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Contributor Disclosures

All topics are updated as new evidence becomes available and our peer review process is complete.
Literature review current through: Dec 2017. | **This topic last updated:** Nov 07, 2017.

INTRODUCTION — Lower gastrointestinal bleeding (LGIB) in infants and children is commonly encountered in clinical practice, although its epidemiology has not been well studied [1-5]. In a large series from 1994, rectal bleeding was the chief complaint in 0.3 percent of more than 40,000 patients presenting to a pediatric emergency department (ED) in Massachusetts [1]. Bleeding was considered to be life-threatening in only four patients (4.2 percent), three of whom had ileocolic intussusception and one of whom had bleeding from a Meckel's diverticulum. In a subsequent nationwide study, 11.6 percent of pediatric patients presenting to the ED with gastrointestinal bleeding were admitted to the hospital, suggesting that most cases presenting to the emergency department with gastrointestinal bleeding are not life-threatening [6]. Approximately 30 percent of the overall cases presenting to the ED were classified as LGIB, 20 percent were upper gastrointestinal bleeding, and the remainder were unspecified.

This topic review will discuss the evaluation of LGIB that presents with bright red blood per rectum, for which the most likely causes are organized by age group. The evaluation of children presenting with hematemesis or other signs of upper gastrointestinal bleeding (UGIB) are discussed separately (see "Approach to upper gastrointestinal bleeding in children"). Many of the causes of LGIB that are listed here are discussed in detail in separate topic reviews.

DEFINITIONS

- **Upper gastrointestinal bleeding** (UGIB) refers to bleeding that originates from the gastrointestinal (GI) tract proximal to the ligament of Treitz (the junction of the duodenum and jejunum). It includes bleeding sources in the esophagus, stomach, and duodenum.
- **Lower gastrointestinal bleeding** (LGIB) refers to bleeding distal to the ligament of Treitz, and thus includes bleeding sources in the small bowel and colon. It is sometimes subcategorized as bleeding from the small bowel (sometimes termed mid-GI bleeding), or bleeding from the colon.

Gastrointestinal bleeding can be further categorized based on qualitative characteristics of the stool:

- **Hematochezia** describes the passage of bright red blood per rectum and usually suggests LGIB, typically from the colon or anus. Rarely, hematochezia can be caused by UGIB, due to rapid intestinal transit time or massive UGIB. (See 'Diagnostic approach' below.)
- **Melena** describes stools that appear black and tar-like, and usually suggests UGIB. Melena may also be caused by blood from the nose that is swallowed or bleeding from the proximal small bowel. Black-appearing stools that do not contain blood may be caused by certain medications or foods. (See 'Diagnostic approach' below.)
- **Occult** (hidden or unseen) GI bleeding is not visible to the patient or physician. It usually presents as iron deficiency anemia or is identified by testing the stool for occult blood. (See 'Testing for blood in the stool' below.)

CAUSES OF BLEEDING — The likely causes of lower gastrointestinal bleeding (LGIB) vary depending upon age. In the United States, the most common causes of rectal bleeding in infants are anal fissure or cow's milk or soy protein-induced colitis [1]. In children 12 months and older, the most common causes of

rectal bleeding are infectious gastroenteritis and anal fissures. In each age group there are other disorders that are less common but important to identify because they may be life-threatening and/or require specific treatment. The spectrum of causes is different in other parts of the world. In a report from India, for example, 24 percent of 85 children bled from amoebic ulcers [2].

In addition, various foods and medications may cause stool to falsely appear bloody or test positive for blood (table 1), as discussed below. (See 'Testing for blood in the stool' below.)

The following discussion will focus on the causes of bleeding most commonly seen in the United States during the neonatal period (younger than one month); infants and toddlers (one month to two years); preschool period (two to five years); and the school-age period or adolescence (older than five years) (figure 1 and table 2). Many of the disorders are encountered in more than one of these age groups.

