Gastrointestinal disorders affecting the pediatric population vary significantly with the age of the child. Consideration of age as well as of signs and symptoms has an important role in distinguishing cause. Infants, for example, may have disorders such as necrotizing enterocolitis (NEC), hypertrophic pyloric stenosis, or intussusception, whereas older children are more likely to present with appendicitis, pancreatitis, or biliary tract disease.

Pediatric gastrointestinal disorders can be divided into different groups on the basis of their unique pathophysiologic mechanisms and reasons for development. Several disorders occur as a normal variant of development (neonatal jaundice, gastroesophageal reflux, hypertrophic pyloric stenosis), congenital malformations (malrotation, Meckel’s diverticulum), or genetic abnormalities (Hirschsprung’s disease). Idiopathic causes include NEC, intussusception, Henoch-Schönlein purpura (HSP), and inflammatory bowel disease (IBD).

Neonatal Jaundice

Perspective

Many infants become jaundiced during the newborn period, most often as a result of a benign, self-limited process. Physiologic jaundice of the newborn, the most common cause of neonatal jaundice, occurs in approximately 60% of normal newborns during the first week of life. Breast milk jaundice is the second most common cause of jaundice in the newborn period.

Principles of Disease

Bilirubin is formed by the breakdown of heme-containing proteins, primarily hemoglobin. Heme protoporphyrin is sequentially degraded into biliverdin and unconjugated bilirubin. Unconjugated bilirubin binds to albumin in the blood and is carried to the liver, where it is conjugated by glucuronol transferase and excreted into bile. Jaundice may be caused by increased amounts of either unconjugated or conjugated bilirubin and becomes clinically apparent when the total bilirubin level reaches approximately 5 mg/dL. Conjugated hyperbilirubinemia occurs when the direct-reacting portion exceeds 2 mg/dL or is greater than 20% of the total.

Physiologic jaundice of the newborn is the most common cause of neonatal jaundice and occurs in normal newborns during the first week of life. Affected infants are born with normal bilirubin levels that gradually increase to a peak level of 6 mg/dL on the third day of life and then decline to normal within 2 weeks. Breast milk jaundice is the next most common cause of hyperbilirubinemia in newborns. Affected infants exhibit the same gradual increase seen with physiologic jaundice. Levels continue to increase, however, and reach a higher peak level at 10 days to 3 weeks of life. Elevated levels may persist for 3 to 10 weeks and then gradually subside.

Toxic levels of bilirubin (more than 20 mg/dL and dependent on age) may be associated with neurotoxicity, encephalopathy, and the development of kernicterus. Kernicterus is characterized by yellow staining in areas of the brain, including the basal ganglia. Clinical manifestations begin with poor feeding and lethargy and may progress to muscle rigidity, opisthotonos, seizures, and death. Survivors may have residual problems with coordination and hearing and learning disabilities. The cornerstones of therapy are phototherapy and exchange transfusion.

Diagnostic Strategies

Although physiologic jaundice of the newborn and breast milk jaundice are most common, it is important to identify truly pathologic causes of jaundice. Initial testing requires determination of fractionated levels of total and direct bilirubin. Box 172-1 lists indications for workup in infants presenting with hyperbilirubinemia. Conjugated (direct) hyperbilirubinemia is always pathologic. In such cases, a minimal workup should include a complete blood count (CBC) with peripheral smear and a Coombs test for immune-mediated major blood group incompatibility. Ill-appearing infants also require finger-stick blood glucose measurement, electrolyte panel, urine assay for reducing substances, and
Indications for Workup in Jaundiced Infants

Jaundice appearing within 24 hours of birth
Elevated direct (conjugated) bilirubin level
Rapidly rising total serum bilirubin unexplained by history or physical examination
Total serum bilirubin approaching exchange level or not responding to phototherapy
Jaundice persisting beyond 3 weeks of age
Sick-appearing infant

Differential Considerations

Birth history should be obtained to elicit any history of trauma because large, resolving hematomas can result in jaundice. Family history should focus on identification of siblings or other relatives with a history of jaundice or genetic or metabolic disorders and any unexplained infant deaths. Tables 172-1 and 172-2 present full lists of differential considerations for jaundiced infants and children, respectively.

Infants with direct hyperbilirubinemia represent a special subset of patients. All infants with direct hyperbilirubinemia require hospital admission and evaluation for the cause of the jaundice based on history and presenting signs and symptoms. This evaluation may include any or all of the following: sepsis workup, TORCHS (toxoplasmosis, other infections, rubella, cytomegalovirus, herpes, syphilis) titers, basic metabolic studies, α1-antitrypsin, sweat test for cystic fibrosis, ultrasound studies, radioisotope (hepatobiliary iminodiacetic acid [HIDA] or diisopropyl iminodiacetic acid [DISIDA] scan, and liver biopsy. In children, hemolytic anemia, infection, and drugs are the most common causes of jaundice. The history should focus on travel, exposures, medications, and associated signs and symptoms, such as fever, malaise, and weight loss. Gentle palpation of the liver is
useful to estimate size, firmness, and tenderness and to distinguish hepatomegaly from liver inflammation.

Management

Treatment of infants with hyperbilirubinemia centers on the prevention of kernicterus. Guidelines for use of phototherapy and exchange transfusion have been recommended by the American Academy of Pediatrics (Fig. 172-1). Feeding should be continued to the extent possible because oral intake stimulates enterohepatic circulation and decreases bilirubin levels. Unless the infant is severely jaundiced, breast-feeding can be continued and supplemented with formula as needed. Infants who are premature or who have significant comorbidities require treatment at lower levels. Phototherapy often is now readily available on an outpatient basis.

Disposition

In general, infants with bilirubin levels greater than 18 to 20 mg/dL require hospital admission and phototherapy. All infants with direct hyperbilirubinemia require admission and workup.

Hypertrophic Pyloric Stenosis

Perspective

Hypertrophic pyloric stenosis is the most common cause of infantile gastrointestinal obstruction beyond the first month of life. This condition occurs in 1 of every 250 live births. Boys are affected at four times the rate of girls. Hypertrophic pyloric stenosis tends to run in families; however, the exact pattern of inheritance is unclear. The incidence rate is 1 in 14 if the father is affected; the rate is even higher if the mother is affected. Whites are affected more often than African Americans, and the disease is rare in Asian Americans.

Principles of Disease

Infants are born with a normal pylorus that undergoes hypertrophy only as time progresses. The exact etiology is unknown. As the pylorus continues to hypertrophy, there is progressive gastric outlet obstruction. As vomiting continues, the infant loses hydrogen and chloride ions through emesis of gastric juices. As this metabolic derangement worsens, the kidney attempts to retain...
hydrogen ions in exchange for potassium, resulting in a hypochloremic, hypokalemic metabolic alkalosis.

Clinical Features

Infants classically present at 2 to 6 weeks old with gradually progressive emesis that becomes projectile and remains nonbilious. Infants remain vigorous with a ravenous appetite. They rapidly finish an entire feeding, only to regurgitate the entire volume in a projectile fashion. In the later stages of the disease, children may exhibit visible waves of abdominal peristalsis in response to intense contractions against an obstruction. Children in the later stages of disease may exhibit marasmus (protein-calorie malnutrition) as a result of impaired nutrient absorption.

Diagnostic Strategies

Children may have a palpable pylorus, commonly referred to as an olive in the right epigastrium, on abdominal examination. Placement of a nasogastric tube and emptying the stomach or placement of the infant in the prone position often facilitates palpation. Hypertrophic pyloric stenosis may be confirmed by ultrasonography or upper gastrointestinal series. Ultrasonography is the diagnostic modality of choice because it is simple, readily available, and without serious complications such as aspiration. With both modalities, reported accuracy is greater than 95%. Ultrasound examination reveals a thickened pylorus, which is diagnostic. A characteristic “string sign,” reflecting passage of contrast material through the narrowed pyloric sphincter, can be seen in the upper gastrointestinal tract. In advanced stages with complete obstruction at the pylorus, plain films may reveal an enlarged body of the stomach and pylorus (Fig. 172-2).

Differential Considerations

Etiologic possibilities vary by whether the course of vomiting has been sudden in onset, gradually progressive, or chronic. The frequency and volume of emesis may have important implications about the severity of disease and the potential risk for dehydration or electrolyte disturbance. In infants, other major considerations in the differential diagnosis include gastroesophageal reflux and malrotation. The age of the child and the timing of vomiting provide important clues to the etiology. Reflux classically begins early in life, usually shortly after birth, and remains relatively constant. With pyloric stenosis, vomiting does not begin until 2 to 3 weeks of age and then becomes increasing severe and projectile; the emesis is rarely if ever bilious. In neonates, bilious vomiting requires diagnostic evaluation to rule out the possibility of malrotation with volvulus. The imaging modality of choice for pyloric stenosis is ultrasonography.

