates the estimated contribution to total cases.

Causes of Noninfectious Diarrhea

**Toxins**
- Drugs
  - ACE inhibitors
  - Alprazolam (Xanax)
  - Antacids (magnesium)
  - Antibiotics
  - Antidepressants
  - Antihypertensives
  - Antiparkinson drugs
  - Beta-blockers
  - Caffeine
  - Cardiac antiarrhythmics
  - Chemotherapy agents
  - Cholesterol-lowering drugs
  - Cholinergic agents
  - Cholinesterase inhibitors
  - Colchicine
  - Digitalis
  - Diuretics
  - Fluorouracil
  - Fluoxetine (Prozac)
  - Histamine H2-receptor antagonists
  - Hydralazine
  - Lactulose
  - Laxatives, cathartics
  - Levodopa
  - Lithium
  - NSAIDs
  - Neomycin
  - Podophyllin
  - Procainamide
  - Prostaglandins
  - Quinidine
  - Ricinoleic acid
  - Theophylline
  - Thyroid hormone
  - Valproic acid

**Dietetic Foods**
- Mannitol
- Sorbitol
- Xylitol

**Fish-Associated Toxins**
- Amnestic shellfish poisoning
- Ciguatera
- Echinoderms
- Neurotoxic shellfish poisoning

Continued
**Box 31-2 Causes of Noninfectious Diarrhea—cont’d**

- Paralytic shellfish poisoning
- Scombroid
- Tetrodotoxin

**Plant-Associated Toxins**
- Herbal preparations
- Horse chestnut
- Mushrooms—*Amanita* species
- Nicotine
- Other plant toxins—pesticides—organophosphates
- Pokeweed
- Rhubarb

**Miscellaneous**
- Allergic reactions
- Carbon monoxide poisoning
- Ethanol
- Heavy metals
- Monosodium glutamate (MSG)
- Opiate withdrawal

**Gastrointestinal Pathology**
- Appendicitis
- Autonomic dysfunction
- Bile acid malabsorption
- Blind loop
- Bowel obstruction
- Celiac disease
- Cirrhosis
- Defects in amino acid transport
- Diverticular disease
- Familial dysautonomia
- Fecal impaction
- Fecal incontinence
- GI bleed
- GI cancer
- Hirschsprung’s disease
- Inflammatory bowel disease (ulcerative colitis, Crohn’s disease)
- Intussusception
- Irritable bowel syndrome
- Ischemic bowel
- Lactose or fructose intolerance
- Malabsorption syndromes
- Malrotation
- Postsurgical
- Postvagotomy
- Radiation therapy
- Short gut syndrome
- Small bowel resection
- Strictures
- Toxic megacolon
- Tropical sprue
- Volvulus
- Whipple’s disease

**Endocrine-Related Conditions**
- Carcinoid syndrome (serotonin)
- Hormonal hypersecretion
- Hyperthyroidism (thyroid hormone)
- Medullary carcinoma of the thyroid (calcitonin)
- Pancreatic cholera (VIP)
- Somatostatinoma (somatostatin)
- Systemic mastocytosis (histamine)
- Zollinger-Ellison syndrome (gastrin)

**Endocrine Pathology**
- Adrenal insufficiency
- Diabetes enteropathy
- Hypoparathyroidism
- Pancreatic insufficiency

**Systemic Illness and Other Causes**
- Alcoholism
- Amyloidosis
- Connective tissue disease
- Cystic fibrosis
- Ectopic pregnancy
- Hemolytic-uremic syndrome
- Henoch-Schönlein purpura
- Lymphoma
- Otitis media—infants
- Pelvic inflammatory disease
- Pneumonia, sepsis
- Pyelonephritis
- Scleroderma, SLE
- Stevens-Johnson syndrome
- Toxoplasmosis
- Wilson’s disease
- Miscellaneous
- Factitious diarrhea
- Runner’s diarrhea

ACE, angiotensin-converting enzyme; GI, gastrointestinal; NSAIDs, nonsteroidal anti-inflammatory drugs; SLE, systemic lupus erythematosus; VIP, vasoactive intestinal polypeptide.