Neonatal period — The most common diagnoses to consider in newborns presenting with LGIB are:

- Swallowed maternal blood
- Anorectal fissures
- Necrotizing enterocolitis
- Malrotation with midgut volvulus
- Hirschsprung disease with enterocolitis
- Coagulopathy
- Brisk upper gastrointestinal (GI) bleeding
- Vascular malformations
- Gastric or duodenal ulcer
- Gastrointestinal duplication cyst

Swallowed maternal blood — In a newborn infant with rectal bleeding, the rectal blood should be tested to determine whether it comes from the infant or whether it represents maternal blood, which may have been swallowed during delivery or ingested during breast feeding from cracked nipples. This is accomplished using the Apt test (hemoglobin alkaline denaturation test), which detects fetal hemoglobin.

The Apt test takes advantage of the different susceptibilities of adult hemoglobin A (HbA) and fetal hemoglobin (HbF) to alkali denaturation. Infant HbF resists denaturation with alkali better than adult HbA. To perform the Apt test, fresh stool is mixed with water (1:5 dilution) and centrifuged, and sodium hydroxide then is added to the supernatant [7]. Adult Hb changes to brown-yellow within two minutes (alkaline hematin), while fetal hemoglobin resists denaturation and retains its pink color. Exposure of the stool sample to air for more than 30 minutes will cause fetal hemoglobin to have the same color change as adult hemoglobin, resulting in a false-positive result. In cases in which the Apt test is inconclusive, a spectrophotometric assay can be used to quantify the color change. HbF >50 percent indicates fetal blood and HbF <10 percent suggests maternal blood [8].

Anal fissures — Anal fissures are the most common cause of rectal bleeding in patients younger than one year and are also common in older children and adults. They are diagnosed easily by spreading the perineal skin to evert the anal canal. In an infant, the history often suggests painful defecation with straining, grunting, and leg stiffening or back arching consistent with withholding behavior and streaks of bright red blood on the surface of the stools. When associated with constipation, anal fissures usually respond to stool softeners and lubricants such as petrolatum (Vaseline). When associated with diarrhea, healing is hastened by keeping the perineum clean and dry [9]. Vigorous wiping or the use of glycerin suppositories should be

avoided because they may further irritate the anal mucosa. (See "Prevention and treatment of acute constipation in infants and children".)

Infants and children are more likely to develop constipation with associated risk for anal fissures during the following periods:

- The introduction of solid foods or cow's milk into the diet
- Toilet training
- School entry

Necrotizing enterocolitis — Necrotizing enterocolitis (NEC) is an acute illness of unclear etiology associated with intestinal necrosis. (See "Pathology and pathogenesis of necrotizing enterocolitis in newborns" and "Clinical features and diagnosis of necrotizing enterocolitis in newborns".)

NEC should be suspected in a newborn with nonspecific systemic signs such as apnea, respiratory failure, lethargy, poor feeding, or temperature instability, and abdominal signs including distention, gastric retention (residual milk in the stomach before a feeding), tenderness, vomiting, diarrhea, and gross or occult LGIB. Although most infants who develop NEC were born prematurely, approximately 13 percent of cases occur in term infants [10]. The disease occurs predominantly in infants receiving enteral nutrition. Characteristic features in the supine abdominal radiograph are seen in most infants with suspected NEC. An abnormal gas pattern, with dilated loops of bowel consistent with ileus, is typically seen in the early stages. Pneumatosis intestinalis, the hallmark of NEC, appears as bubbles of gas in the bowel wall or in the portal system.

Malrotation with midgut volvulus — Newborns who have malrotation with midgut volvulus typically present with abdominal distension, emesis which may or may not be bilious, and melena or hematochezia (in 10 to 20 percent of cases). Bilious emesis in the neonatal period should be assumed to represent a surgical emergency due to obstruction until proven otherwise.