Many causes of vomiting do not have a true gastrointestinal origin, including sepsis, increased intracranial pressure, middle ear disturbances, urinary tract infections, inborn errors of metabolism, pain, medications, and drug intoxications. Differential considerations for vomiting in children vary by age (Table 172-3).
Management

Treatment consists of fluid and electrolyte replacement and surgical consultation. Fluid resuscitation should begin with a bolus of 20 mL/kg of normal saline, followed by additional boluses as necessary to treat signs of shock. When the patient is stable and shows no signs of shock, 5% dextrose and half-normal saline at 1.5 to 2 times maintenance may be administered. Potassium supplementation is often necessary. Hypertrophic pyloric stenosis is a chronic, progressive disease, not an acute ischemic process. Confirmatory radiographic diagnosis with ultrasonography may be done on a semiurgent basis as the patient's condition warrants. Pediatric surgical consultation is recommended although usually not emergent. The definitive corrective surgical procedure is the Ramstedt pyloromyotomy, which has an excellent track record of safety. More recently, laparoscopic pyloromyotomy has gained increasing acceptance as being safe and effective. Associated mortality is rare.

Disposition

Most children probably are best managed with hospital admission for rehydration and correction of electrolyte abnormalities. Imaging and surgical consultation are obtained on a semi-elective basis.

Table 172-3 Differential Considerations for Vomiting by Age

<table>
<thead>
<tr>
<th>ETIOLOGIC CATEGORY</th>
<th>INFANCY</th>
<th>CHILDHOOD</th>
<th>ADOLESCENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mechanical</td>
<td>Gastroesophageal reflux</td>
<td>Constipation</td>
<td>Constipation</td>
</tr>
<tr>
<td>Malrotation with midgut volvulus</td>
<td>Malrotation with midgut volvulus</td>
<td>Incarcerated hernia</td>
<td>Incarcerated hernia</td>
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<tr>
<td>Pyloric stenosis</td>
<td>Pyloric stenosis</td>
<td>Meckel's diverticulum</td>
<td>Meckel's diverticulum</td>
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<tr>
<td>Intussusception</td>
<td>Intussusception</td>
<td>Bowel obstruction</td>
<td>Bowel obstruction</td>
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<tr>
<td>Bowel obstruction</td>
<td>Bowel obstruction</td>
<td>Incarcerated hernia</td>
<td>Incarcerated hernia</td>
</tr>
<tr>
<td>Inflammatory or infectious</td>
<td>Tracheoesophageal fistula</td>
<td>Necrotizing enterocolitis</td>
<td>Necrotizing enterocolitis</td>
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<tr>
<td>Gastroenteritis</td>
<td>Gastroenteritis</td>
<td>Otitis media</td>
<td>Appendicitis</td>
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<td>Sepsis</td>
<td>Sepsis</td>
<td>Appendicitis</td>
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<td>Henoch-Schönlein purpura</td>
<td>Pancreatitis</td>
<td>Gastritis</td>
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<td>Meningitis</td>
<td>Meningitis</td>
<td>Henoch-Schönlein purpura</td>
<td>Biliary tract disease</td>
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<td>Pneumonia</td>
<td>Biliary tract disease</td>
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</tr>
<tr>
<td>Otitis media</td>
<td>Otitis media</td>
<td>Gastritis or gastroenteritis</td>
<td>Gastritis or gastroenteritis</td>
</tr>
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<td>Urinary tract infection</td>
<td>Urinary tract infection</td>
<td>Urinary tract infection</td>
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<td>Hydrocephalus</td>
<td>Hydrocephalus</td>
<td>Migraine headache</td>
<td>Migraine headache</td>
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<tr>
<td>Intracranial hemorrhage</td>
<td>Intracranial hemorrhage</td>
<td>Hydrocephalus</td>
<td>Hydrocephalus</td>
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<tr>
<td>Intracranial tumor</td>
<td>Intracranial tumor</td>
<td>Intracranial hemorrhage</td>
<td>Intracranial hemorrhage</td>
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<tr>
<td>Metabolic</td>
<td>Diabetic ketoacidosis</td>
<td>Diabetic ketoacidosis</td>
<td>Diabetic ketoacidosis</td>
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<tr>
<td>Congenital adrenal hyperplasia</td>
<td>Congenital adrenal hyperplasia</td>
<td>Urea cycle defects</td>
<td>Urea cycle defects</td>
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<td>Urea cycle defects</td>
<td>Urea cycle defects</td>
<td>Fatty acid oxidation disorders</td>
<td>Fatty acid oxidation disorders</td>
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<td>Organic acidurias</td>
<td>Organic acidurias</td>
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<tr>
<td>Amino acidopathies</td>
<td>Amino acidopathies</td>
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<tr>
<td>Fatty acid oxidation disorders</td>
<td>Fatty acid oxidation disorders</td>
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<td></td>
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<tr>
<td>Other or atypical</td>
<td>Occult trauma (abuse)</td>
<td>Sickle cell</td>
<td>Sickle cell</td>
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<tr>
<td>Toxic ingestions</td>
<td>Toxic ingestions</td>
<td>Toxic ingestions</td>
<td>Toxic ingestions</td>
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<tr>
<td>Munchausen syndrome by proxy</td>
<td>Munchausen syndrome by proxy</td>
<td>Occult trauma (abuse)</td>
<td>Occult trauma (abuse)</td>
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<tr>
<td></td>
<td></td>
<td>Munchausen syndrome by proxy</td>
<td>Munchausen syndrome by proxy</td>
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</tbody>
</table>
Malrotation with Midgut Volvulus

Perspective

Malrotation occurs in 1 in 500 live births and has a male predominance by at least 2:1. Among infants with malrotation, volvulus eventually will develop in approximately 75%, and 75% of these infants present within the first month of life. Overall, 90% of patients present within the first year of life, although cases of adult midgut volvulus have been reported. Bilious emesis is the hallmark presentation and is seen in more than 75% of cases. Malrotation with volvulus carries a mortality rate of 3 to 15%.

Principles of Disease

During embryologic development, the gastrointestinal tract rotates around the superior mesenteric artery. As it completes the rotation, the duodenum forms a C-loop and is fixed to the retroperitoneum in the left upper quadrant at the ligament of Treitz. The cecum becomes similarly fixed in the right lower quadrant. The duodenum and cecum normally come to lie widely separated and loosely connected by a broad-based mesentery. They are fixed firmly in position by peritoneal attachments called Ladd’s bands. In cases of malrotation, the duodenum and the cecum do not rotate completely but end up close to each other, suspended in the midgut region by their vascular attachment containing the superior mesenteric artery. This unusually close proximity of the intervening mesentery results in a short stalk of mesentery that easily twists on itself, resulting in obstruction of the distal duodenum and compression of the superior mesenteric artery. Vascular compression results in ischemia of the bowel and, if it is not rapidly reversed, necrosis of the bowel wall in 1 to 2 hours. Twisting of the pedicle also results in various degrees of obstruction secondary to Ladd’s bands that are malpositioned and straddling the duodenum.

Bilious emesis is the hallmark presentation and is a result of severe obstruction. Any pigmented staining of the vomitus suggests the presence of bile. When bile is initially produced, it is bright yellow and turns green only with time and oxidative exposures. Differential coloring of bile-stained emesis, yellow versus green, is not predictive of surgical condition.

Clinical Features

Infants classically present with sudden-onset bilious emesis and abdominal distention. The obstruction may be relatively high, however; consequently, a distended abdomen may not always be present. Infants usually appear quite ill and may present in shock. Infants also may present with a history of intermittent, relatively mild episodes of emesis that suddenly become more intense. Although bilious emesis in a neonate suggests the possibility of acute obstruction and volvulus, presenting manifestations in children may be nonspecific, such as abdominal distention or an ill appearance.

Diagnostic Strategies

Diagnostic strategies may include a plain film of the abdomen, an upper gastrointestinal series, or a computed tomography (CT) scan of the abdomen. The imaging modality of choice is an upper gastrointestinal series with small bowel follow-through. Findings on plain abdominal films may include air-fluid levels suggesting obstruction, dilated loops overlying the liver, and a paucity of small bowel gas distally. In addition, a “double-bubble sign” may be present. The classic double bubble represents a dilated stomach and obstructed proximal duodenum and is seen with duodenal atresia and malrotation with midgut volvulus. Duodenal atresia is limited to the newborn nursery and is manifested within 24 hours of life. Malrotation with midgut volvulus typically is manifested with bilious vomiting within the first month of life. The modified double-bubble sign seen with hypertrophic pyloric stenosis represents a dilated body of the stomach and pylorus and is associated with nonbilious emesis.