### CLINICAL APPROACH

#### Emergency Assessment and Stabilization

An immediate assessment should be made of the patient’s stability, including maintenance of the airway, adequacy of oxygenation and ventilation, and circulation, with particular attention to volume status. Tachycardia, orthostatic hypotension, poor skin turgor and color, diaphoresis, and mental status changes all are characteristic of hypovolemia and hypoperfusion. The bedside ultrasound can be used quickly to check volume status. Patients with a poorly filled, rapidly beating heart and a collapsed inferior vena cava require immediate rehydration.\(^9\)\(^10\) Associated septic shock may contribute to the hypotension and general organ hypoperfusion, and diarrhea may be a manifestation of toxic shock syndrome. A diarrhea-associated acid-base disorder should be suspected in patients with Kussmaul’s respirations, a significant anion gap on basic metabolic panel reflecting lactic acidosis from significant volume loss, or a nonanion gap metabolic acidosis associated with massive bicarbonate loss. After stabilization, a secondary survey may elucidate the potential cause of the diarrhea and direct further evaluation and treatment.

#### Secondary Survey

The physical examination should assess the patient’s overall health, toxicity, fever, volume status, and signs of a surgical abdomen and determine the presence of blood in the stool. Young healthy adults may maintain a normal blood pressure and heart rate even with significant dehydration. In patients who are taking antiarrhythmic or beta-blocker medications or have conduction disease or fixed-paced rhythms, heart rate may not be a reliable indicator of volume status. Signs of volume depletion and impending shock include dry mucosa, poor skin turgor, decreased urine output, and mental status changes. Children will have sunken eyes, depression of the fontanel, decrease in urine output (number of wet diapers), and decrease in alertness and activity.\(^11\)
### Table 31-1  Factors Increasing Probability of Nonbenign Diarrhea