Symptomatic malrotation is life-threatening and requires emergent evaluation and treatment. The diagnosis can be suggested by plain abdominal radiographs, but normal radiographs do not exclude it. A limited upper gastrointestinal (UGI) contrast series is the best examination to visualize the position of the duodenum; failure of the duodenum to cross the midline confirms the presence of malrotation. UGI contrast series should be performed whenever possible, under fluoroscopy and by an experienced pediatric radiologist. The addition of a small bowel follow-through or barium enema to check for colonic malrotation is prudent in patients in whom the UGI is normal but there is a high index of suspicion or signs of a distal bowel obstruction. Barium contrast studies may reveal a corkscrew appearance of the twisted small bowel, or a "bird's peak" if complete obstruction is present. The clinical presentation, evaluation, and management of malrotation are discussed in greater detail separately. (See "Intestinal malrotation in children".)

In experienced hands, intestinal malrotation can be diagnosed by color Doppler ultrasonography. Malrotation is seen as inversion of the superior mesenteric artery (SMA) and superior mesenteric vein (SMV) relationship, with the SMA on the right and SMV on the left, along with other findings such as duodenal dilatation with a tapering configuration [11-13]. A normal ultrasound, however, does not exclude malrotation. (See "Intestinal malrotation in children", section on 'Imaging in stable patients'.)

Hirschsprung disease — Newborns with Hirschsprung disease frequently have delayed passage of meconium (>48 hours after birth). Some present with acute obstruction manifested by vomiting (which may be bilious or feculent) and abdominal distension. Other infants may present at several weeks of age with progressive constipation or diarrhea associated with abdominal distension. Only one-quarter of the patients have blood in the stool. The diagnosis of Hirschsprung disease should also be suspected in older infants with chronic refractory constipation, such as those who need mechanical or pharmacologic assistance to

defecate. The diagnosis also is suggested by explosive expulsion of gas and stool after the digital rectal examination (squirt sign or blast sign). (See "Congenital aganglionic megacolon (Hirschsprung disease)".)

Significant blood in the stool with abdominal distension in infants with known or suspected Hirschsprung disease may be indicative of Hirschsprung-associated enterocolitis (toxic megacolon) and should be considered a medical emergency. In this situation, the rectum needs to be immediately decompressed by either rectal exam and/or the placement of a rectal tube, and appropriate antibiotic therapy should be initiated. (See "Emergency complications of Hirschsprung disease".)

In a stable patient, a contrast enema may be used as an initial test; the study is performed "unprepped", meaning that the stool should not be evacuated by enema or other means prior to the test. When diagnostic, the enema demonstrates marked dilation of the unaffected colon proximal to the aganglionic segment, which is smaller in comparison. Anorectal manometry (ARM) can be used for confirmatory testing if needed, and is also necessary to diagnose ultra-short segment Hirschsprung disease. In centers with expertise, ARM can be the initial diagnostic test, although it does not identify the extent of disease. The gold standard for diagnosis is the demonstration of complete absence of ganglion cells in the Meissner and the Auerbach plexus on a biopsy specimen of intestinal mucosa and submucosa. Treatment generally is surgical resection of the aganglionic segment.

Coagulopathy — Several types of coagulopathies can present during the newborn period. Most present with other bleeding symptoms, such as a large cephalohematoma after vaginal delivery, oozing from the umbilical stump, prolonged bleeding after circumcision or blood sampling, or intracranial hemorrhage in a term infant [14]. Occasionally, these coagulopathies come to medical attention because of LGIB, although this rarely occurs during the neonatal period.

- Vitamin K deficient bleeding (previously known as hemorrhagic disease of the newborn) – This disorder may occur in infants who do not receive vitamin K administration at birth; the risk is increased by maternal ingestion of warfarin, or certain antibiotics and anticonvulsants. The disorder presents with cutaneous, gastrointestinal, and intracranial bleeding in neonates, typically developing within the first week of life. (see "Overview of vitamin K", section on 'Vitamin K deficient bleeding in newborns and young infants'). Rarely, neonates with underlying disorders of fat absorption (eg, cystic fibrosis, biliary atresia, alpha-1-antitrypsin deficiency) will present with GI bleeding related to vitamin K deficiency, although this typically presents after the newborn period.

