

THE DIGESTIVE SYSTEM

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CHAPTER 126

Digestive System Assessment

HISTORY

After identifying the major gastrointestinal (GI) symptoms, the onset and progression should be determined (improved, unchanged, worsening, involvement of other organ systems). Characterization of signs and symptoms should identify factors that trigger, aggravate, or alleviate the symptoms; the timing, frequency, and duration of symptoms; relationship to meals and defecation; and associated symptoms (e.g., fever or weight loss). Other key history includes exposures to others (family, school contacts, pets), travel, environmental exposure, and impact of illness on the child (school absences, weight loss).

PHYSICAL EXAMINATION

The history may suggest a diagnosis and direct the evaluation, which should include a full examination as well as a thorough abdominal examination. Extraintestinal disorders may produce GI manifestations (e.g., emesis with group A streptococcal pharyngitis, abdominal pain with lower lobe pneumonia, respiratory distress with COVID-19). The examination should begin with a careful external visual inspection for abdominal distention, bruising or discoloration, abnormal abdominal veins, jaundice, surgical scars, and ostomies. Abnormalities of intensity and pitch of bowel sounds can occur with bowel obstruction. When palpating for tenderness, the examiner should note location, facial expression, guarding, and rebound tenderness. Palpation can also detect enlargement of the liver or spleen as well as feces and masses. If detected, organomegaly should be measured (with a tape measure), noting tenderness or abnormal firmness or contour. A rectal examination, including inspection for fissures, skin tags, abscesses, and fistulous openings, may be performed for children with history suggesting constipation, GI bleeding, abdominal pain, chronic diarrhea, and suspicion of inflammatory bowel disease (IBD). Digital rectal examination should include assessment of anal sphincter tone, anal canal size and elasticity, tenderness, extrinsic masses, presence of fecal impaction, and caliber of the rectum. Stool should be tested for occult blood.

SCREENING TESTS

A **complete blood count** may provide evidence for inflammation (white blood cell [WBC] and platelet count), poor nutrition

or bleeding (hemoglobin, red blood cell [RBC] volume, reticulocyte count), and infection (WBC number and differential, presence of toxic granulation). Serum electrolytes, blood urea nitrogen (BUN), and creatinine help define hydration status. Tests of liver dysfunction include total and direct bilirubin, alanine aminotransferase, aspartate aminotransferase for evidence of hepatocellular injury, and γ -glutamyltransferase or alkaline phosphatase for evidence of bile duct injury. **Hepatic synthetic function** can be assessed by coagulation factor levels, prothrombin time, and albumin level. Pancreatic enzyme tests (amylase, lipase) provide evidence of pancreatic injury or inflammation. Urinalysis can gauge dehydration and identify a possible source of protein loss. Stool may be examined for blood or calprotectin (inflammation).

DIAGNOSTIC IMAGING

Radiology

A plain abdominal x-ray to document excessive retained stool when history is consistent with constipation and encopresis is not necessary, as physical examination alone can confirm the diagnosis. A kidney-ureter-bladder (KUB) x-ray may demonstrate lower lobe pneumonia.

Endoscopy

Endoscopy permits the direct visualization of the interior of the gut. **Video endoscopes** may be used in very small infants by pediatric gastroenterologists. **Wireless capsule endoscopy** (Fig. 126.1) enables visualization of lesions beyond the reach of conventional endoscopes.

Consultation with a pediatric gastroenterologist for endoscopy is recommended for further evaluation of suspected esophageal or gastric inflammation unresponsive to medications, to confirm the diagnosis of eosinophilic esophagitis or celiac disease, to evaluate GI bleeding, to investigate suspected IBD, or to screen for polyp disorders. In addition, a trained endoscopist can remove esophageal and gastric foreign bodies, place feeding tubes, and treat bleeding ulcers or varices.

COMMON MANIFESTATIONS OF GASTROINTESTINAL DISORDERS

Abdominal Pain

General Considerations

Abdominal pain can result from injury to the intraabdominal organs or overlying somatic structures in the abdominal wall, or from extraabdominal diseases. **Visceral pain** results

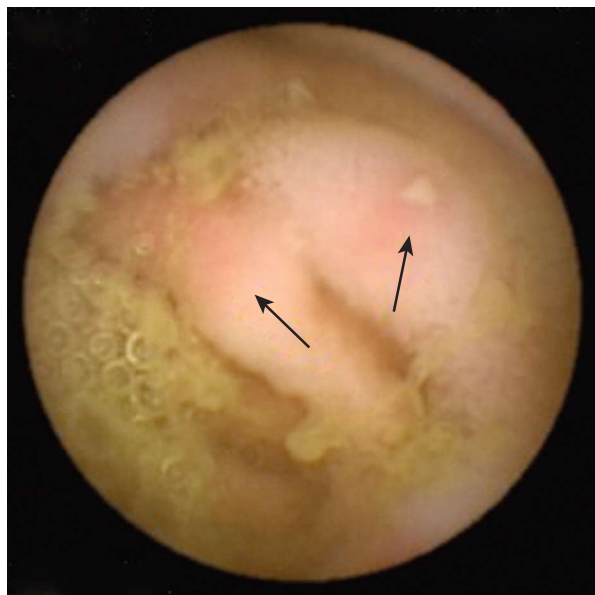


FIGURE 126.1 Wireless capsule endoscopy: Aphthous ulcers (arrows) in jejunum diagnostic of Crohn disease in patient with negative findings on upper endoscopy and colonoscopy.

when autonomic nerves within the gut detect injury, transmitting sensation by nonmyelinated fibers. The pain is vague, dull, slow in onset, and poorly localized. A variety of stimuli, including normal peristalsis and various intraluminal chemical and osmotic states, activate these fibers to some degree. Regardless of the stimulus, visceral pain is perceived when a threshold of intensity or duration is crossed. Lower degrees of activation may result in perception of nonpainful or perhaps vaguely uncomfortable sensations, whereas more intensive stimulation of these fibers results in pain. Overactive sensation may be the basis of some kinds of abdominal pain, such as **functional abdominal pain** and **irritable bowel syndrome (IBS)**.

In contrast to visceral pain, **somatic pain** results when overlying body structures are injured. Somatic structures include the parietal peritoneum, fascia, muscles, and skin of the abdominal wall. In contrast to pain emanating from visceral injury, somatic nociceptive fibers are myelinated and are capable of rapid transmission of well-localized painful stimuli. When intraabdominal processes cause inflammation or injury to the parietal peritoneum or abdominal wall structures, poorly localized visceral pain becomes well-localized somatic pain. In acute appendicitis, the initial activation of visceral nociceptive fibers yields poorly localized discomfort in the mid-abdomen. When the inflammatory process extends to the overlying parietal peritoneum, the pain becomes severe and localizes to the right lower quadrant. This is called **somatoparietal pain**.