<table>
<thead>
<tr>
<th>FACTOR</th>
<th>SPECIFIC PATHOGEN(S) AND OTHER CONSIDERATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presentation to a health care facility</td>
<td>Degree of illness overall greater in patients seeking evaluation; increased probability of “not norovirus” cause to 50%</td>
</tr>
<tr>
<td>Travel history</td>
<td>Especially foreign travel and to endemic areas of dysenteric disease</td>
</tr>
<tr>
<td>Recent hospitalization</td>
<td><em>Clostridium difficile</em> from antibiotic exposure</td>
</tr>
<tr>
<td>Day care attendance</td>
<td>Rotavirus, <em>Shigella</em>, <em>Giardia</em></td>
</tr>
<tr>
<td>Nursing home residence</td>
<td><em>C. difficile</em>, medication side effects, tube feedings, ischemic colitis, fecal impaction, and overflow diarrhea</td>
</tr>
<tr>
<td>Wilderness exposure</td>
<td><em>Giardia</em> or <em>Cryptosporidium</em></td>
</tr>
<tr>
<td>Antibiotic therapy</td>
<td><em>C. difficile</em>, antibiotic side effects</td>
</tr>
<tr>
<td>Raw shellfish, farm animals and fair livestock, pet reptiles or amphibians, petting zoos</td>
<td><em>Salmonella</em> species, <em>Escherichia coli</em> O157:H7 and non-O157 Shiga toxin—producing <em>E. coli</em>, <em>Vibrio</em> species</td>
</tr>
<tr>
<td>Epidemic of multiple patients with a short time of onset</td>
<td>Norovirus; less commonly, <em>Campylobacter jejuni</em>, <em>Salmonella</em> species, <em>Cryptosporidium</em></td>
</tr>
<tr>
<td>Acute vomiting and diarrhea after eating suspected contaminated food</td>
<td><em>Bacillus cereus</em>, <em>Clostridium botulinum</em>, <em>Staphylococcus aureus</em></td>
</tr>
<tr>
<td>Epidemic of severe gastroenteritis traced to eggs, poultry, meat, or dairy products</td>
<td><em>C. jejuni</em>, <em>Salmonella</em> species</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td><em>Giardia lamblia</em>, <em>Entamoeba histolytica</em></td>
</tr>
<tr>
<td>Nausea, vomiting</td>
<td>Severe bacterial infections: <em>Salmonella</em>, <em>Campylobacter</em>, <em>Shigella</em>, EPEC, <em>Yersinia</em> or <em>Vibrio</em> species</td>
</tr>
<tr>
<td>Bloody stool</td>
<td>Also consider surgical abdomen, GI bleeding</td>
</tr>
<tr>
<td>Fever</td>
<td>Inflammatory bowel disease</td>
</tr>
<tr>
<td>Rectal pain</td>
<td></td>
</tr>
<tr>
<td>Tenesmus</td>
<td></td>
</tr>
<tr>
<td>Diarrhea &gt;7-14 days’ duration</td>
<td>Protozoa and microsporidia, <em>C. difficile</em>, <em>Campylobacter</em>, Shiga toxin—producing <em>E. coli</em></td>
</tr>
<tr>
<td>Hemolytic uremic syndrome</td>
<td><em>E. coli</em> O157:H7 or other species</td>
</tr>
<tr>
<td>Stool WBC count</td>
<td>Not reliable for diagnosis of bacterial cause</td>
</tr>
<tr>
<td>Colonic ulcerations</td>
<td>Inflammatory bowel disease</td>
</tr>
<tr>
<td>Proctitis</td>
<td>Bacterial cause highly probable</td>
</tr>
<tr>
<td>Pseudomembranes</td>
<td>Toxic megacolon, <em>C. difficile</em></td>
</tr>
<tr>
<td>Chronic disease (e.g., cirrhosis, DM)</td>
<td>Complicated course expected with any form of diarrheal illness</td>
</tr>
<tr>
<td>Organ transplantation</td>
<td>Abnormally severe illness from rotavirus and adenovirus</td>
</tr>
<tr>
<td>Increased frequency of cytomegalovirus</td>
<td></td>
</tr>
<tr>
<td>Severe illness from dysenteric diarrhea</td>
<td></td>
</tr>
<tr>
<td>Spore-forming protozoa and microsporidia</td>
<td></td>
</tr>
<tr>
<td>HIV infection, other immunodeficiency disorders</td>
<td>Severe illness from common bacteria, spore-forming protozoa, and microsporidia</td>
</tr>
<tr>
<td></td>
<td>Increased frequency of cytomegalovirus and <em>Mycobacterium avium</em> complex</td>
</tr>
</tbody>
</table>

*DM, diabetes mellitus; EPEC, enteropathogenic *E. coli*; GI, gastrointestinal; HIV, human immunodeficiency virus; WBC, white blood cell.*

Particular attention should be given to the abdominal examination. Focal abdominal pain with peritoneal findings may be caused by an acute surgical abdomen with symptoms mimicking those of severe gastroenteritis. A rectal examination should be performed to detect fecal impaction, melena, or hematochezia. Gross blood may be consistent with invasive, infectious diarrhea but may be the harbinger of many other pathologic states that manifest with gastrointestinal bleeding. Histamine-induced skin changes may be indicative of an intestinal parasitic infection. The patient should be assessed for specific toxidromes, such as cholinergic or sympathomimetic states that may be clues to a noninfectious cause.