- Hemophilia – About 25 percent of children with hemophilia present with a bleeding episode, and the remaining 75 percent are identified because of a family history of the disorder. Less than 5 percent of individuals present with bleeding in the neonatal period [14,15]. Hemophilia should be suspected in any male infant with an isolated prolongation of activated partial thromboplastin time (aPTT). (See "Clinical manifestations and diagnosis of hemophilia", section on 'Initial presentation'.)

- Von Willebrand disease – Von Willebrand disease (VWD) is a common disorder, but most individuals are asymptomatic. Presentation during the neonatal period or infancy is rare. (See "Clinical presentation and diagnosis of von Willebrand disease".)

- A variety of other congenital and acquired disorders of hemostasis present with bleeding symptoms. In most cases, there is evidence of bleeding from non-GI sources (eg, petechiae, mucocutaneous bleeding, or bruising) to suggest a disorder of coagulation. These findings warrant evaluation for a bleeding disorder. (See "Approach to the child with bleeding symptoms".)

Infants and toddlers — Causes of bleeding in infants and toddlers (one month to two years) include:

- Anal fissures (especially around the introduction of solid food or cow's milk)

- Milk or soy protein-induced colitis (allergic colitis)

- Intussusception

- Infectious colitis
- Meckel's diverticulum
- Lymphonodular hyperplasia
- Gastrointestinal duplication cyst
- Coagulopathy
- Eosinophilic gastrointestinal disease (EGID)
- Infantile and very early onset inflammatory bowel disease (VEO-IBD)

Anal fissures — Anal fissures are discussed above. (See 'Anal fissures' above.)

Milk- or soy-induced colitis — Milk- or soy protein-induced colitis is an inflammatory reaction caused by ingestion of cow's milk or soy proteins, and is a common cause of bloody stools in infants. It occurs almost exclusively in infants and usually resolves within 6 to 18 months of age. It can occur in infants who are formula-fed, or less commonly in breast-fed infants because of cow's milk in the mother's diet. Affected infants have loose stools, often with occult or gross blood, but are otherwise healthy. Although cow's milk is the most common trigger in Western populations, up to 25 percent of patients with cow's milk protein intolerance will have a cross-reaction to soy protein, and a few infants are sensitive to other food proteins. Thus, the general term for these disorders is "food protein-induced colitis." Treatment involves meticulous elimination of the causative protein from the mother's diet if the infant is breastfed, or the use of a casein-hydrolysate formula. About 10 percent of infants are sensitive to the casein-hydrolysate formula and require an amino acid-based formula. The intolerance usually resolves by 18 months of age, at which time an unrestricted diet can be resumed. (See "Food protein-induced proctocolitis of infancy".)

Food protein-induced enteropathy and enterocolitis syndrome (FPIES) are separate but related disorders, in which vomiting rather than rectal bleeding is the prominent feature, and infants are generally ill-appearing and more symptomatic. (See "Food protein-induced proctocolitis of infancy" and "Food protein-induced enterocolitis syndrome (FPIES)".)

Intussusception — Intussusception is the most common cause of intestinal obstruction in infants between 6 and 36 months of age. Approximately 60 percent of affected children are younger than one year old, and 80 percent are younger than two years. In this age group, intussusception usually is idiopathic and occurs in the ileocecal region, in contrast to older children in whom a polyp or Meckel's diverticulum or other lesions often serve as a lead point. The clinical presentation, diagnosis, and management of intussusception are discussed in detail in a separate topic review. (See "Intussusception in children".)