The diagnostic procedure of choice to determine midgut volvulus is the upper gastrointestinal series, which reveals abnormal position of the duodenal C-loop and small bowel with a characteristic corkscrew appearance. It is very low
Vomiting in childhood is common and occurs across a wide spectrum of illnesses (see Table 172-3). Causes vary by the age of the child and whether the course of vomiting has been sudden in onset, gradually progressive, or chronic. Gastroesophageal reflux classically begins early in life, usually shortly after birth, and remains relatively constant. With pyloric stenosis, vomiting does not begin until the age of 2 to 3 weeks and then becomes increasingly severe, projectile, and nonbilious. Acute obstruction causes sudden-onset vomiting, which may be bilious. In a neonate, bilious vomiting requires diagnostic evaluation to rule out the possibility of malrotation with volvulus. NEC is another consideration but most often is manifested within the first weeks of life. Neonates with NEC may present with obstructive signs and symptoms, including bilious emesis and abdominal distention. Whereas malrotation usually is associated with a paucity of small bowel air on plain films, NEC is characterized radiographically by diffusely dilated loops of small bowel. In addition, the presence of pneumatosis intestinalis is diagnostic of NEC but is not a feature of malrotation.

Management

Infants with bilious emesis of sudden onset who appear ill or have a distended abdomen require emergency consultation with a pediatric surgeon. Intravenous access should be obtained and laboratory studies performed for CBC, electrolyte values, and liver function. Repeated fluid boluses of normal saline, 20 mL/kg, are necessary until adequate circulation has been obtained. A finger-stick sample for blood glucose determination and specimens for blood and urine cultures should be obtained. A nasogastric or orogastric tube should be placed. After consultation with a pediatric surgeon, an upper gastrointestinal series may be needed emergently. Ill-appearing infants require broad-spectrum, triple-antibiotic coverage with ampicillin, gentamicin, and either clindamycin or metronidazole. Time is of the essence in the evaluation and operative management of these patients. Rapid pediatric surgical consultation should be obtained in any neonate or infant with bilious vomiting even before diagnostic studies have been performed, especially for infants who appear ill. In contrast to hypertrophic pyloric stenosis, in which surgery does not need to be immediate, operative intervention must be rapid to save the bowel from necrosis.

Disposition

Patients thought to have malrotation require definitive imaging and an immediate surgical evaluation. If the diagnosis is confirmed or equivocal, hospital admission and surgical management are required.

Necrotizing Enterocolitis

Perspective

NEC, the most common gastrointestinal emergency in neonates, affects 2000 to 4000 infants in the United States every year. NEC also is the most common cause of intestinal perforation during the newborn period. Because most affected infants are premature and acquire the condition in the neonatal intensive care unit, NEC usually is not considered a disease of the emergency department (ED). Many of these infants may be discharged relatively early, however, because they are “feeding and growing” and come to treatment in the first month of life. Ten percent of infants with NEC are full-term babies. Development of NEC is related closely to gestational age. In infants born at 24 to 28 weeks of gestation, NEC develops within 2 to 3 weeks of life; in those born at 29 to 32 weeks of gestation, it is seen within 1 to 3 weeks of life. Among more mature or full-term infants, NEC tends to develop in the first week of life. Complications in children who survive NEC may include strictures (in 10-20% of cases), fistulas, and short gut syndrome.

Principles of Disease

The exact pathophysiologic mechanism of NEC is unclear but seems to be multifactorial. Proposed risk factors include prematurity, aggressive enteral feedings, birth-related hypoxic-ischemic insults, and infectious causes. Prematurity is the most common and universally accepted risk factor; 90% of all affected infants are premature at birth. Rapid advancement of feedings also has been associated with increased rates of NEC. Infection has been implicated as an important causative mechanism. Evidence suggests that hypoxic-ischemic insults are not an independent risk factor for the development of NEC. The primary pathologic event may be inflammation or injury to the intestinal wall, which begins in the mucosa and then extends transmurally. The distal ileum and proximal colon are more commonly affected, and the involvement may be continuous or patchy.

Clinical Features

Infants with NEC present with feeding intolerance and emesis. Emesis may be either nonbilious or bilious. On occasion, individual loops of bowel become distended with air and are palpable on abdominal examination. In the more severe stages of the disease, infants may appear extremely ill, with hematemesis, hematochezia, and shock. NEC commonly is placed into one of the following three categories:

1. **Type A** (simple enterocolitis): mild clinical course, no perforation
2. **Type B** (mild perforation): few signs of peritonitis, perforation
3. **Type C** (severe perforation): peritonitis, systemic signs
# Gastrointestinal Disorders

## Chapter 172

### Diagnostic Strategies

Dilated loops of bowel are a common but nonspecific finding in stage I. Another early and more specific radiographic sign is the loss of a normal symmetrical gas pattern and replacement with an asymmetrical pattern of bowel gas, with a variable degree of dilation. Intramural air (pneumatosis intestinalis) is specific for NEC and is present in stage II (Fig. 172-6). Pneumatosis is present in 75% of patients with NEC. Air also may be seen within the biliary tract (portal vein gas) or occasionally in the gastric wall (pneumatosis gastralis; see Fig. 172-6). Portal vein gas is present in 10 to 30% of cases. Ultrasonography and barium enema, which have been described as adjunctive diagnostic imaging modalities in patients with suspected NEC, are rarely helpful in the ED. No individual laboratory features are diagnostic or specific for NEC.

### Disposition

Children thought to have NEC require admission to an intensive care unit (either pediatric or neonatal) and pediatric surgical consultation.

### Gastroesophageal Reflux

#### Perspective

Gastroesophageal reflux, one of the most common causes of vomiting during infancy, refers to the regurgitation of stomach contents into the esophagus.

#### Principles of Disease

Gastroesophageal reflux occurs as a result of an incompetent lower esophageal sphincter. Chronic reflux of gastric contents into the esophagus may result in esophagitis, aspiration, and failure to thrive if it is severe.

#### Clinical Features

Clinical manifestations occur along a wide spectrum of disease, ranging from occasional episodes of spitting up to severe, persistent vomiting and failure to thrive. Gastroesophageal reflux generally responds to conservative measures and resolves with age. It
usually begins shortly after birth and resolves by the age of 1 year. The disorder may be associated with stereotypical opisthotonic movements, collectively referred to as Sandifer’s syndrome. Affected children exhibit extension and stiffening of the arms and legs and extension of the neck, often accompanied by a shrill or guttural cry. It may be associated with a brief period of apnea and pallor as formula is refluxed into the esophagus. Sandifer’s syndrome most commonly occurs shortly after feeding and usually is not associated with cyanosis.

Diagnostic Strategies

In the ED, the diagnosis of gastroesophageal reflux typically is made on the basis of a careful history and physical examination. However, for ill patients in whom the diagnosis is uncertain, other diagnostic studies are available with gastroenterology consultation, such as esophageal pH probes to check for reflux of acid, barium swallow, and direct visualization by endoscopy.

Differential Considerations

Children with gastroesophageal reflux exhibit nonbilious emesis that begins shortly after birth and is relatively constant over time. Typically, no sudden starting or ending point, as would be suggested by an acute obstruction, can be identified. Vomiting usually is neither gradually progressive nor projectile, as seen with pyloric stenosis. Most children with gastroesophageal reflux of milder severity continue to gain weight.

Management

Most infants respond to conservative measures, such as smaller feedings, frequent “burpings,” thickening of formula with cereal, and semiupright position for 45 minutes to 1 hour after feeding. Pharmacologic regimens are reserved for more severe cases. Weight loss is an important historical feature and necessitates pediatric gastroenterology referral for evaluation. Children exhibiting failure to thrive or esophagitis often are prescribed ranitidine and metoclopramide. Ranitidine, a histamine blocker, reduces gastric acid secretion. Metoclopramide increases lower esophageal tone, reduces pyloric sphincter tone, and increases gastric motility. Patients not responding to medical management occasionally require surgical intervention with a Nissen fundoplication, which involves wrapping of a portion of the stomach around the esophagus to prevent the reflux.

Disposition

Most children can be discharged home safely with conservative measures. Children who exhibit failure to thrive should be referred to a pediatrician or pediatric gastroenterologist for consideration of pharmacologic management. Children with dehydration and children in whom other diagnoses have yet to be excluded may benefit from hospital admission and further evaluation.