Characterization of the Diarrheal Syndrome

**Acute Infectious Diarrhea.** Most viral and many bacterial agents cause a self-limited, secretory diarrhea that lasts less than 14 days and causes only mild dehydration and minimal systemic symptoms. These infections do not require extensive testing and are treated symptomatically. In the United States, monitoring of pathogens causing this type of acute gastroenteritis demonstrates that 90% of the infections are caused by norovirus species. All other potential causes of diarrhea are highly improbable unless certain historical and clinical findings are present. Bacterial and protozoal agents less commonly cause diarrhea syndromes indistinguishable from norovirus infection with a nontoxic, self-limited course. A Bayesian approach to diagnosing and treating acute diarrhea has been proposed. The clinical evaluation should screen for all factors (Table 31-1) that may change the probability of “not norovirus” from 10% to 50% or greater. With one or more of these findings present, empirical antibiotic or other specific therapy may be indicated, as well as clinical testing to determine the exact causative disorder.
Persistent diarrhea is defined as that lasting for more than 14 days, and chronic diarrhea, more than 30 days. Infectious agents of persistent and chronic diarrhea include bacteria, parasites, and rarely viruses. Common bacterial pathogens include Aeromonas, Plesiomonas, Campylobacter, Clostridium difficile, Salmonella, and Mycobacterium tuberculosis. Parasites causing chronic diarrhea are colonic forms such as Amoeba, Trichuris, Yersinia, and Schistosoma species or small intestinal pathogens such as Giardia, Cryptosporidium, Cyclospora, Isospora, and Strongyloides. In developing countries, chronic diarrhea is more likely to have a bacterial cause. In developed countries, chronic diarrhea is caused by noninfectious disorders such as irritable bowel syndrome, malabsorption syndromes, laxative abuse, and inflammatory bowel disease. Testing for HIV or immunodeficiency is important because patients with these conditions commonly have chronic diarrhea. Evaluation should include testing for cryptosporidia, microsporidia, mycobacteria (i.e., Mycobacterium avium complex), herpes simplex virus (HSV), Isospora, Cyclospora, and cytomegalovirus. In addition, parasitic and helminthic infections should be ruled out.

Noninfectious Diarrhea. Noninfectious causes of diarrhea (see Box 31-2) are responsible for approximately 15% of all cases of diarrheal illness. The distinction between infectious and noninfectious causes may not be clinically apparent. A complete evaluation should consider possible surgical pathology of the abdomen, including gastrointestinal bleeding, ischemic bowel, acute appendicitis, intussusception, and inflammatory bowel obstruction. The differential diagnosis also includes possible toxic exposures or ingestions, such as heavy metal poisoning, or ingestion of plant-borne or fish-borne toxins. Endocrine pathology, such as adrenal insufficiency, hyperthyroidism, diabetic enteropathy, and hormone-secreting tumors, and other systemic illnesses should be considered, and special attention should be directed at underlying medical conditions, medication use, and past surgical history.

Ancillary Testing

Most cases of acute diarrhea are self-limited, and laboratory and diagnostic tests should be kept to a minimum unless required for epidemiologic studies. Testing is indicated in patients who have a high probability of a “non-norovirus” clinical picture and have worrisome historical data, signs, and symptoms associated with an increased probability of those causes. Ancillary testing should never compromise empirical treatment when indicated (as discussed later). Fever with a toxic appearance and volume depletion, blood- or mucus-containing stools, frequent voluminous stools, and other risk factors (see Table 31-1) should prompt a diligent search for a specific causative disorder in order to guide appropriate therapy. A white blood cell count is rarely helpful and not sensitive or specific enough to aid in diagnostic decision-making, although hemoglobin determination is useful to screen for anemia from blood loss, and abnormalities in platelet and coagulation parameters may contribute to identification of a cause for gastrointestinal bleeding. A comprehensive chemistry panel, including renal function tests, can be important when significant volume loss is suspected or when significant diarrhea has been present for 48 to 72 hours. Liver function studies, thyroid tests, serum lipase assay, and a pregnancy test may be helpful in selected cases.

Hemoccult and fecal cell count: The presence of fecal leukocytes is not specific or sensitive enough to use as the sole criterion to decide which patients with presumed bacterial gastroenteritis should be treated empirically with antibiotics. With inflammatory diarrhea of various causes, red and white blood cells are seen on stool examination. Included are bacterial, parasitic, and noninfectious causes, such as chemotherapy, radiation therapy, hypersensitivity reactions, autoimmune disorders, and inflammatory bowel disease. The presence of fecal leukocytes does not delineate which patients would benefit from empirical antimicrobial therapy. The presence of blood does not always correlate with the presence of fecal leukocytes, so reliance on positive stool guaiac test result alone as a rationale for antibiotic therapy is not recommended. The presence of blood without fecal leukocytes may indicate amebiasis, malignancies, heavy metal poisoning, fissures, hemorrhoids, bowel ischemia, or primary gastrointestinal bleeding.