Referred pain is a painful sensation in a body region distant from the true source of pain. The location of referred pain is predictable based on the locus of visceral injury. Stomach pain is referred to the epigastric and retrosternal regions, and liver and pancreas pain is referred to the epigastric region. Gallbladder pain often is referred to the region below the right scapula. Somatic pathways stimulated by small bowel visceral afferents affect the periumbilical area, and colonic injury results in infraumbilical referred pain.

Acute Abdominal Pain

Distinguishing Features

Acute abdominal pain can signal the presence of a dangerous intraabdominal process (e.g., appendicitis or bowel obstruction) or may originate from extraintestinal sources (e.g., lower lobe pneumonia or urinary tract stone). Not all episodes of acute abdominal pain require emergency intervention. Appendicitis and volvulus, for example, must be ruled out as quickly as possible. Few patients presenting with acute abdominal pain actually have a surgical emergency, but they must be separated from cases that can be managed conservatively.

Initial Diagnostic Evaluation

Table 126.1 lists a diagnostic approach to acute abdominal pain in children. Events that occur with a discrete, abrupt onset, such as passage of a stone, perforation of a viscus, or vascular infarction, result in a sudden **onset of pain**. Gradual onset of pain is common with infectious or inflammatory causes, such as appendicitis and IBD.

A standard group of laboratory tests usually is performed for acute abdominal pain (see Table 126.1). An abdominal x-ray series evaluates for bowel obstruction, fecalith, or nephrolithiasis. Ultrasound or computed tomography (CT) can visualize the appendix if appendicitis is suspected but the diagnosis remains in doubt. If the initial evaluation suggests intussusception, ultrasound followed by a barium or pneumatic (air) enema may be used to initially diagnose and then treat this condition (see Chapter 129).

Differential Diagnosis

Table 126.2 lists the differential diagnosis of acute abdominal pain in children. The urgent task of the clinician is to rule out surgical emergencies. In young children, malrotation with volvulus, incarcerated hernia, congenital anomalies, and intussusception are common concerns. In older children and teenagers, appendicitis is more common. An acute surgical abdomen is characterized by signs of peritonitis, including tenderness, abdominal wall rigidity, guarding, and absent or diminished bowel sounds. Helpful characteristics of onset, location, referral, and quality of pain are noted in Table 126.3.

Functional Abdominal Pain and Irritable Bowel Syndrome

Recurrent abdominal pain is a common problem, affecting more than 10% of all children. The peak incidence occurs between ages 7 and 12 years. Although the differential diagnosis of recurrent abdominal pain is fairly extensive (Table 126.4), most children do not have a serious (or even identifiable) underlying illness causing the pain.

Differential Diagnosis

Children with **functional abdominal pain** characteristically have pain almost daily. The pain is not associated with meals or relieved by defecation and is often associated with a tendency toward anxiety and perfectionism. Symptoms are often exacerbated by stress at school or novel social situations. The pain often is worst in the morning and often prevents or delays children from attending school. IBS is a subset of functional abdominal pain, characterized by onset of pain at the time of a change in stool frequency or consistency, and relief of pain with defecation. Symptoms in IBS are linked to gut motility.

TABLE 126.1 Diagnostic Approach to Acute Abdominal Pain

HISTORY	
Onset	Sudden or gradual, prior episodes, association with meals, history of injury
Nature	Sharp vs dull, colicky or constant, burning
Location	Epigastric, periumbilical, generalized, right or left lower quadrant, change in location over time
Associated symptoms	Fever, vomiting (bilious?), diarrhea (bloody?), abdominal distention
Extraintestinal symptoms	Cough, dyspnea, dysuria, urinary frequency, flank pain, hematuria
Course of symptoms	Worsening or improving, change in nature or location of pain, aggravating and alleviating factors, last menstrual period
PHYSICAL EXAMINATION	
General position	Growth and nutrition, general appearance, hydration, degree of discomfort, body position
Abdominal	Tenderness, distention, bowel sounds, rigidity, guarding, mass
Genitalia	Testicular torsion, hernia, pelvic inflammatory disease, ectopic pregnancy
Surrounding structures	Breath sounds, rales, rhonchi, wheezing, flank tenderness, tenderness of abdominal wall structures, ribs, costochondral joints
Rectal examination	Perianal lesions, stricture, tenderness, fecal impaction, blood
LABORATORY	
CBC, C-reactive protein, ESR	Evidence of infection or inflammation
AST, ALT, GGT, bilirubin	Biliary or liver disease
Amylase, lipase	Pancreatitis
Urinalysis	Urinary tract infection; bleeding due to stone, trauma, or obstruction
Pregnancy test	Ectopic pregnancy
RADIOLOGY	
Plain flat and upright abdominal films	Bowel obstruction, appendiceal fecalith, free intraperitoneal air, kidney stones, constipation, mass, lower lobe pneumonia
CT or MRI scan	Intraabdominal or pelvic abscess, appendicitis, Crohn disease, pancreatitis, gallstones, kidney stones
Barium enema	Intussusception, malrotation
Ultrasound	Gallstones, appendicitis, intussusception, pancreatitis, kidney stones
Colonoscopy	Colitis
Upper endoscopy	Suspected peptic ulcer or esophagitis

ALT, Alanine aminotransferase; AST, aspartate aminotransferase; CBC, complete blood count; CT, computed tomography; ESR, erythrocyte sedimentation rate; GGT, γ -glutamyltransferase; MRI, magnetic resonance imaging.