Intussusception

Perspective

Intussusception is the most common cause of intestinal obstruction in children younger than 2 years and occurs most commonly in infants 5 to 12 months of age. An estimated incidence of 1 per 2000 children younger than 15 years, with a male predominance, has been reported. Siblings of affected children have a relative risk 15 to 20 times higher than that in the general population. The mortality rate for untreated intussusception is high.

Principles of Disease

The exact etiology of intussusception is unclear, but the most prevalent theory relates to a lead point that causes telescoping of one segment of intestine into another. As the process continues and intensifies, edema develops and obstructs venous return, resulting in ischemia of the bowel wall. As ischemia of the bowel wall continues, peritoneal irritation ensues, and perforation may occur.

In younger children, lead points are most often the result of enlarged Peyer’s patches secondary to a recent viral infection. In children older than 5 years, an underlying lesion is found in more than 75% of cases; lesions include HSP vasculitis, Meckel’s diverticulum, lymphoma, polyps, postsurgical scars, celiac disease, and cystic fibrosis. Intussusception may occur at any point along the gastrointestinal tract. Ileocolic intussusceptions are most common. Ileoileal intussusception may occur in children with HSP.

Clinical Features

The classic triad of clinical findings in intussusception consists of abdominal pain, vomiting, and bloody stools. All three features are present in less than one third of patients, however. Three quarters of patients with intussusception have two of these findings, and 13% have either none or only one. In a typical presentation, the child experiences cyclic episodes of severe abdominal pain. The pain typically lasts 10 to 15 minutes and has a periodicity of 15 to 30 minutes. During the painful episodes, the child is inconsolable, often described as drawing the legs up to the abdomen and screaming in pain. The clinical presentation occasionally does not include more typical evidence of pain; instead, the child presents with profound lethargy. Vomiting and diarrhea may be associated features. Blood may be present in either the stool or the emesis. Diarrhea containing mucus and blood constitutes the classic “currant jelly” stool most often associated with intussusception, although in actuality this finding is relatively infrequent. Children often have had a recent viral illness. Palpation of the abdomen may reveal a sausage-like mass in the right upper quadrant representing the actual intussusceptum and an empty space in the right lower quadrant representing the movement of the cecum out of its normal position. This combination of findings is called Dance’s sign and is considered pathognomonic for intussusception. Its occurrence is relatively uncommon, however. Intussusception usually is not associated with a high fever; however, low-grade fevers may occur.

Diagnostic Strategies

Initial screening films should be obtained, with a minimum of two views of the abdomen. Attention should be focused on visualization of the entire colon and in particular the cecum. Films also should be examined for evidence of a soft tissue mass or mass effect, obstruction, target sign (representing air in the intussusceptum as it telescopes into adjacent bowel), meniscus sign (representing air compressed like a meniscus from invaginating bowel), and free air (Fig. 172-7). Normal findings on radiographs of the abdomen revealing complete visualization of the entire colon, including the cecum, make intussusception unlikely; however, indeterminate or nonspecific findings on films in which the entire colon cannot be visualized do not rule out intussusception and require additional imaging. Ultrasonography is the least invasive and most commonly used modality to visualize intussusception and is highly accurate. Ileocolic intussusceptions are most common and are easily detected by ultrasonography, even in inexperienced hands. The goal of the ultrasound examination is to visualize the ileocecal junction, which should be in the
and forth, frequently moaning or crying. Children with ischemic pain exhibit symptoms out of proportion to the findings on examination. They may appear diaphoretic, clammy, or pale and complain of excruciating abdominal pain although exhibiting only mild tenderness to palpation and no localizing findings.

Differential Considerations

Differential considerations for abdominal pain in children by age are listed in Table 172-4. Slow, progressive onset of pain is more likely to be associated with appendicitis, constipation, or pancreatitis. Children with peritoneal irritation invariably lie still, often on the side with the knees bent, and refrain from all extraneous movement. Sudden onset of severe pain is associated most often with acute obstruction or vascular occlusion, as seen with intussusception, volvulus, or torsion of the testicle or ovary. Children with intussusception have severe colicky pain and often rock back right lower quadrant but may be translocated into the right middle to upper quadrant. On the ultrasound scan, the intussusception appears on the transverse view as a multilayered or wrapped complex mass and on the longitudinal view as a tube within a tube as the ileum projects up into the cecum (Fig. 172-8). Because of the readily identifiable anatomic location and the absence of ionizing radiation, this is usually the diagnostic imaging modality of choice. Contrast enemas may be both diagnostic and therapeutic and, with a high index of suspicion (i.e., children with the triad of paroxysms of pain, vomiting, and blood in stool), may be done as first-line therapy. The enema will reveal a sharp cutoff at the point where the intussusceptum meets the contrast material (Fig. 172-9). Air-contrast enemas are equally efficacious, have success rates averaging more than 60%, and are preferred to barium contrast studies by some physicians.28-31 Either type of enema requires readily available backup by a pediatric surgeon in the event of failure of the bowel to reduce or perforation. Before any radiologic studies are performed, intravenous access should be established, and the patient should receive at least one bolus of 20 mL/kg of normal saline along with appropriate parenteral pain relief.
Hirschsprung's disease occurs at a rate of 1 in 5000 live births and is four to five times more common in boys. It usually is sporadic in occurrence but may be associated with Down syndrome or a variety of other anomalies of the gastrointestinal, genitourinary, or central nervous system.32

## Disposition

Children with suspected intussusception require definitive imaging with ultrasonography. Children with documented intussusception require reduction with either enema or surgery. Hospital admission is recommended for all patients after reduction.

## Hirschsprung’s Disease

### Perspective

Hirschsprung’s disease accounts for approximately 20% of cases of partial intestinal obstruction in early infancy. Hirschsprung’s disease occurs at a rate of 1 in 5000 live births and is four to five times more common in boys. It usually is sporadic in occurrence but may be associated with Down syndrome or a variety of other anomalies of the gastrointestinal, genitourinary, or central nervous system.32

### Principles of Disease

Hirschsprung’s disease represents congenital aganglionosis of the colon and is characterized by an absence of ganglion cells in the myenteric plexus of the distal colon.32 The anus is invariably involved, with aganglionic bowel usually extending proximally 4 to 25 cm. Absence of colonic ganglion cells interferes with that segment's ability to relax, creating a functional obstruction. Stool accumulates proximal to the level of obstruction and produces dilation of the colon, the so-called megacolon.

### Clinical Features

Neonates with Hirschsprung’s disease often present in the nursery with failure to pass meconium. Infants brought to the ED may have a history of constipation and obstipation. Vomiting, irritability, and abdominal distention may be features. Symptoms and signs may be subtle and include a history of chronic constipation and poor weight gain or failure to thrive. Hirschsprung’s disease usually is diagnosed in infancy; however, a spectrum of disease is recognized, and presentation may be later in life. Children who appear ill with fever should be evaluated for enterocolitis and toxic megacolon. Enterocolitis is characterized by abdominal distention, bloody stools, fever, and elevated white blood cell count.

### Diagnostic Strategies

Plain films of the abdomen may reveal evidence of fecal impaction with proximal obstruction, air-fluid levels, and dilated colon. A barium enema revealing a narrowed aganglionic segment with proximal dilation is highly suggestive of Hirschsprung’s disease.32,33 The diagnosis is confirmed by biopsy or manometry.

### Differential Considerations

Constipation is one of the most common causes of abdominal pain and vomiting in children.33,34 Children in the process of “potty training” occasionally become pathologic in their ability to delay defecation. Pathologic causes of constipation are
uncommon. In addition to Hirschsprung's disease, considerations include cystic fibrosis, infantile botulism, and hypothyroidism. An acquired variant of the disease also may occur in which other factors produce similar dilated colonic findings, resulting in acquired megacolon. Risk factors include anal fissures, fecal impaction, toilet training issues, and neuromuscular dysfunction secondary to neurologic disease, drugs, or metabolic causes.

The exact definition of constipation is elusive because it varies with age and diet. Infants during the first few months of life may have stool frequencies that range from one per feeding to one every other day, with breast-fed infants having more frequent stools than formula-fed infants. Frequency continues to decrease with age such that stools may average two or three per day during the first year of life and one or two per day from 1 to 5 years of age. Defecation occurs as a combination of physiologic, behavioral, and psychological factors. Relaxation of the external sphincter required for defecation is under voluntary control, whereas relaxation of the internal sphincter is involuntary. Children with a history of unpleasant or painful experiences associated with defecation may contract the external sphincter voluntarily in an effort to delay defecation for as long as possible. Accumulation of stool over time causes the rectum to dilate and decrease its propulsive activity, resulting in an increasing capacity for stool and chronic constipation. Acute episodes may be the result of dietary changes, travel, lack of normal exercise, or stress.