Assays for calprotectin and lactoferrin, produced by leukocytes, are sensitive and specific and may be more useful than microscopic examination of the stool, but these tests are rarely, if ever, of use in the ED.

C. difficile toxin assay: This test is indicated if the patient reports recent antibiotic use. C. difficile–associated diarrhea most commonly occurs during or shortly after the antibiotic course. In 25% to 40% of cases, however, onset of the diarrhea may be delayed as long as 12 weeks after antibiotic therapy. The most commonly implicated antibiotics are cephalosporins, penicillins, and clindamycin. Although C. difficile accounts for only 10 to 20% of antibiotic-associated diarrhea, an assay for C. difficile toxin gives a positive result in nearly all cases of antibiotic-associated pseudomembranous colitis. Approximately 3% of adult patients and 65% of newborns may be colonized with C. difficile.

Escherichia coli O157:H7 toxin assay: This test is considered in endemic areas and in patients with suspected hemolytic-uremic syndrome.

Stool culture for bacteria: Stool cultures may be warranted in patients who are febrile, toxic appearing, immunocompromised, at the extremes of age, experiencing a prolonged course, or not responding to conventional treatment. Studies have shown a 2% positive rate, thus proving that routine cultures are of limited value.

Stool examination for ova and parasites: The assessment of stool for ova and parasites is not routinely recommended. This study is used in patients with chronic diarrhea (Entamoeba histolytica, Cryptosporidium); patients with a history of travel to developing countries, particularly to Nepal or areas of Russia (Cryptosporidium, Giardia, Cyclospora); patients with exposure to infants in daycare centers (Cryptosporidium, Giardia); and patients with HIV infection (E. histolytica, Giardia).

Giardia antigen assay and serologic testing for amebiasis may be considered in patients exposed to poor sanitation, HIV-infected patients, patients with a history of travel to developing countries, patients with a history of backpacking, and patients with daycare exposures.

Urinalysis: A urinalysis and a urine pregnancy test should be obtained only when urinary tract infection is a possibility, a gastrointestinal origin for the symptoms is not clear, or pregnancy is suspected.

Radiographic studies: Plain radiographs and contrast computed tomography (CT) may be indicated for patients thought to have a surgical abdomen and to identify pathologic abnormalities, such as tumor, obstruction, free air, fistulae, blind loops, and those associated with Crohn’s disease.

Gastrointestinal referral: Referral may be indicated in the evaluation of chronic diarrhea and for workup beyond the scope of the ED (e.g., endoscopy, further stool studies, biopsy).

EMPIRICAL MANAGEMENT

Oral rehydration is the treatment of choice for mild to moderate fluid losses (Fig. 31-1). Oral rehydration can be accomplished with sports beverages, commercial rehydration solutions, or a balanced clear liquid diet in the home (e.g., consisting of water,
salt-containing liquids such as canned soups, and potassium from oranges or bananas). The WHO has defined an oral rehydration solution (WHO-ORS) that can be made by dissolving the following in 1 L of clean water:
- 3.5 g of sodium chloride
- 2.9 g of trisodium citrate or 2.5 g of sodium bicarbonate
- 1.5 g of potassium chloride
- 20 g of glucose or 40 g of sucrose