TABLE 126.2 Differential Diagnosis of Acute Abdominal Pain

TRAUMATIC	
Duodenal hematoma	
Ruptured spleen	
Perforated viscus	
Traumatic pancreatitis	
FUNCTIONAL	
Constipation*	
Irritable bowel syndrome*	
Dysmenorrhea*	
Mittelschmerz (ovulation)*	
Infantile colic*	
Abdominal migraine	
INFECTIOUS	
Appendicitis*	
Viral or bacterial gastroenteritis/mesenteric adenitis*	
Abscess	
Spontaneous bacterial peritonitis	
Pelvic inflammatory disease	
Cholecystitis	
Urinary tract infection*	
Pneumonia	
Bacterial typhilitis	
Hepatitis/perihepatitis	
GENITAL	
Testicular torsion/epididymitis	
Ovarian torsion	
Ruptured ovarian cyst	
Ectopic pregnancy	
GENETIC	
Sickle cell crisis*	
Familial Mediterranean fever	
Porphyria	
Hereditary angioedema	
METABOLIC	
Diabetic ketoacidosis	
Fabry disease	
INFLAMMATORY	
Inflammatory bowel disease*	
Vasculitis	
Henoch-Schönlein purpura* (IgA vasculitis)	
Pancreatitis	
OBSTRUCTIVE	
Intussusception*	
Malrotation with volvulus	
Ileus*	
Incarcerated hernia	

(Continued)

TABLE 126.2 Differential Diagnosis of Acute Abdominal Pain—cont'd

Postoperative adhesion
Distal intestinal obstruction syndrome (cystic fibrosis)
Duplication cyst
Congenital stricture
BILIARY
Gallstone
Gallbladder hydrops
Biliary dyskinesia
PEPTIC
Gastric or duodenal ulcer
Gastritis*
Esophagitis
RENAL
Kidney stone
Hydronephrosis/ureteropelvic junction obstruction

*Common.

Pain is commonly accompanied in both groups of children by school avoidance, secondary gains, anxiety about imagined causes, lack of coping skills, and disordered peer relationships (Table 126.5).

Distinguishing Features

One needs to distinguish between functional pain/IBS and more serious underlying disorders. **Warning signs** for underlying illness are listed in Table 126.6. If present, further investigation is necessary. Some laboratory evaluation is warranted even in the absence of warning signs. The initial evaluation recommended in Table 126.7 is a sensible approach, avoiding unnecessary testing and providing ample sensitivity for most serious underlying disorders. While waiting for laboratory and ultrasound results, a 3-day trial of a lactose-free diet can evaluate for lactose intolerance. If tests are normal and no warning signs are present, testing should be stopped. If there are warning signs, progression of symptoms, or laboratory abnormalities that suggest a specific diagnosis, additional investigation may be warranted.

Treatment of Recurrent Abdominal Pain

A child who is repeatedly kept home from school because of pain receives reinforcement in the form of being excused from

TABLE 126.3 Distinguishing Features of Abdominal Pain in Children

DISEASE	ONSET	LOCATION	REFERRAL	QUALITY	COMMENTS
Functional: irritable bowel syndrome	Recurrent	Periumbilical, splenic, and hepatic flexures	None	Dull, crampy, intermittent; duration 2 hr	Family stress, school phobia, diarrhea and constipation; hypersensitive to pain from distention
Esophageal reflux	Recurrent, after meals, at bedtime	Substernal	Chest	Burning	Sour taste in mouth; Sandifer syndrome
Duodenal ulcer	Recurrent, before meals, at night	Epigastric	Back	Severe burning, gnawing	Relieved by food, milk, antacids; family history important; gastrointestinal bleeding
Pancreatitis	Acute	Epigastric-hypogastric	Back	Constant, sharp, boring	Nausea, emesis, marked tenderness
Intestinal obstruction	Acute or gradual	Periumbilical–lower abdomen	Back	Alternating cramping (colic) and painless periods	Distention, obstipation, bilious emesis, increased bowel sounds
Appendicitis	Acute	Periumbilical or epigastric; localizes to right lower quadrant	Back or pelvis if retrocecal	Sharp, steady	Nausea, emesis, local tenderness, ± fever, avoids motion
Meckel diverticulum	Recurrent	Periumbilical–lower abdomen	None	Sharp	Hematochezia; painless unless intussusception, diverticulitis, or perforation
Inflammatory bowel disease	Recurrent	Depends on site of involvement		Dull cramping, tenesmus	Fever, weight loss, ± hematochezia
Intussusception	Acute	Periumbilical–lower abdomen	None	Cramping, with painless periods	Guarded position with knees pulled up, currant jelly stools, lethargy
Lactose intolerance	Recurrent with milk products	Lower abdomen	None	Cramping	Distention, gaseousness, diarrhea
Urolithiasis	Acute, sudden	Back	Groin	Severe, colicky pain	Hematuria
Pyelonephritis	Acute, sudden	Back	None	Dull to sharp	Fever, costochondral tenderness, dysuria, urinary frequency, emesis
Cholecystitis and cholelithiasis	Acute	Right upper quadrant	Right shoulder	Severe, colicky pain	Hemolysis ± jaundice, nausea, emesis

Modified from Andreoli TE, Carpenter CJ, Plum F, et al. Cecil Essentials of Medicine. Philadelphia: WB Saunders; 1986.

TABLE 126.4 Differential Diagnosis of Recurrent Abdominal Pain

Functional abdominal pain*
Irritable bowel syndrome*
Chronic pancreatitis
Gallstones
Peptic disease
Duodenal ulcer
Gastric ulcer
Esophagitis
Lactose intolerance*
Fructose malabsorption
Inflammatory bowel disease*
Crohn disease
Ulcerative colitis
Constipation*
Obstructive uropathy
Congenital intestinal malformation
Malrotation
Duplication cyst
Stricture or web
Celiac disease*
Abdominal migraine
Eosinophilic esophagitis*

*Common.

responsibilities and withdraws from full social functioning. This tends to both increase anxiety and prolong the course. To break the cycle of pain and disability, the child with functional pain must be assisted in **returning to normal activities** immediately. Instead of being sent home from school with stomachaches, a child may be allowed to take a short break from class until symptoms abate. The child and parents should be informed that pain is likely to be worse on the day the child returns to school as anxiety worsens dysmotility and enhances pain perception. Medications may be helpful. Probiotics and peppermint oil can be beneficial in treating IBS. Dietary changes such as the low FODMAP (fermentable oligo-di-mono-saccharides and polyols) diet (reducing highly fermentable sugars in the diet) can be beneficial in a subset of patients. In difficult and persistent cases, cognitive behavioral therapy, amitriptyline, or a selective serotonin reuptake inhibitor may be helpful. When significant anxiety or social dysfunction persists, a mental health professional should be consulted.

Vomiting

Vomiting is a coordinated, sequential series of events that leads to forceful oral emptying of gastric contents. It is a common problem in children and has many causes. Vomiting should

TABLE 126.5 Rome IV Criteria for Pediatric Functional Gastrointestinal Syndromes

A. DIAGNOSTIC CRITERIA FOR CHILDHOOD FUNCTIONAL ABDOMINAL PAIN

Must be fulfilled for at least 2 months before diagnosis; must be met at least 4 times per month and include all of the following:

1. Episodic or continuous abdominal pain that does not occur solely during physiological events such as eating and menses
2. Insufficient criteria for other functional gastrointestinal diseases including irritable bowel syndrome, functional dyspepsia, or abdominal migraine.
3. After appropriate evaluation, the abdominal pain cannot be fully explained by another medical condition.