Management

Initial management is focused on ensuring adequate fluid and electrolyte status. Abdominal films should be obtained. With evidence of acute obstruction, seen as marked dilation, decompression may be necessary. Decompression usually can be accomplished easily with a rectal tube. Children who appear ill with fever should be evaluated for enterocolitis and toxic megacolon. Enterocolitis is characterized by abdominal distention, bloody stools, fever, and an elevated white blood cell count. Patients with enterocolitis require broad-spectrum, triple-antibiotic coverage with ampicillin, gentamicin, and either clindamycin or metronidazole. Urgent consultation should be made with a pediatric surgeon. Definitive therapy is surgical, with resection of the aganglionic segments. Acquired megacolon is managed by decompressing the colon and addressing the underlying problems.

Management of constipation requires three considerations: cleanout, maintenance, and behavior modification. Acute constipation is easier to manage because fewer functional problems are involved. The acute management of constipation usually is relatively easy and requires primarily the cleaning out of stool. Most experts recommend an approach that includes stool softeners or laxatives. Dietary modifications include increasing fiber and water in the diet and avoiding foods that may be constipating. Management of chronic constipation is more difficult and usually requires a multidisciplinary approach with attention to behavior modification. Children with an underlying pathologic process, such as cystic fibrosis, may benefit from more intense measures, such as administration of large volumes of polyethylene glycol (GoLYTELY).

Disposition

Unless children appear ill, most can be managed safely on an outpatient basis.

Meckel's Diverticulum

Perspective

Meckel's diverticulum is the most common congenital malformation of the small intestine and follows the rule of 2’s: The diverticulum is 2 cm wide and 2 cm long and usually located within 2 feet of the ileocecal valve. Moreover, the condition occurs in 2% of the population, and only 2% of affected patients ever become symptomatic. Half of all patients with the condition become symptomatic by the age of 2 years, and most present by the age of 20 years.

Principles of Disease

Diverticula are remnants of the omphalomesenteric duct and contain bowel wall, with 60% containing heterotopic tissue. This tissue most commonly involves gastric mucosa, but other types include pancreatic, duodenal, and endometrial tissue. Bleeding occurs when acid secretion from ectopic gastric mucosa causes ulceration and erosion.

Clinical Features

Patients are classically boys younger than 5 years who present with massive, painless rectal bleeding of acute onset; however, it can occur at any age. Some children may have some complaints of abdominal cramping. In older children, the presentation may be similar to appendicitis. The blood often is described as brick red. Complications may include intussusception, obstruction, perforation, and peritonitis.

Diagnostic Strategies

A technetium scan, also known as Meckel’s scan, is the diagnostic modality of choice and has an accuracy of 90% when ectopic gastric mucosa is present. Administration of pentagastrin, cimetidine, or glucagon may increase the sensitivity of the test. CT scan of the abdomen may be obtained to look for signs of obstruction or if the diagnosis is unclear. Definitive diagnosis is confirmed by laparoscopy or laparotomy.

Differential Considerations

Gastrointestinal bleeding is uncommon in childhood. The first step in evaluation of a child with suspected gastrointestinal bleeding is to determine whether the substance is actually blood. Children commonly eat or drink substances containing dyes that lead to fictitious changes in the stool's color. A simple Hemoccult test of the stool or Gastrocult test of the emesis can document the presence of hemoglobin. False-positive results may occur with red meat and iodine. Patients consuming products with bismuth (Pepto-Bismol), iron, and spinach may have black stools falsely appearing melanotic; the Hemoccult test result is negative.

After it has been determined that the substance is blood, the second step is to determine its origin. The location of bleeding often is difficult to determine but may be theorized on the basis of the appearance of the blood. Hematemesis implies bleeding proximal to the ligament of Treitz. Blood exposed to gastric acids for any time develops the classic coffee-grounds appearance. Bright red upper gastrointestinal bleeding implies either more proximal bleeding, such as from varices or esophagitis, or brisk gastric or duodenal bleeding. Bleeding that originates beyond the ligament of Treitz but proximal to the ileocecal valve results in melena. Hematochezia, with a visibly red to maroon appearance of the blood, implies bleeding from the descending colon. Distal lesions, such as fissures or hemorrhoids, can result in bright red
blood. Barium contrast studies of the upper and lower gastrointestinal tract sometimes are helpful; however, nuclear medicine scans (Meckel’s scan) are the procedure of choice for detection of Meckel’s diverticulum. Endoscopic examination has the highest rate of determining the location of bleeding; however, the diagnosis often is confirmed first on Meckel’s scan.

In neonates, the most common etiologic category of gastrointestinal bleeding is idiopathic. Careful examination of the rectum should be performed because the most identifiable cause of bleeding is a fissure or excoriation of the perirectal area. In young neonates, an Apt test may be performed to determine if the blood is maternal or fetal: 1% sodium hydroxide is added to the bloody stool. Fetal hemoglobin resists oxidation and remains pinkish red, whereas maternal hemoglobin changes to dark brown.

Another common cause of gastrointestinal bleeding in infancy is milk protein allergy. Affected children typically are younger than 6 months with a history of sudden-onset mucoid, blood-streaked stools. Children otherwise appear well. Although the causative allergy most commonly is to milk protein, gastrointestinal bleeding may occur with consumption of any protein and has been described in relation to soybean-based products. Children with persistent perianal excoriations and fissures refractory to standard emollients may be infected with group A streptococci and may benefit from treatment with an oral penicillin. Table 172-5 lists the differential considerations for gastrointestinal bleeding in children by age.

### Management

Management of gastrointestinal bleeding begins by assessment of circulatory status and ensuring that it is adequate. Screening laboratory studies should include CBC, coagulation studies (PT and PTT), and type and screen. Two radiographic views of the abdomen should be obtained in patients in whom obstruction or perforation is suspected. A technetium scan is the imaging modality of choice to evaluate for Meckel’s diverticulum. Consultation with a pediatric surgeon should be obtained.

### Disposition

Children with suspected Meckel’s diverticulum should undergo Meckel’s scan. Children with minor bleeding and normal findings on screening laboratory studies may be observed closely on an outpatient basis. Children with more active bleeding should be hospitalized for care by either a pediatric surgeon or a pediatric gastroenterologist.

### Henoch-Schönlein Purpura

**Perspective**

HSP, also known as anaphylactoid purpura, is a systemic vasculitis commonly associated with abdominal pain and rash. It is most common in children 4 to 11 years of age but also may occur in adults. HSP occurs most commonly in the spring after a viral upper respiratory infection. It also has been associated with insect stings and certain drugs.

**Principles of Disease**

HSP is a hypersensitivity vasculitis with immune complex deposition with immunoglobulin A, mainly affecting the arterioles and capillaries. Although it is most well known for its characteristic petechial to purpuric rash, HSP is a systemic vasculitis and may affect any blood vessel. Less common manifestations are seen in cases in which the disease is more severe or widespread.

### Clinical Features

Symptoms include abdominal pain, nausea, vomiting, and diarrhea. Patients most often are diagnosed clinically on the basis of the classic rash, abdominal pain, microscopic hematuria, and arthralgias. The classic rash is palpable purpura located on the buttocks and lower extremities (Fig. 172-10). Up to 70% of patients with HSP have gastrointestinal complaints. Microscopic
hematuria occurs in 50%. Intussusception may occur and may be atypical in that ileoileal intussusception is more common than the ileocolic intussusception that normally occurs. The syndrome often is relapsing and remitting for several weeks and may be associated with arthralgias. Neurologic involvement also may be a feature of HSP, although this manifestation is uncommon in children.

Diagnostic Strategies

Patients most often are diagnosed clinically on the basis of the classic rash, abdominal pain, microscopic hematuria, and mild arthralgias. Screening studies should include a CBC with differential and platelet counts, urinalysis, blood culture, and sedimentation rate determination. Children with worrisome abdominal pain require CT to rule out ileoileal intussusception.

Differential Considerations

The most important entity in the differential diagnosis for this type of rash is meningococccemia, in which the patient has a fever and looks very ill. It is essential to rule out meningococccemia because the condition is life-threatening and requires an entirely different approach to management involving hospitalization, fluid resuscitation, and intravenous antibiotics. The classic triad of palpable purpura, abdominal pain, and hematuria in an otherwise well-appearing child virtually ensures the diagnosis. Erythema nodosum occasionally is confused with the rash of HSP; however, the rash of erythema nodosum is described most often as subcutaneous purplish red nodules with the appearance of a bruise on the extensor surfaces of the distal extremities. Erythema nodosum usually involves only the shins but in more severe cases also may involve the forearms, hands, and feet.