Patients may awaken from sleep with severe abdominal pain, which causes them to be irritable and draw up their legs. They vomit and may pass a stool and improve temporarily before the cycle repeats. Eventually, patients become apathetic and pale, and may pass a bloody, mucoid stool. A sausage-shaped mass in the distribution of the colon, typically in the area of the transverse colon, may occasionally be palpable on abdominal examination. The stool contains gross or occult blood in most but not all cases, and sometimes has the appearance of "currant jelly."

Ultrasonography is the method of choice to detect intussusception in most institutions. The diagnosis can also be established with an air or water-soluble contrast enema, which also can treat ("reduce") the intussusception in 75 to 90 percent of children in whom a lead point is not present. The choice of procedure varies with the experience of the radiologist.

Meckel's diverticulum — Meckel's diverticulum results from incomplete obliteration of the omphalomesenteric duct. It is usually asymptomatic, but may cause painless rectal bleeding, which may be chronic and insidious, or acute and massive [16]. The bleeding is often caused by mucosal ulceration of

adjacent small bowel tissue due to production of acid by ectopic gastric tissue within the diverticulum. Other complications associated with a Meckel's diverticulum are obstruction, perforation, diverticulitis, and intussusception. Sixty percent of pediatric patients having complications from a Meckel's diverticulum are younger than two years of age. (See "Meckel's diverticulum".)

The "rule of twos" is used to describe the epidemiology of Meckel's diverticulum [17]: it occurs in two percent of the population with a male-to-female ratio of 2:1, is found within two feet of the ileocecal valve, involves two types of tissue (gastric and intestinal epithelium), and is two inches long. Approximately 2 percent of individuals with a Meckel's diverticulum develop a complication over the course of their lives. There is probably no familial predisposition for Meckel's diverticulum, although a few cases of occurrence within the same family have been reported [18].

The diagnosis is made by a Meckel scan. The scan consists of the intravenous administration of 99m technetium pertechnetate, which has an affinity for gastric mucosa, followed by scintigraphy to identify areas of ectopic gastric mucosa. The accuracy of a Meckel scan is improved by administration of histamine type 2 receptor antagonists (H2 blockers) for 24 to 48 hours before the test. Any Meckel's diverticulum that is symptomatic should be resected. An asymptomatic Meckel's diverticulum discovered incidentally at laparotomy usually also is resected in children. However, whether such asymptomatic lesions should be resected in adults is controversial because of the low incidence of complications in older patients who have never had symptoms [19].

Lymphonodular hyperplasia — Lymphonodular hyperplasia is a common finding in infants and young children who undergo endoscopy or radiographic studies of the intestinal tract [20,21]. The etiology is unknown. Many consider it to be a normal finding [22,23], whereas others believe it to be an immunologic response to a variety of stimulants [21,24,25]. It occurs frequently in children with food protein induced colitis, in whom it may be an abnormal finding at colonoscopy [26]. (See 'Milk- or soy-induced colitis' above and "Food protein-induced proctocolitis of infancy".)

Because lymphonodular hyperplasia disrupts the normal mucosa, it may potentially lead to mucosal thinning and predisposes to ulceration, which may cause hematochezia [20]. Blood loss is usually minimal and painless but is present in multiple stools [27]. The use of stool softeners may help to reduce blood loss and minimize parental anxiety. Lymphonodular hyperplasia resolves spontaneously over time and is an unlikely source of bleeding in older children [27].

Gastrointestinal duplication cyst — Gastrointestinal duplication cysts can be found at any level of the GI tract and frequently do not communicate with the bowel lumen. Gastric mucosa (present in approximately 50 percent of duplication cysts) can ulcerate, perforate, and form fistulas. Formation of a gastric duplication-colonic fistula is unusual but can result in a lower gastrointestinal bleed [28]. In addition, a duplication cyst that communicates with the intestine can result in bleeding into the GI tract. Gastrointestinal duplication cysts tend to present in infancy if they are symptomatic, but they may present in any age group, and often remain asymptomatic. (See "Endoscopic ultrasound for the characterization of subepithelial lesions of the upper gastrointestinal tract", section on 'Duplication cysts'.)