B. DIAGNOSTIC CRITERIA FOR IRRITABLE BOWEL SYNDROME

Must include all of the following:

1. Abdominal pain at least 4 days per month associated with one or more of the following:
 - a. Related to defecation
 - b. A change in the frequency of stool.
 - c. A change in the form (appearance of stool)
2. In children with constipation, the pain does not resolve with resolution of the constipation.
3. After appropriate evaluation, the symptoms cannot be fully explained by another medical condition.

*Criteria fulfilled at least 2 months before diagnosis.

C. DIAGNOSTIC CRITERIA FOR ABDOMINAL MIGRAINE

Must include all of the following occurring at least twice:*

1. Paroxysmal episodes of intense, acute periumbilical midline or diffuse abdominal pain lasting 1 hour or more (should be the most severe and distressing symptom).
2. Episodes are separated by weeks to months.
3. The pain is incapacitating and interferes with normal activities.
4. Stereotypical pattern and symptoms in the individual patient.
5. The pain is associated with 2 or more of the following:
 - a. anorexia
 - b. nausea
 - c. vomiting
 - d. headache
 - e. photophobia
 - f. pallor
6. After appropriate evaluation, the symptoms cannot be fully explained by another medical condition.

*Criteria fulfilled for at least 6 months before diagnosis.

D. DIAGNOSTIC CRITERIA FOR CYCLIC VOMITING SYNDROME

Must include all of the following:

1. The occurrence of 2 or more periods of intense, unremitting nausea and paroxysmal vomiting, lasting hours to days within a 6-month period.
2. Episodes are stereotypical in each patient.
3. Episodes are separated by weeks to months with return to baseline health between episodes.
4. After appropriate medical evaluation, the symptoms cannot be attributed to another condition.

E. FUNCTIONAL CONSTIPATION

Must include 2 or more of the following occurring at least once per week for a minimum of 1 month with insufficient criteria for the diagnosis of irritable bowel syndrome:

1. 2 or fewer defecations in the toilet per week in a child of developmental age of at least 4 years.
2. At least 1 episode of fecal incontinence per week.
3. History of retentive posturing or excessive volitional stool retention.
4. History of painful or hard bowel movements.
5. Presence of a large fecal mass in the rectum.
6. History of large diameter stools that can obstruct the toilet.

After appropriate evaluation, the symptoms cannot be fully explained by another medical condition.

Courtesy of the Rome Foundation.

TABLE 126.6 Warning Signs of Underlying Illness in Recurrent Abdominal Pain

Vomiting
Abnormal screening laboratory study (anemia, hypoalbuminemia, inflammatory markers)
Fever
Bilious emesis
Growth failure
Pain awakening child from sleep
Weight loss
Location away from periumbilical region
Blood in stools or emesis
Delayed puberty
Nocturnal diarrhea
Dysphagia
Arthritis
Perianal disease
Family history of inflammatory bowel disease, celiac disease, or peptic ulcer disease

TABLE 126.7 Suggested Evaluation of Recurrent Abdominal Pain

INITIAL EVALUATION	FOLLOW-UP EVALUATION*
Complete history and physical examination	CT or MRI scan of the abdomen and pelvis with oral, rectal, and intravenous contrast
Ask about “warning signs” (see Table 126.6)	Celiac disease serology—tissue transglutaminase IgA antibody
Determine degree of functional impairment (e.g., missing school)	Barium upper GI series with small bowel follow-through
CBC	Endoscopy of the esophagus, stomach, and duodenum
ESR	Colonoscopy
Amylase, lipase	
Urinalysis	
Abdominal ultrasound—examine liver, bile ducts, gallbladder, pancreas, kidneys, ureters	
Trial of 3-day lactose-free diet	

*Consider using one or more of these to investigate warning signs, abnormal laboratory tests, or specific or persistent symptoms.

CBC, Complete blood count; CT, computed tomography; ESR, erythrocyte sedimentation rate; GI, gastrointestinal.

be distinguished from **regurgitation** of stomach contents, also known as gastroesophageal reflux (GER) or “spitting up.” Although the end result of vomiting and regurgitation is similar, they have completely different characteristics. Vomiting is usually preceded by nausea and is accompanied by forceful gagging and retching. Regurgitation, on the other hand, is effortless and not preceded by nausea.

Differential Diagnosis

In neonates with true vomiting, congenital obstructive lesions should be considered. Allergic reactions to formula in the first

2 months of life may present with vomiting. **Infantile GER** (“spitting up”) occurs in up to half of infants and can be large in volume, but is effortless, and these infants do not appear ill. **Pyloric stenosis** occurs in the first 1–2 months of life and is characterized by steadily worsening, forceful vomiting that occurs immediately after feedings. A visibly distended stomach, often with visible peristaltic waves, is often seen before vomiting. Pyloric stenosis is more common in male infants; the family history may be positive. Other congenital obstructive lesions, such as intestinal duplication cysts, atresias, webs, and midgut malrotation, should be considered. Metabolic disorders (e.g., organic acidemias, galactosemia, urea cycle defects, adrenogenital syndromes) may present with vomiting in infants. In older children with acute vomiting, viruses and food poisoning are common. Other infections, especially streptococcal pharyngitis, urinary tract infections, and otitis media, commonly result in vomiting. When vomiting is chronic, central nervous system (CNS) causes (increased intracranial pressure, migraine) must be considered. **Cyclic vomiting syndrome** or abdominal migraine may be the etiology of recurrent vomiting. When abdominal pain or bilious emesis accompanies vomiting, evaluation for bowel obstruction, peptic disorders, and appendicitis must be immediately initiated.

Distinguishing Features

Table 126.8 lists common diagnoses that must be considered and their important historical features. Given the frequency of viral gastroenteritis as the etiology, it is important to be alert for unusual features that suggest another diagnosis. Viral gastroenteritis usually is not associated with severe abdominal pain or headache and does not recur at frequent intervals.

Physical examination should include assessment of the child’s hydration status, including examination of vital signs as well as capillary refill, moistness of mucous membranes, and skin turgor (see Chapter 38). The chest should be auscultated for evidence of rales or other signs of pulmonary involvement. The abdomen must be examined carefully for distention, organomegaly, bowel sounds, tenderness, and guarding. Testing stool for occult blood should be considered if ulcer disease or severe esophagitis is suspected.