Management

Most children with HSP can be managed with close follow-up and require no treatment other than symptomatic support. Severe or intermittent abdominal pain in children with HSP is suggestive of intussusception; imaging to investigate this possibility should be done with ultrasonography, followed by CT scan if the ultrasound study is normal because ileoileal intussusception may occur and is not detected easily by ultrasonography. Management with steroids is controversial. Corticosteroids, at a dose of 1 mg/kg/day (maximum, 60 mg), are reserved for patients with illness on the more severe end of the spectrum, with abdominal pain, hematuria, or arthralgias.

Disposition

Indications for hospital admission include uncertain diagnosis to exclude the possibility of meningococccemia, severe abdominal pain, and vomiting. Most patients can be managed safely with close outpatient follow-up.

Inflammatory Bowel Disease

Perspective

The two major entities included within the category of IBD are Crohn’s disease and ulcerative colitis. Approximately 20% of patients present before the age of 20 years. Most patients do not experience symptoms until adolescence, although childhood presentations have been described. IBD is rare in children younger than 1 year.

Principles of Disease

Ulcerative colitis is an inflammatory disease primarily involving the mucosa and submucosa of the rectum and distal colon. Crohn’s disease is a transmural inflammatory disease that may involve any portion of the intestinal tract. The most common area of involvement in cases with single-segment disease is distal ileum; however, multiple segments in different areas may be involved. Chronic inflammation may result in the formation of abscess, fistula, or stricture.

Clinical Features

Although patients experiencing complications frequently present to the ED, the diagnosis is rarely made in this setting. More commonly, children with known disease present in the midst of a flare, usually with bloody diarrhea and abdominal pain. Extraintestinal manifestations also occur and include fever, anemia, oral ulcerations, erythema nodosum, pyoderma gangrenosum, uveitis, liver dysfunction, and failure to thrive. Some of these manifestations may occur even before the child has experienced any gastrointestinal symptoms. The most feared complication is toxic megacolon, which classically is manifested with abdominal pain, fever, and bloody diarrhea and is associated most often with ulcerative colitis.

Diagnostic Strategies

Flares of IBD are diagnosed by the increased frequency of diarrheal or bloody stools and abdominal pain. Patients who appear ill require plain films of the abdomen to rule out toxic megacolon. Patients with toxic megacolon usually have a fever, appear volume depleted, and demonstrate significant abdominal tenderness to palpation. X-ray films reveal dilation of the transverse colon to more than 6 to 7 cm in diameter. Free air also should be looked for because perforation may occur. Screening laboratory studies should include a CBC with differential and platelet counts, type and screen, coagulation studies (PT and PTT), and electrolyte panel.
Differential Considerations

There are a wide number of differential considerations for abdominal pain and gastrointestinal bleeding (see Tables 172-4 and 172-5). Gastroenteritis is the most common consideration in this clinical scenario. Children experiencing their first episode of IBD are much more likely to be misdiagnosed with an acute gastroenteritis. Children outside the usual age at presentation also are more likely to be misdiagnosed with gastroenteritis. Children with recurrent symptoms or a family history of IBD should be referred to a pediatric gastroenterologist for further evaluation.

Management

Initiation of corticosteroid therapy in conjunction with a gastroenterologist constitutes the mainstay for treatment of acute exacerbations. Prednisone at a dose of 1 mg/kg/day (maximum, 60 mg/day) usually is recommended and should be provided in consultation with a pediatric gastrointestinal specialist. Other agents commonly used include sulfasalazine, azathioprine, and a host of other immunosuppressive agents. Management in the ED begins with attention to volume status and resuscitation with boluses of 20 mL/kg of normal saline until the volume status is adequate. Patients with suspected toxic megacolon require intravenous broad-spectrum, triple-antibiotic therapy (with ampicillin, gentamicin, and metronidazole) and immediate surgical consultation.

Disposition

Indications for admission include children who are dehydrated and toxic or ill appearing and those patients for whom the diagnosis is uncertain. Children with ongoing diarrhea productive of bloody stools usually benefit from intravenous fluids until the flare has been controlled. Children with evidence of toxic megacolon require surgical consultation and admission.

Gastrointestinal Foreign Bodies

Perspective

Most gastrointestinal foreign bodies occur in toddlers, who experience life by first putting objects in their mouths. Children younger than 3 years are particularly at risk because of the combination of inappropriate mouthing of objects and a general lack of coordination in swallowing. Although food is the most common esophageal foreign body in adults, coins are most common in children. Children with mental retardation swallow a variety of objects. Adolescents occasionally swallow objects in an attempt at suicide. Rectal foreign bodies are uncommon in the pediatric age group.

Principles of Disease

Most swallowed foreign bodies pass without difficulty. Foreign bodies may become lodged in any of three areas of normal physiologic narrowing: upper esophageal sphincter (cricopharyngeus muscle)—thoracic inlet (C6-T1), aortic arch—tracheal bifurcation (T4-6), and lower esophageal sphincter—diaphragmatic hiatus (T10-11). In general, 80 to 90% of objects that have made it into the stomach are passed without difficulty. Aspirated objects generally produce persistent coughing, wheezing, or increased work of breathing. Objects that have been swallowed may result in the child’s remaining asymptomatic or may produce symptoms ranging from persistent gagging to drooling, continuous dry heaves, or wheezing. Larger foreign bodies may compress the airway and cause significant respiratory distress. Rapidly progressive symptoms of dysphagia, pain, respiratory distress, or fever raise the possibility of a perforation. Perforation is uncommon, even with sharp objects such as straight pins. The ileocecal valve is the most common site for perforation, which occurs in less than 1% of patients. Button batteries warrant special mention. Button batteries in the esophagus should be removed as rapidly as possible because erosions and mediastinitis ultimately may occur. Button batteries in the stomach usually pass without difficulty and do not require removal unless they fail to pass the pylorus within 24 to 48 hours of ingestion. The National Capital Poison Center in Washington, DC, operates a 24/7 website (http://www.poison.org/battery/) and hotline (202-625-3333) for battery ingestion cases. On occasion, objects pass into the stomach that are too large to pass through the pylorus; this is uncommon and is heralded by persistent vomiting. Long-present unrecognized foreign bodies may result in erosion, perforation, infection, stricture, or fistula formation.

Diagnostic Strategies

Plain radiography is the most common method of visualizing location and is used to verify positioning past the lower esophageal sphincter into the stomach. Patients who are symptomatic after foreign body ingestion require imaging to determine location. On occasion, patients who are asymptomatic also have objects remaining in the esophagus. Radiographs should include anteroposterior and lateral views of the chest, neck, and abdomen (Fig. 172-11). The lateral view helps delineate soft tissues in the hypopharynx and evaluate for swelling, particularly if the foreign body is either unknown or of a nonradiopaque material. A single view of the neck, chest, and upper abdomen often can be obtained easily in the pediatric patient. Unless the patient becomes symptomatic, repeated films are otherwise never necessary. Asking parents to sieve the stool typically is unproductive, and even sharp objects such as pins and razor blades usually pass through the gastrointestinal tract without incident. Patients presenting with a history of button battery ingestion represent the exception in that they require repeated films to document passage beyond the pylorus. Rather than using standard radiography, some institutions have noted success with intradepartment fluoroscopy or hand-held metal detectors. Contrast studies may be helpful to delineate nonradiopaque foreign bodies or to evaluate for perforations.

Differential Considerations

Not all foreign bodies are radiopaque and visible with standard radiography. Patients who remain symptomatic require further contrast-enhanced imaging or direct visualization.

Management

If the object has made it into the stomach, usually it will pass without incident, and no further treatment is necessary. If the foreign body is in the esophagus, most experts recommend removal within 24 hours to decrease the risk of aspiration and esophageal erosion. The preferred method by which to remove esophageal foreign bodies is controversial and varies by institution. Options include fluoroscopic Foley catheter removal, bougie advancement into the stomach, endoscopic removal in the ED, and removal by rigid bronchoscopy under general anesthesia in the
approximately 1 case in 50,000 in children and carries a mortality rate of 14%. In adults, pancreatitis is associated most commonly with alcohol and biliary tract disease. In children, a fairly equal association of approximately 10 to 20% each has been documented for trauma, infection, structural disease, systemic disease, and drugs or toxins. Mumps is the most common viral cause of pancreatitis and accounts for 10 to 15% of all cases. Idiopathic causes account for 30% of cases. Biliary obstruction should be considered in the adolescent.