Infantile and very early onset inflammatory bowel disease — Rarely, inflammatory bowel disease (IBD) presents before six years of age. Although definitions vary, this is usually known as very early onset IBD (VEO-IBD), and VEO-IBD that presents before two years of age is sometimes termed infantile IBD [29].

VEO-IBD is phenotypically and genetically distinct from IBD presenting in older patients (table 3). The disease tends to be severe, with more rapid progression and poor responsiveness to most conventional therapies. The distinct phenotype and early age of onset suggests a pronounced genetic susceptibility and dysregulated immune response. Indeed, approximately 25 percent of patients have an underlying immunodeficiency, which can affect therapy. Whole exome sequencing has identified several novel gene variants that are associated with VEO-IBD, including monogenic forms caused by mutations in single genes (eg, *EPCAM, IL10, IL10RA, IL10RB, FOXP3, LRBA, SKIV2L, TTC37, TTC7A*), which often present during

infancy [30-32]. Some of these mutations affect hematopoietic cells (eg, *IL10, IL10RA, XIAP* and *FOXP3*), and affected patients are candidates for hematopoietic stem cell transplantation. As an example, mutations in *IL10RA* can present with severe intractable enterocolitis, perianal disease, folliculitis and arthritis [32]. An approach to the evaluation of patients with VEO-IBD is outlined in the algorithm (algorithm 1). (See "Clinical presentation and diagnosis of inflammatory bowel disease in children", section on 'Early onset IBD'.)

Preschool period — The following disorders that occur in infancy are also important causes of rectal bleeding during the preschool period (ages two to five years):

- Anal fissures (especially around toilet training) (see 'Anal fissures' above)
- Intussusception (see 'Intussusception' above)
- Meckel's diverticulum (see 'Meckel's diverticulum' above)

In addition, the following disorders warrant consideration in the preschool age group, as discussed below:

- Infectious colitis
- Hemolytic-uremic syndrome (HUS)
- Immunoglobulin A vasculitis (IgAV; Henoch-Schönlein purpura [HSP])
- Juvenile polyps
- Very early onset inflammatory bowel disease (VEO-IBD)
- Solitary rectal ulcer syndrome (SRUS)

Infectious colitis — A number of pathogens can cause LGIB in preschool children: *Salmonella, Shigella, Campylobacter, E. coli 0157:H7, and Clostridium difficile* (C. difficile) are the most common*; Yersinia* infection typically causes non-bloody diarrhea. Other causes of colitis include parasites such as *Entamoeba histolytica* and viruses such as adenovirus, Cytomegalovirus (CMV) and Herpes simplex virus (HSV). Potential pathogens in immunocompromised children include *Mycobacteria* and *Aeromonas hydrophila,* although *Aeromonas* infection typically presents with non-bloody diarrhea but can be associated with bloody diarrhea in some cases. *Neisseria gonorrhea*, *Chlamydia trachomatis* and *Plesiomonas shigelloides* also occasionally produce bloody stools. Infection should be considered in children presenting with bleeding accompanied by dysenteric symptoms (eg, fever, abdominal pain, tenesmus, small volume bloody stools). (See appropriate topic reviews).

The diagnosis of an infectious etiology is made by isolating the organism from the stool or blood. Other ancillary tests such as occult blood, fecal leukocytes, fecal calprotectin, or fecal lactoferrin are nonspecific. In one study, fecal lactoferrin appeared to be the most accurate index test of the three assays for predicting inflammatory diarrhea [33].