Laboratory evaluation of vomiting should include serum electrolytes, tests of renal function, complete blood count, amylase, lipase, and liver function tests. Additional testing may be required immediately when history and examination suggest a specific etiology. Ultrasound is useful to look for pyloric stenosis, gallstones, renal stones, hydronephrosis, biliary obstruction, pancreatitis, malrotation, intussusception, and other anatomic abnormalities. CT may be indicated to rule out appendicitis or to observe structures that cannot be visualized well by ultrasound. Barium studies can show obstructive or inflammatory lesions of the gut and can be therapeutic, as in the use of contrast enemas for intussusception (see Chapter 129).

Treatment needs to address the consequences and the causes of the vomiting. Dehydration must be treated with fluid resuscitation. This can be accomplished in most cases with oral fluid-electrolyte solutions, but intravenous (IV) fluids may be required. Electrolyte imbalances should be corrected by appropriate choice of fluids. Underlying causes should be treated when possible.

TABLE 126.8 Differential Diagnosis and Historical Features of Vomiting

DIFFERENTIAL DIAGNOSIS	HISTORICAL CLUES
Viral gastroenteritis	Fever, diarrhea, sudden onset, absence of pain
Gastroesophageal reflux	Effortless, not preceded by nausea, chronic
Hepatitis	Jaundice, history of exposure
Extragastrintestinal infections	
Otitis media	Fever, ear pain
Urinary tract infection	Dysuria, unusual urine odor, frequency, incontinence
Pneumonia	Cough, fever, chest discomfort
Allergic	
Milk or soy protein intolerance (infants)	Associated with particular formula or food, blood in stools
Other food allergy (older children)	
FPIES	Acute onset vomiting, diarrhea, dehydration, shock
Peptic ulcer or gastritis	Epigastric pain, blood or coffee-ground material in emesis, pain relieved by acid blockade
Appendicitis	Fever, abdominal pain migrating to the right lower quadrant, tenderness
Pancreatitis	Severe epigastric abdominal pain
Anatomic obstruction	
Intestinal atresia	Neonate, usually bilious, polyhydramnios
Midgut malrotation	Pain, bilious vomiting, GI bleeding, shock
Intussusception	Colicky pain, lethargy, vomiting, currant jelly stools, mass occasionally
Duplication cysts	Colic, mass
Pyloric stenosis	Nonbilious vomiting, postprandial, <4 mo old, hunger, progressive weight loss
Hirschsprung disease	Abdominal distention, lack of stools
Bacterial gastroenteritis	Fever, often with bloody diarrhea
CNS	
Hydrocephalus	Large head, altered mental status, bulging fontanelles
Meningitis	Fever, stiff neck
Migraine syndrome	Attacks scattered in time, relieved by sleep; headache
Cyclic vomiting syndrome	Similar to migraine, usually no headache
Brain tumor	Morning vomiting, accelerating over time, headache, diplopia
Motion sickness	Associated with travel in vehicle
Labyrinthitis	Vertigo
Metabolic disease	Presentation early in life, worsens when catabolic or exposure to substrate
Pregnancy	Morning, sexually active, cessation of menses
Drug reaction or side effect	Associated with increased dose or new medication
Cancer chemotherapy	Temporarily related to administration of chemotherapeutic drugs
Eosinophilic esophagitis	May have dysphagia or abdominal pain

CNS, Central nervous system; FPIES, food protein–induced enterocolitis syndrome; GI, gastrointestinal.

Antiemetic medications should not be prescribed until the etiology of the vomiting is known, and then only for severe symptoms. Drugs that block serotonin 5-HT₃ receptors, such as ondansetron and granisetron, are frequently used for viral gastroenteritis and can improve tolerance of oral rehydration therapy. They are helpful for chemotherapy-induced vomiting, often combined with dexamethasone. No antiemetic should be used in patients with surgical emergencies or when a specific

treatment of the underlying condition is possible. Correction of dehydration, ketosis, and acidosis is helpful to reduce vomiting in most patients with viral gastroenteritis.

Acute and Chronic Diarrhea

Diarrhea is a major cause of childhood morbidity and mortality worldwide. Deaths from diarrhea are rare in

industrialized countries but are common elsewhere. **Acute diarrhea** is a major problem when it occurs with malnutrition or in the absence of basic medical care (see [Chapter 30](#)). In North America, most acute diarrhea is viral and is self-limited, requiring no diagnostic testing or specific intervention. Bacterial agents tend to cause much more severe illness and typically are seen in food-associated outbreaks or in regions with poor public sanitation. Bacterial enteritis should be suspected when there is **dysentery** (bloody diarrhea with fever) and whenever severe symptoms are present. These infections can be diagnosed by stool culture or other nucleic acid assays for specific pathogens. **Chronic diarrhea** lasts more than 2 weeks and has a wide range of possible causes, including serious and benign conditions that are more difficult to diagnose.

Parents use the word *diarrhea* to describe loose or watery stools, excessively frequent stools, or stools that are large in volume. Constipation with overflow incontinence can be mislabeled as diarrhea. A more exact definition of diarrhea is excessive daily stool liquid volume (>10 mL stool/kg body weight/day). Stool texture, volume, and frequency help to characterize the episode of diarrhea.

Differential Diagnosis

Diarrhea may be classified by etiology or by physiologic mechanisms (secretory or osmotic) and chronicity (acute vs chronic). Etiologic agents include viruses, bacteria or their toxins, chemicals, parasites, malabsorbed substances, and inflammation. [Table 126.9](#) lists common causes of diarrhea in childhood. **Secretory diarrhea** occurs when the intestinal mucosa directly secretes fluid and electrolytes into the stool and is the result of inflammation (e.g., IBD, chemical stimulus). Secretion also is stimulated by mediators of inflammation and by various hormones, such as vasoactive intestinal peptide secreted by a neuroendocrine tumor. Cholera is a secretory diarrhea stimulated by the enterotoxin of *Vibrio cholerae*, which causes increased levels of cyclic adenosine monophosphate (cAMP) within enterocytes and leads to secretion into the small-bowel lumen.