**Principles of Disease**

Whether the result of trauma, obstruction, or inflammation, a series of events occurs, resulting in inflammation, edema, and autodigestion of pancreatic tissue by pancreatic enzymes. In severe cases, the inflammatory process may progress to necrosis and hemorrhage, resulting in necrotizing or hemorrhagic pancreatitis. Further complications include the formation of abscesses, pseudocysts, and fistulas.

**Clinical Features**

Patients classically present with complaints of severe epigastric pain that radiates to the back. Pain is gradually progressive and constant and often is associated with nausea and vomiting. Pain classically is described as being worse with eating. Significant abdominal tenderness usually can be elicited in the epigastric area, accompanied by voluntary guarding and hypactive bowel sounds. The abdomen may be slightly distended. Patients with biliary colic and gallstone pancreatitis have variable presentations from classic right upper quadrant pain associated with nausea and vomiting to intermittent epigastric pain and jaundice.

**Diagnostic Strategies**

Screening laboratory studies reveal elevations in serum lipase. Evidence of liver inflammation and elevated bilirubin and alkaline phosphatase may be seen in patients with biliary obstruction. Plain films of the abdomen may be indicated to investigate the possibility of free air or obstruction. An ileus pattern is common, often with a sentinel loop of dilated small bowel noted in the left upper quadrant. An ultrasound study or CT scan may be helpful to evaluate anatomy for congenital malformations or biliary tract disease and to evaluate for pseudocyst or abscess formation. In patients with pseudocysts, hemorrhagic pancreatitis may develop that may become life-threatening. For patients with respiratory distress, a chest film can be helpful to evaluate for a coexistent pleural effusion caused by the pancreatitis.

**Differential Considerations**

Slow, progressive onset of pain is more likely to be associated with appendicitis, constipation, or pancreatitis. Children with peritoneal irritation invariably lie still, often on the side with knees bent, and refrain from all extraneous movement. Sudden onset of severe pain is associated most often with acute obstruction or vascular occlusion as seen with intussusception, volvulus, or torsion. Differential considerations for abdominal pain in children by age are listed in Table 172-4; differential considerations for the causes of pancreatitis are listed in Table 172-6.

**Management**

Management begins with attention to volume status and correction of electrolyte abnormalities if present. A bedside finger-stick blood glucose value should be obtained. Adequate pain relief with parenteral narcotics should be provided. The patient should be

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**Figure 172-11.** Plain radiographs obtained in a child with an esophageal coin foreign body. Posteroanterior (A) and lateral (B) views show the expected orientation for a coin lodged in the esophagus. (Courtesy Mark A. Hostetler, MD.)

operating room. Foley catheter removal and bougienage do not require sedation in cooperative patients, but young children invariably require sedation in the operating room. Children should be positioned on the side to decrease the risk of aspiration. With the last two methods, the technique involves actually holding onto the foreign body, which—at least theoretically—decreases the chance of inadvertent aspiration. They also have the additional benefit of directly visualizing the integrity of the mucosa and evaluating for perforation. Gastric foreign bodies generally do not require removal. Indications for surgical removal of gastric foreign bodies include objects that are more than 2 cm in width, objects that are more than 5 cm in length, and objects that are sharp (pins, razor blades).

**Disposition**

Esophageal foreign bodies require removal as described previously. After foreign bodies have made it into the stomach, most pass without difficulty, and no further follow-up is necessary. Button batteries constitute the exception and necessitate follow-up films to document passage beyond the pylorus.

**Pancreatitis**

**Perspective**

Pancreatitis is uncommon in childhood, especially in children younger than 10 years. Pancreatitis has an incidence of...
Appendicitis

Perspective

Appendicitis is the most common surgical condition involving the abdomen and the most common nontraumatic surgical emergency in children. Approximately 200,000 appendectomies are performed every year, and appendicitis develops in approximately 1 of every 15 people in the general population sometime during their lifetime. The peak age of incidence of appendicitis is between 9 and 12 years, and it is uncommon in children younger than 5 years.

Acute appendicitis has an overall mortality rate of less than 1%. For unruptured appendicitis, the mortality rate is 0.1%; the mortality rate increases to approximately 3% for ruptured appendicitis. In children, the rate of appendiceal perforation before surgery varies, ranging from 17 to 40%, and is inversely related to age, with higher rates of perforation occurring in younger age groups. In children younger than 2 years, perforation will have occurred in 90% by the time of surgery.

Principles of Disease

The appendix is a blind pouch that may become obstructed. After it is obstructed, a vicious circle is established, with increasing edema, vasocongestion, inflammation, ischemia, infarction, necrosis, and perforation. In adults, a thicker appendiceal wall protects against perforation, and a well-developed omentum aids in walling off the infection to prevent its diffuse spread. Children have neither, so rupture tends to occur earlier and diffuse peritonitis develops more readily.

Clinical Features

Patients classically present with a constellation of symptoms that includes abdominal pain, nausea, vomiting, fever, and anorexia. All of these symptoms are gradually progressive during 4 to 24 hours. Abdominal pain usually is described as vague, crampy, and periumbilical on origination, which then becomes more severe, constant, and localized to the right lower quadrant. Fever usually develops later, sometimes not until after the patient has presented to the ED. Nausea and vomiting are progressive and most often are associated with anorexia. Patients occasionally may have a multiphasic course to their illness, which begins with the classic abdominal pain, with progression of symptoms until they suddenly resolve, followed several days later by development of fever, chills, and abdominal pain. This course represents acute appendicitis with spontaneous rupture and formation of an abscess.

Physical examination may reveal several classic findings. In patients with inflammation surrounding the appendix, peritoneal findings that localize to the right lower quadrant are typical. Pain occurs with movement, so the patient prefers to lie still. Patients are unable to jump up and down and complain that even rocking the bed or tapping their heels causes pain. The abdomen usually is quiet, with an absence of bowel sounds. Rebound tenderness can be elicited in the right lower quadrant. Rovsing’s sign also may be present and consists of severe pain in the right lower quadrant occurring when the examiner presses in the left lower quadrant and rapidly releases the examining hand. Other findings associated with appendicitis include the psoas and obturator signs. The psoas sign is pain elicited by having the patient, in lateral decubitus position, hyperextend the right thigh at the hip, thereby stretching the psoas muscle, which overlies the inflamed appendix. The obturator sign similarly is pain elicited by having the patient internally rotate the flexed right thigh against resistance. These signs are not frequently present but when present increase significantly the likelihood of appendicitis.

Diagnostic Strategies

Appendicitis may be diagnosed on the basis of history and physical findings alone. Patients with the appropriate constellation of findings consistent with appendicitis may not require any testing and can proceed directly to the operating room for either laparoscopic or open exploration. Patients in whom the diagnosis is suspected usually require some diagnostic workup. Screening studies should include a CBC with differential, urinalysis, and pregnancy test. An estimated 96% of patients with appendicitis have either an elevated white blood cell count greater than 10,000/µL or a left-shifted differential with more than 75% neutrophils. Although an elevated white blood cell count supports the diagnosis, it is not specific for appendicitis. The appendix is close to the ureters, so...
appendicitis may induce some degree of sterile pyuria. Inflammatory changes related to appendicitis generally result in fewer than 5 to 10 white and red blood cells per high-power field and an absence of bacteria. Findings in excess of these amounts suggest a urinary tract etiology (e.g., infection, stone, mass, trauma). Consideration also may be given to a rapid streptococcal test in patients with a red or sore throat as streptococcal pharyngitis may present with abdominal pain.

Diagnostic imaging options include plain films of the abdomen, ultrasonography, and limited CT scan of the appendix. Plain films help rule out free air or obstruction and occasionally show an appendiceal fecalith, also called an appendicolith (Fig. 172-12). Although the presence of an appendicolith is essentially pathognomonic for acute appendicitis, it is present in only 10% of cases. Ultrasound findings consistent with appendicitis include an enlarged, noncompressible appendix that is painful during scanning (Fig. 172-13A and B). A fecalith may be evident inside an enlarged and inflamed appendix, the so-called target sign. Appendiceal ultrasound studies have a sensitivity, specificity, and overall accuracy of 90 to 95%. Ultrasonography imaging is particularly useful to evaluate for abscesses and fluid collections; however, a great deal of operator and reader variability is characteristic (Fig. 172-13C). Ultrasonography does not involve exposure to ionizing radiation. Use of an ultrasound examination as an initial screen for appendicitis, in which only patients with normal or equivocal scans were required to undergo CT, resulted in a sensitivity of 96% and a negative predictive value of 98%.