- **Salmonellosis** – *Salmonella* gastroenteritis occurs most commonly in children younger than five years of age and has its highest incidence in the first year, during which it is accompanied by bacteremia in approximately 5 percent of patients [34]. A larger inoculum is needed to produce infection compared with *Shigella*. Blood is a less common finding in the stool with *Salmonella* infection compared with *Shigella*. The white blood cell (WBC) count is variable but may have a left shift. A stool smear stained with methylene blue often shows "sheets" of mononuclear cells. This helps to distinguish *Salmonella* infection from other organisms (especially *Shigella*, in which the stool tends to have "sheets" of polymorphonuclear cells [PMNs]), although this is not diagnostic and stool culture remains the best method to establish the diagnosis. Although of unproven benefit, antimicrobial therapy is indicated for patients younger than three months and for other children at increased risk for invasive disease, including those with malignancies, hemoglobinopathies, chronic gastrointestinal tract disease, and immunocompromised states [35]. Antimicrobial therapy is not indicated for patients with uncomplicated

mild to moderate gastroenteritis caused by nontyphoidal *Salmonella* species because therapy does not shorten the duration of the disease and can prolong the duration of excretion of *Salmonella* organisms [35]. (See "Nontyphoidal Salmonella: Gastrointestinal infection and carriage".)

- **Shigellosis** – Shigellosis occurs most commonly in children between one and four years of age. Patients typically present with fever, abdominal pain, and watery diarrhea that becomes bloody. The total WBC count may be normal or markedly elevated, but the differential may show a greater count of band forms as compared with PMNs. Untreated, the disease typically lasts for 7 to 10 days, but organism carriage can continue for as long as three weeks. Potential complications of shigellosis are intestinal perforation, toxic megacolon, dehydration, sepsis, hyponatremia and hypoglycemia, pneumonia, HUS, and seizures or encephalopathy [36]. The impact of antibiotic therapy on reducing the occurrence and severity of these complications is unclear. (See "Shigella infection: Clinical manifestations and diagnosis", section on 'Clinical manifestations'.)

  Antibiotic therapy eradicates the organism from the gastrointestinal tract, reduces the intensity and duration of the illness, and limits the likelihood of spread to other contacts [37]. (See "Shigella infection: Treatment and prevention in children".)

- **Campylobacter** – *C. jejuni* usually produces dysentery (bloody diarrhea) in patients up to eight years of age. It is isolated with equal frequency to *Salmonella* and *Shigella*. Most cases will resolve spontaneously within one week [38]. Treatment with appropriate antibiotics modestly reduces the duration of intestinal symptoms and is suggested for patients with severe disease or risk for severe disease. (See "Clinical manifestations, diagnosis, and treatment of Campylobacter infection".)

- **Escherichia coli** – *E. coli* is an important cause of food-borne diarrheal illness and can occur in clusters of cases. The most serious complication is the development of HUS after an infection with the enterohemorrhagic *E. coli* O157:H7 or other Shiga toxin-producing E. coli. HUS is characterized by a "triad" of microangiopathic hemolytic anemia, thrombocytopenia, and acute renal injury, which may eventually lead to life-threatening kidney failure. Early antibiotic administration may increase the risk of developing HUS, perhaps because of enhanced toxin release as the bacteria are killed. This possibility was demonstrated in a prospective study of 71 children younger than the age of 10 years with *E. coli* O157:H7 isolated from stool; those receiving antibiotics were more likely to develop HUS (56 versus 8 percent) [39]. (See "Pathogenic Escherichia coli" and 'Hemolytic-uremic syndrome' below.)

- **Yersinia** – *Yersinia* gastroenteritis is associated with bloody stools in up to 25 percent of patients. In addition to occasional bloody diarrhea, it more typically causes fever, non-bloody diarrhea, right lower quadrant pain due to terminal ileitis and mesenteric adenitis, which mimics appendicitis and Crohn disease. Most cases resolve spontaneously within two weeks. The benefit of antimicrobial treatment remains unproven and treatment is generally not recommended except for patients with severe disease or an underlying comorbid illness. (See "Clinical manifestations and diagnosis of Yersinia infections".)