Osmotic diarrhea occurs after malabsorption of an ingested substance, which “pulls” water into the bowel lumen. A classic example is the diarrhea of lactose intolerance. Osmotic diarrhea also can result from generalized maldigestion, such as that seen with pancreatic insufficiency or with

TABLE 126.9 Differential Diagnosis of Diarrhea

	INFANT	CHILD	ADOLESCENT
ACUTE			
Common	Gastroenteritis* Systemic infection Antibiotic associated	Gastroenteritis* Food poisoning Systemic infection Antibiotic associated	Gastroenteritis* Food poisoning Antibiotic associated
Rare	Primary disaccharidase deficiency FPIES Hirschsprung toxic colitis Adrenogenital syndrome Opiate withdrawal (neonate)		Hyperthyroidism Appendicitis
CHRONIC			
Common	Postinfectious secondary lactase deficiency Cow's milk/soy protein intolerance Chronic nonspecific diarrhea of infancy (toddler's diarrhea) Celiac disease Cystic fibrosis	Postinfectious secondary lactase deficiency Irritable bowel syndrome Celiac disease Lactose intolerance Giardiasis Inflammatory bowel disease	Irritable bowel syndrome Inflammatory bowel disease Lactose intolerance Giardiasis Laxative abuse (anorexia nervosa) Celiac disease
Rare	Primary immune defects Familial villous atrophy Secretory tumors Congenital chloride diarrhea Acrodermatitis enteropathica Lymphangiectasia Abetalipoproteinemia Eosinophilic gastroenteritis Short-bowel syndrome Autoimmune enteropathy/IPEX-like disorders Malabsorption syndromes Factitious	Acquired immune defects Secretory tumor Pseudoobstruction Factitious AIDS enteropathy Eosinophilic gastroenteritis	Secretory tumors Primary bowel tumor AIDS enteropathy Addison disease Parasitic infections

*Gastroenteritis includes viral (rotavirus, norovirus, astrovirus) and bacterial (Salmonella, Shigella, Escherichia coli, Clostridium difficile, Yersinia, Campylobacter, other) agents.

FPIES, Food protein–induced enterocolitis syndrome; IPEX, immune dysregulation, polyendocrinopathy, enteropathy, X-linked.

intestinal injury. Certain nonabsorbable laxatives, such as polyethylene glycol and milk of magnesia, also cause osmotic diarrhea. Fermentation of malabsorbed substances (e.g., lactose) often occurs, resulting in gas, cramps, and acidic stools. A common cause of chronic loose stools in early childhood is **functional diarrhea**, commonly known as **toddler's diarrhea**. This condition is defined by frequent watery stools in the setting of normal growth and weight gain and is caused by excessive intake of sweetened liquids overwhelming the disaccharide absorptive capacity of the intestine. Diarrhea typically improves tremendously when the child's beverage intake is reduced or changed.

Distinguishing Features

Stools are isosmotic, even in osmotic diarrhea, because of the relatively free exchange of water across the intestinal mucosa. Osmoles present in the stool are a mixture of electrolytes and other osmotically active solutes. To determine whether the diarrhea is osmotic or secretory, the **osmotic gap** is calculated:

$$\text{Osmotic gap} = 290 - 2([\text{Na}^+] + [\text{K}^+])$$

Assuming the stool is isosmotic (an osmolarity of 290 mOsm/L), the measured stool sodium and potassium (and their associated anions, accounting for an equal amount) accounts for much of the osmolality. Secretory diarrhea is characterized by an osmotic gap of less than 50. A number significantly higher than 50 defines osmotic diarrhea and indicates that malabsorbed substances other than electrolytes account for fecal osmolarity.

Another way to differentiate between osmotic and secretory diarrhea is to stop all feedings (in hospitalized patients receiving IV fluids). If the diarrhea stops completely while the child is NPO (nothing by mouth), the patient has osmotic diarrhea. A child with a pure secretory diarrhea would continue to have massive stool output. Neither method for classifying diarrhea works perfectly because most diarrheal illnesses are a mixture of secretory and osmotic components. Viral enteritis damages the intestinal lining, causing malabsorption and osmotic diarrhea. The associated inflammation results in release of mediators that cause excessive secretion. A child with viral enteritis may have decreased stool volume while NPO, but the secretory component of the diarrhea persists until the inflammation resolves.

The **history** should include the onset of diarrhea, number and character of stools, estimates of stool volume, and presence of other symptoms, such as blood in the stool, fever, and weight loss. Recent travel and exposures should be documented, dietary factors should be investigated, and a list of medications recently used should be obtained. Factors that seem to worsen or improve the diarrhea should be determined. **Physical examination** should be thorough, evaluating for abdominal distention, tenderness, quality of bowel sounds, presence of blood in the stool or a large fecal mass on rectal examination, and anal sphincter tone.

Laboratory testing should include stool culture and complete blood count if bacterial enteritis is suspected. If diarrhea occurs after a course of antibiotics, a *Clostridium difficile* toxin assay should be ordered; if stools are reported to be oily or fatty, fecal fat content or fecal elastase to test for pancreatic insufficiency should be measured. Tests for specific diagnoses should be sent when appropriate (e.g., serum antibody tests for celiac disease; colonoscopy for suspected ulcerative colitis). A trial of

lactose restriction for several days or lactose breath hydrogen testing is helpful in the evaluation of lactose intolerance.

Constipation and Encopresis

Constipation is a common problem in childhood. When a child is thought to be constipated, the parents may be concerned about straining with defecation, hard stool consistency, large stool size, decreased stool frequency, fear of passing stools, or any combination of these. Physicians define **constipation** as *two or fewer stools per week or passage of hard, pellet-like stools for at least 2 weeks*. A common pattern of constipation is **functional constipation**, which is characterized by two or fewer stools per week, voluntary withholding of stool, and infrequent passage of large-diameter, often painful stools. Children with functional fecal retention often exhibit "retentive posturing" (standing or sitting with legs extended and stiff or crossed legs) and have associated fecal incontinence caused by leakage of retained stool (**encopresis**).

Differential Diagnosis

The differential diagnosis is listed in [Table 126.10](#). **Functional constipation** commonly occurs during toilet training, when the child is unwilling to defecate on the toilet. Retained stool becomes harder and larger over time, leading to painful defecation. This aggravates voluntary withholding of stool, with perpetuation of the constipation.

Hirschsprung disease is characterized by delayed meconium passage in newborns, abdominal distention, vomiting, occasional fever, and foul-smelling stools. This condition is caused by failure of ganglion cells to migrate into the distal bowel, resulting in spasm and functional obstruction of the aganglionic segment. Only about 6% of infants with Hirschsprung disease pass meconium in the first 24 hours of life, compared with 95% of normal infants. Most affected babies rapidly become ill with symptoms of enterocolitis or obstruction. Affected older children do not pass large-caliber stools because of rectal spasm, and they do not have encopresis. Other causes of constipation include spinal cord abnormalities, hypothyroidism, medications, cystic fibrosis, intestinal pseudoobstruction syndromes, and anorectal malformations (see [Table 126.10](#)). A variety of systemic disorders affecting metabolism or muscle function can result in constipation. Children with **developmental disabilities** have a great propensity for constipation because of diminished capacity to cooperate with toileting, reduced effort or control of pelvic floor muscles during defecation, and diminished perception of the need to pass stool.