Limited CT of the appendix is the most recent technology to have emerged in terms of imaging. Limited CT scans of the appendix have sensitivity and specificity rates of 95 to 100%. The ready availability of appendiceal CT has been shown to reduce the negative laparotomy rate from 20% to 7%; the highest accuracy is reported with use of new focused appendiceal CT techniques with rectal administration of contrast material. Imaging of the pelvis and the right lower quadrant (with either CT or ultrasonography) not only establishes the diagnosis of appendicitis but also aids in identifying the two most common considerations in the differential diagnosis: IBD and mesenteric adenitis. An important point is that CT scanning involves exposure to a significant amount of ionizing radiation, which places children at increased risk for future malignant neoplasms. Beginning in the past decade, the number of CT scans has increased dramatically (nearly 700%), and appendicitis is believed to be the single primary reason accounting for its surge in use. In weighing all of the risks and benefits, sequential imaging with ultrasonography followed by CT scan is likely to be the best choice.

**Differential Considerations**

Mesenteric adenitis is the most common condition associated with the signs and symptoms of appendicitis. It often is associated with significant diffuse tenderness and may localize in the right middle to lower quadrant. Children often lack fever, however, and
do not have true peritoneal signs. Mesenteric adenitis usually follows a viral illness and results from nonspecific inflammation of mesenteric lymph nodes. It is perhaps even more common than appendicitis. Differential considerations for abdominal pain by age are listed in Table 172-4. Other considerations include nonaccidental trauma (abuse), malingering, and Munchausen syndrome.67 Girls of reproductive age merit consideration of a gynecologic origin and require a pregnancy test and pelvic examination; the possibility of ovarian torsion, particularly if the pain seems to be severe and out of proportion to physical examination findings, should not be overlooked. Boys require an external genital examination to rule out the possibility of testicular torsion. Cryptorchidism in the face of acute abdominal pain should raise suspicion for testicular torsion.

Management

In patients with suspected appendicitis, NPO status should be maintained and an intravenous line established. Most patients have vomiting and anorexia and benefit from at least one fluid bolus of 20 mL/kg of normal saline and then administration of 1.5 to 2 times maintenance fluids with dextrose 5% in water and half-normal saline and administration of antiemetics. Screening studies should be initiated, and appropriate consultation with a surgeon is indicated. Ongoing pain should be addressed adequately. Intravenous narcotics are safe and effective and should not alter the diagnostic accuracy of the physical examination. Patients with high fever, suspected perforation, or unusual delay to surgery require coverage with intravenous broad-spectrum antibiotics, which should be initiated in the ED after consultation with the surgeon.68,69 A reasonable regimen for antibiotic therapy consists of ampicillin, gentamicin, and either metronidazole or clindamycin. Nasogastric tubes are reserved for patients with persistent nausea or vomiting related to abdominal distention or ileus.

Disposition

Children with appendicitis as documented by physical examination or diagnostic imaging are hospitalized for appendectomy. Patients with nonspecific symptoms and signs who do not undergo definitive diagnostic study can be hospitalized and observed for a period of 12 to 24 hours or, with adequate family and social support, discharged home with careful instructions to return for re-examination.

Biliary Tract Disease

Perspective

Biliary tract disease is uncommon in childhood and has causes distinct from those of disease in adults. In children, gallstones are associated with hemolytic disease, cystic fibrosis, total parenteral nutrition, sepsis, and dehydration.60,61 Ceftriaxone also has been associated with sludging and biliary disease, particularly in neonates. Acute acalculous cholecystitis has been associated with Rocky Mountain spotted fever and a variety of bacterial infections, including those due to Salmonella and Shigella organisms. Hydrops of the gallbladder (i.e., fluid distention of the gallbladder from chronic cystic duct inflammation or obstruction) is associated with viral upper respiratory or gastrointestinal infections, Kawasaki disease, streptococcal pharyngitis, mesenteric adenitis, nephrotic syndrome, and leptospirosis.

Principles of Disease

Gallstones are classified as either cholesterol or pigment stones. Pigment stones occur in childhood, whereas cholesterol stones usually do not begin appearing until adolescence. Pigment stones result from the excess breakdown of red blood cells and are seen most commonly in hemolytic anemia, such as from sickle cell disease and spherocytosis. Gallstones occurring in infants usually are associated with abdominal surgery, sepsis, NEC, or administration of total parenteral nutrition.21 In young children, gallstones most commonly develop as a result of their hemolytic disease. Adolescents form gallstones in association with oral contraceptives, pregnancy, obesity, or underlying hemolytic disease. In acute acalculous cholecystitis, the ultrasound scan will reveal evidence of gallbladder inflammation in the absence of calculi. Hydrops of the gallbladder is an acute noninflammatory, noninfectious process that results in a markedly enlarged gallbladder without evidence of calculi.

Clinical Features

Similar to adult patients, pediatric patients usually present with right upper quadrant pain that radiates through to the back and may be associated with fever, nausea, and vomiting. Jaundice occurs in one third of patients.71,72

Diagnostic Strategies

Biliary tract disease usually is associated with elevations in liver enzymes and bilirubin; however, absence of elevations does not exclude the diagnosis. Accordingly, right upper quadrant ultrasound imaging should be undertaken in cases in which gallbladder disease is suspected. Elevations in alkaline phosphatase suggest cholestasis. Elevated white blood cell counts are nonspecific but if associated with fever may represent an acute infectious process (e.g., ascending cholangitis). Ultrasonography is the imaging modality of choice. It can reproduce pain on scanning (sonographic Murphy’s sign) and determine the presence of gallstones, amount of dilation of the gallbladder and bile ducts, gallbladder wall thickness, presence of pericystic fluid consistent with the diagnosis, and anatomic configuration of the biliary and pancreatic collecting system. When ultrasound findings are equivocal or normal in the face of strong clinical evidence, the “gold standard” modality for biliary tract imaging is still considered to be cholescintigraphy (DISIDA scanning); rarely, percutaneous cholecystocholangiography is necessary.71 Although only 15% of gallstones in adults are calcified and visible on plain radiographs of the abdomen, 50% of stones are visible in children.72,73

Differential Considerations

Biliary tract disease is uncommon in children and requires consideration of underlying or coexistent disease. Differential considerations for abdominal pain by age are listed in Table 172-4.

Management

Management of biliary tract disease begins with attention to fluid and electrolyte status. Adequate pain control should be provided, usually with a parenteral opioid. Asymptomatic patients with incidental findings of gallstones require no further therapy in the ED and may be referred to a surgeon for outpatient care. Patients who are febrile usually can be managed safely on an outpatient basis with adequate pain control. Febrile patients require hospital admission and intravenous antibiotics. A reasonable regimen for empirical antibiotic coverage consists of ampicillin and gentamicin plus either clindamycin or metronidazole.

Several management options have been used in the treatment of children with cholecystitis: medical management with ursodeoxycholic acid to dissolve the stone, expectant management, and
surgical management. Laparoscopic cholecystectomy is considered safe and effective in children.

Disposition
Indications for hospital admission include pain control, hydration, fever, and need for operation.

KEY CONCEPTS
- Physiologic jaundice of the newborn and breast milk jaundice are the most common causes of jaundice in the neonatal period.
- Direct hyperbilirubinemia in infants is always pathologic and requires a detailed workup.
- Hypertrophic pyloric stenosis is manifested with gradually progressive nonbilious emesis that becomes projectile.
- Hypochloremic, hypokalemic metabolic alkalosis is the classic electrolyte abnormality for children with hypertrophic pyloric stenosis.
- Bilious vomiting in the neonate should initiate a diagnostic evaluation for possible malrotation and volvulus.
- In infants with bilious emesis, a toxic appearance, a distended abdomen, or an acute obstructed pattern on abdominal radiographs constitutes a true surgical emergency.
- Necrotizing enterocolitis occurs more commonly in premature infants, but 10% of affected infants are full term.

- Gastroesophageal reflux usually responds to conservative measures (positioning, thickening of formula, smaller and more frequent feedings).
- The classic clinical triad in intussusception consists of abdominal pain, vomiting, and heme-positive stools; however, the triad occurs in less than one third of patients.
- Children with intussusception may present without pain and with lethargy alone.
- Hirschsprung's disease is a common cause of constipation in the neonate and usually is manifested by delayed passage of meconium.
- Meckel's diverticulum classically is manifested in children younger than 5 years with painless brick-red rectal bleeding.
- More than 90% of gastrointestinal foreign bodies pass without difficulty.
- Causes of pancreatitis in children include viruses, trauma, drugs, and toxins.
- Imaging strategies for children with suspected appendicitis that include initial ultrasound examination followed by CT scan of the abdomen have been shown to reduce costs and negative laparotomy rates.
- Cholelithiasis is diagnosed with right upper quadrant ultrasound imaging; management strategies include medical and surgical approaches.

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