Replacement of micronutrients, particularly copper and zinc, has been recommended, especially in developing countries. The concept of bowel rest has been abandoned because it may worsen diarrhea and lead to more severe dehydration. The choice of oral rehydration fluids depends on the extent of dehydration and the underlying health of the patient. In otherwise healthy patients with mild to moderate dehydration, fluids such as sports drinks, diluted fruit juices, and soft drinks supplemented with soups, broths, or crackers may be sufficient to replace the fluid and sodium losses associated with acute diarrhea. Such frequently used “clear liquids” may contain excess sugars and insufficient sodium content, however, leading to an osmotic diarrhea. Beverages containing caffeine should be avoided because caffeine increases cyclic adenosine monophosphate levels and may cause a secretory diarrhea. Milk and other products containing lactose also should be avoided because viral and bacterial pathogens, responsible for many cases of diarrhea, may cause a transient lactase deficiency, leading to malabsorption and osmotic diarrhea. Food intake is encouraged, but foods high in simple sugars should be avoided because the osmotic effect is counterproductive. Foods with a high fat content may delay gastric emptying and should be avoided. The BRAT (bananas, rice, apples, and toast) diet has long been recommended, particularly with pediatric patients. Although no controlled studies have examined the efficacy of the BRAT diet, it remains a commonly recommended strategy. The pectin in the peel of apples is constipating (pectin, found in fruit peel, is the "pectate" in Kapectate), and bananas provide potassium. If this diet is used for extended periods, adequate provision of protein and energy needs of the patient becomes a concern.

In patients with evidence of more severe dehydration, intravenous fluid resuscitation with normal saline or lactated Ringer’s solution is the preferred treatment. Pediatric patients should receive a bolus of 20 mL/kg of normal saline, which may be repeated as indicated. Specific treatment for diarrhea should be directed toward the suspected cause. In patients with suspected surgical pathology, further diagnostic testing and surgical consultation may be required. With toxic exposures, treatment consists of early decontamination, supportive care, and, if appropriate, administration of specific antidotes. Other noninfectious causes of diarrhea are treated as indicated.

Because the specific pathogen causing infectious diarrhea is rarely identified in the ED and the results of cultures are usually unavailable, any antimicrobial treatment is empirical and guided by knowledge of the common causes of infectious diarrhea (see Box 31-1). Viral and noninvasive bacterial gastroenteritis tend to be self-limiting and require only supportive therapy. Empirical antibiotic treatment is directed against invasive bacterial and parasitic organisms that cause the greatest harm. Antibiotic treatment is initiated in patients with a suspected invasive process and severe diarrhea, systemic symptoms, fever, or abdominal pain and in patients who appear toxic. The current recommendation for empirical treatment of a systemically ill—appearing adult is ciprofloxacin, 500 mg orally twice a day, or levofloxacin, 500 mg orally every 24 hours for 3 to 5 days. Fluoroquinolones are efficacious against most organisms that cause dysenteric illnesses and have been shown to be more effective than trimethoprim-sulfamethoxazole. Fluoroquinolones should not be administered to pregnant patients or children younger than 18 years. The antibiotic treatment of severe gastroenteritis in children has been associated with the development of hemolytic-uremic syndrome.

![Figure 31-1](image-url). Approach to the patient with acute diarrhea. Abx, antibiotics; CT, computed tomography; ED, emergency department; GE, gastroenterologist; GI, gastrointestinal; IV, intravenous; temp., temperature; US, ultrasonography; VSs, vital signs.)
Probiotics have been used as an alternative to traditional antibiotic therapy for diarrhea. *Lactobacillus* and other bacteria have proved to be effective in restoring the normal gastrointestinal flora that is disrupted during diarrhea illness. This approach has been most effective with traveler’s diarrhea and nonspecific diarrhea in children.

**DISPOSITION**

Most patients with uncomplicated, acute diarrhea can be discharged home after assessment and symptomatic relief. Hospitalization rarely is required for diarrhea secondary to viral and many forms of bacterial gastroenteritis, which tend to be self-limiting. Often the exact causative agent of diarrhea is not identified in the ED. An understanding of common causes and their treatment and recognition of patients at risk for a more severe clinical course are essential for the appropriate disposition. In patients with severe dehydration, hemodynamic instability, or a toxic appearance and in high-risk groups, hospital admission is warranted for continuous monitoring, further treatment, and definitive management when initial evaluation and stabilization are complete.

*The references for this chapter can be found online by accessing the accompanying Expert Consult website.*
References