Distinguishing Features

[Table 126.10](#) includes typical characteristics of the common causes of constipation. Congenital malformations usually cause symptoms from birth. Functional constipation is overwhelmingly the most common diagnosis in older patients. Constipation in older children often begins after starting school, when free and private access to toilets may be restricted. Use of some drugs, especially opiates and some psychotropic medications, also is associated with constipation.

For a few specific causes of constipation, directed diagnostic testing can make the diagnosis. A narrowed, aganglionic distal bowel and dilated proximal bowel on barium enema suggests Hirschsprung disease. Rectal suction biopsy confirms

TABLE 126.10 Common Causes of Constipation and Characteristic Features

CAUSES OF CONSTIPATION	CLINICAL FEATURES
Hirschsprung disease	<i>History:</i> Failure to pass stool in first 24 hr, abdominal distention, vomiting, symptoms of enterocolitis (fever, foul-smelling diarrhea, megacolon). Not associated with large-caliber stools or encopresis <i>Examination:</i> Snug anal sphincter, empty, contracted rectum. May have explosive release of stool as examiner's finger is withdrawn <i>Laboratory:</i> Absence of ganglion cells on rectal suction biopsy specimen, absent relaxation of the internal sphincter, "transition zone" from narrow distal bowel to dilated proximal bowel on barium enema
Functional constipation	<i>History:</i> No history of significant neonatal constipation, onset at potty training, large-caliber stools, retentive posturing, may have encopresis <i>Examination:</i> Normal or reduced sphincter tone, dilated rectal vault, fecal impaction, soiled underwear, palpable fecal mass in left lower quadrant <i>Laboratory:</i> No abnormalities, barium enema would show dilated distal bowel
Anorectal, intestinal and colonic malformations/syndromes	<i>History:</i> Constipation from birth due to abnormal anatomy <i>Examination:</i> Anorectal abnormalities are shown easily on physical examination
Anal stenosis	Anteriorly displaced anus is found chiefly in females, with a normal-appearing anus located close to the posterior fourchette of the vagina
Anteriorly displaced anus	
Imperforate anus	<i>Laboratory:</i> Barium enema shows the anomaly
Colonic stricture	Genetic or idiopathic decreased or absent peristalsis
Intestinal pseudoobstruction	
Multisystem disease	<i>History:</i> Presence of other symptoms or prior diagnosis
Muscular dystrophy	<i>Examination:</i> Specific abnormalities may be present that directly relate to the underlying diagnosis
Cystic fibrosis	<i>Laboratory:</i> Tests directed at suspected disorder confirm the diagnosis
Diabetes mellitus	
Developmental delay	
Celiac disease	
Spinal cord abnormalities	<i>History:</i> History of swelling or exposed neural tissue in the lower back, history of urinary incontinence
Meningomyelocele	<i>Examination:</i> Lax sphincter tone due to impaired innervation, visible or palpable abnormality of lower back usually (but not always) present
Tethered cord	<i>Laboratory:</i> Bony abnormalities often present on plain x-ray. Magnetic resonance imaging (MRI) of spinal cord reveals characteristic abnormalities
Sacral teratoma or lipoma	
Drugs	<i>History:</i> Recent use of drugs known to cause constipation (lead poisoning, opiates)
Narcotics	<i>Examination:</i> Features suggest functional constipation
Psychotropics	<i>Laboratory:</i> No specific tests available

the absence of ganglion cells in the rectal submucosal plexus, with hypertrophy of nerve fibers. Lack of internal anal sphincter relaxation can be shown by anorectal manometry in Hirschsprung disease. Hypothyroidism is diagnosed by examination and by thyroid function testing. Anorectal malformations are easily detected by rectal examination. Cystic fibrosis (meconium ileus) is diagnosed by sweat chloride determination or *CFTR* gene mutation analysis (see Chapter 137). Most children with constipation have functional constipation and do not have any laboratory abnormality. Examination reveals normal or reduced anal sphincter tone (owing to stretching by passage of large stools). Fecal impaction is usually present, but a large-caliber, empty rectum may be found if a stool has just been passed.

Evaluation and Treatment of Functional Constipation

In most cases of constipation, the history is consistent with functional constipation—absence of neonatal constipation, active fecal retention, and infrequent, large stools with soiling. In these patients, no testing other than a good physical

examination is necessary. Young children with painful defecation must have a prolonged course of stool softener therapy to alleviate fear of defecation. The child should be asked to sit on the toilet for a few minutes on awakening in the morning and immediately after meals, when the colon is most active and it is easiest to pass a stool. Use of a positive reinforcement system for taking medication and sitting on the toilet is helpful for younger children. The stool softener chosen should be non-habit forming, safe, and palatable. Polyethylene glycol and milk of magnesia are the most commonly used agents.

Gastrointestinal Bleeding

GI tract bleeding can be an emergency when large-volume bleeding is present, but even the presence of small amounts of blood in stool or emesis is sufficient to cause concern. Evaluation of bleeding should include confirmation that blood truly is present, estimation of the amount of bleeding, stabilization of the patient's intravascular blood volume, localization of the source of bleeding, and appropriate treatment of the underlying cause. When bleeding is massive, it is crucial that

TABLE 126.11 Causes and Distinguishing Characteristics of Gastrointestinal Bleeding

AGE	TYPE OF BLEEDING	CHARACTERISTICS
NEWBORN		
Ingested maternal blood*	Hematemesis or rectal, large	Apt test indicates adult hemoglobin is present, cracked maternal nipples
Peptic disease	Hematemesis, amount varies	Blood found in stomach on lavage
Coagulopathy	Hematemesis or rectal, bruising, other sites	History of home birth (no vitamin K)
Allergic colitis*	Streaks of bloody mucus in stool	Eosinophils in feces and in rectal mucosa
Necrotizing enterocolitis	Rectal	Sick infant with tender and distended abdomen
Duplication cyst	Hematemesis	Cystic mass in abdomen on imaging study
Volvulus	Hematemesis, hematochezia	Acute tender, distended abdomen
INFANCY TO 2 YEARS OLD		
Peptic disease	Usually hematemesis, rectal possible	Epigastric pain, coffee-ground emesis
Esophageal varices	Hematemesis	History or evidence of liver disease
Intussusception*	Rectal bleeding	Crampy pain, distention, mass
Meckel diverticulum*	Rectal	Massive, bright red or maroon blood; no pain
Bacterial enteritis*	Rectal	Bloody diarrhea, fever
NSAID injury	Usually hematemesis, rectal possible	Epigastric pain, coffee-ground emesis
>2 YEARS OLD		
Peptic disease	See above	See above
Esophageal varices	See above	See above
NSAID injury*	See above	See above
Inflammatory bowel disease	Usually rectal	Crampy pain, poor weight gain, diarrhea
Bacterial enteritis*	See above	See above
Pseudomembranous colitis	Rectal	History of antibiotic use, bloody diarrhea
Juvenile polyp	Rectal	Painless, bright red blood in stool; not massive
Meckel diverticulum*	See above	See above
Nodular lymphoid hyperplasia	Rectal	Streaks of blood in stool, no other symptoms
Angiodysplasia	Rectal	Difficult to diagnosis, often requires capsule endoscopy
Mallory-Weiss syndrome*	Hematemesis	Bright red or coffee-ground, follows retching
Hemolytic uremic syndrome	Rectal	Thrombocytopenia, anemia, uremia
Henoch-Schönlein purpura	Rectal	Purpuric rash, nephritis, arthritis, colitis
Hemorrhoids	Rectal	Dilated external veins, blood with wiping

*Common.

NSAID, Nonsteroidal antiinflammatory drug.

the patient receive adequate resuscitation with fluid and blood products before moving ahead with diagnostic testing.

Differential Diagnosis

GI bleeding has different causes at different ages (Table 126.11).

Distinguishing Features

Red substances in foods, beverages, or medications (such as cefdinir) occasionally can be mistaken for blood. A test for occult blood is worth performing whenever the diagnosis is in doubt.

The GI tract may not be the source of the observed fecal blood. A history of cough and examination of the mouth, nostrils, and lungs is needed to exclude these as a source of hematemesis. Blood in the toilet or diaper may be coming from the urinary tract, vagina, or even a severe diaper rash. If the bleeding is GI, it is important to determine the source as the upper GI tract or distal to the ligament of Treitz. Vomited blood is always proximal. Rectal bleeding may be coming from anywhere in the gut. When dark clots or melena are seen mixed with stool, a higher location is suspected, whereas bright red

TABLE 126.12 Evaluation of Gastrointestinal Bleeding

LABORATORY INVESTIGATION	
All Patients	
CBC and platelet count	
Coagulation tests: prothrombin time, partial thromboplastin time	
Tests of liver dysfunction: AST, ALT, GGT, bilirubin	
Occult blood test of stool or vomitus	
Blood type and crossmatch	
Evaluation of Bloody Diarrhea	
Stool culture or PCR, <i>Clostridium difficile</i> toxin	
Sigmoidoscopy or colonoscopy	
CT or MRI enteroscopy	
Evaluation of Rectal Bleeding with Formed Stools	
External and digital rectal examination	
Sigmoidoscopy or colonoscopy	
Meckel scan	
Mesenteric arteriogram	
Video capsule endoscopy	
INITIAL RADIOLOGIC EVALUATION	
All Patients	
Abdominal x-ray series	
Evaluation of Hematemesis	
Barium upper GI series if endoscopy not available	
Evaluation of Bleeding with Pain and Vomiting (Bowel Obstruction)	
Abdominal x-ray series	
Pneumatic or contrast enema	
Upper GI series	

ALT, Alanine aminotransferase; AST, aspartate aminotransferase; CBC, complete blood count; CT, computed tomography; GGT, γ -glutamyltransferase; GI, gastrointestinal.

blood on the surface of stool probably is coming from lower in the colon. When upper GI tract bleeding is suspected, a nasogastric tube may be placed and gastric contents aspirated for evidence of recent bleeding.

The location and hemodynamic significance of the bleeding can also be assessed by history and examination. The parents should be asked to quantify the bleeding. Details of associated symptoms should be sought. Assessment of the vital signs including orthostatic changes when bleeding volume is large, pulses, capillary refill, and assessment of pallor of the mucous membranes provides valuable information. Laboratory assessment and imaging studies should be ordered as indicated (Table 126.12).

Treatment

Treatment of GI bleeding should begin with an initial assessment, rapid stabilization, and a logical sequence of diagnostic tests. When a treatable cause is identified, specific therapy should be started. In many cases, the amount of blood is small, and no resuscitation is required. For children with large-volume bleeds, the ABCs of resuscitation (airway, breathing, circulation) should be addressed first (see Chapters 38 and 40). Oxygen should be administered, and the airway should be protected with an endotracheal tube if massive hematemesis is present. Fluid boluses and transfusion with packed RBCs as required should be administered through two large-bore IVs. Frequent reassessment should continue to ensure maintenance of physiologic stability (Fig. 126.2).

PEARLS FOR PRACTITIONERS

See *The Digestive System: Pearls for Practitioners* at the end of this section.

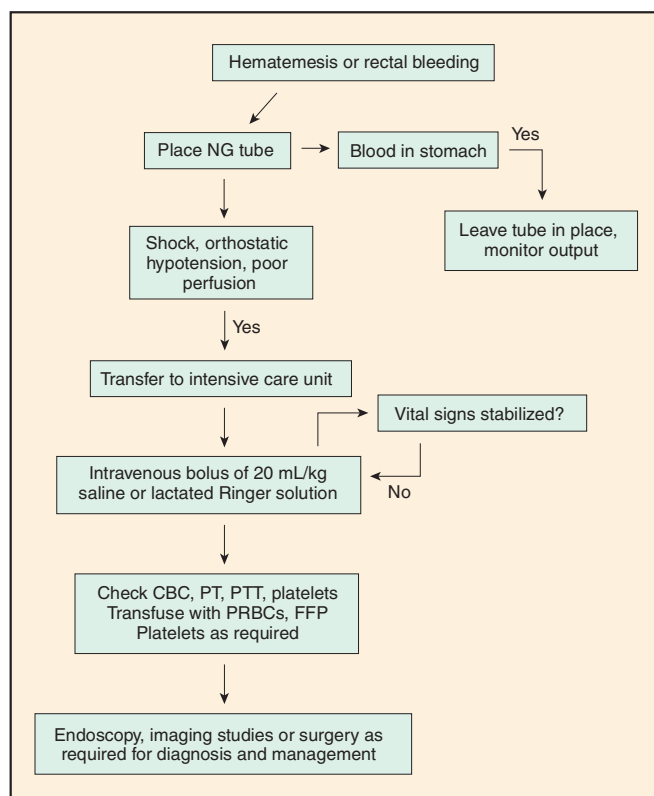


FIGURE 126.2 Initial management of gastrointestinal bleeding. CBC, Complete blood count; FFP, fresh frozen plasma; NG, nasogastric; PRBCs, packed red blood cells; PT, prothrombin time; PTT, partial thromboplastin time.