- **Clostridium difficile** – *C. difficile* enteritis usually occurs after exposure to antibiotics, especially ampicillin, clindamycin, and cephalosporins. However, community-associated infection with a highly toxigenic strain of *C. difficile* has been reported in otherwise healthy children who had minimal or no exposure to antibiotics, and *C. difficile* infection can be the initial manifestation of inflammatory bowel disease (IBD). In more recent years, increased incidence and severity of this infection has been attributed largely to the emergence of a new virulent strain of *C. difficile* (BI/NAP1/027), which may cause severe life-threatening illness [40]. (See "Clostridium difficile infection in children: Microbiology, pathogenesis, and epidemiology", section on 'Antibiotic exposure'.)

  The clinical disease varies from mild diarrhea to a dysenteric syndrome. Diagnosis is made most reliably by the detection of *C. difficile* polymerase chain reaction (PCR), but also may be made by the detection of *C. difficile* toxin by enzyme-linked immunosorbent assay in the stool. Stool culture for *C. difficile* does not differentiate toxin- from non-toxin-producing strains, and up to 50 percent of healthy

neonates and infants can be colonized with this organism. The colonization rate decreases to less than 5 percent in children older than the age of two years. (See "Clostridium difficile infection in children: Clinical features and diagnosis".)

If possible, the antibiotics that predisposed to the *C. difficile* infection should be stopped or changed. Treatment is indicated for children with severe or persistent disease; usually with oral or if necessary intravenous metronidazole, or oral vancomycin. Fecal microbiota transplantation is sometimes used for patients with recurrent disease despite antibiotic therapy [41,42]. (See "Clostridium difficile infection in children: Treatment and outcome" and "Fecal microbiota transplantation in the treatment of recurrent Clostridium difficile infection".)

- **Other organisms** – Occult or overt bleeding can occur as a result of infection with *N. gonorrhoeae*, *C. trachomatis*, and HSV. Sexual abuse should be considered in children when these organisms are recovered. (See "Evaluation of sexual abuse in children and adolescents".)

  In immunocompromised patients, *Mycobacterium tuberculosis* can affect any part of the gastrointestinal tract, but most commonly affects the cecum and ileocecal valve. Tuberculous enteritis can present with fever, weight loss, bloody diarrhea, and abdominal pain. Endoscopically and histologically, tuberculous enteritis can mimic Crohn disease, but if tuberculosis is suspected, PCR testing of colonic biopsy tissue can confirm the diagnosis. CMV can also cause chronic colitis or enteritis in immunosuppressed individuals [43]. (See "Abdominal tuberculosis".)

Hemolytic-uremic syndrome — HUS is characterized by the simultaneous occurrence of microangiopathic hemolytic anemia, thrombocytopenia, and acute renal injury. The highest rates are in children under the age of five years. Most cases are associated with a prodromal infection with an enteropathogen producing a shiga-like toxin, such as *E. coli* 0157:H7, in which case diarrhea is a prominent feature and is frequently bloody. The HUS typically develops 5 to 10 days after the onset of the diarrhea. Thus, HUS can be considered a complication of infectious colitis, rather than an independent cause of colitis. (See "Clinical manifestations and diagnosis of Shiga toxin-producing Escherichia coli (STEC) hemolytic uremic syndrome (HUS) in children".)

IgA vasculitis (Henoch-Schönlein purpura) — Immunoglobulin A vasculitis (IgAV; Henoch-Schönlein purpura [HSP]) is a systemic vasculitis of unclear etiology characterized by palpable cutaneous purpura (picture 1), abdominal pain, and arthralgias. It is primarily a childhood disease that occurs between the ages of 3 and 15 years. Up to 50 percent of patients develop gross or occult gastrointestinal bleeding, and up to 50 percent develop renal disease. The abdominal pain in IgAV (HSP) is due to the presence of purpuric lesions within the gastrointestinal tract, or may be caused by intussusception, in which the involved bowel serves as a lead point. (See "IgA vasculitis (Henoch-Schönlein purpura): Clinical manifestations and diagnosis".)

Juvenile polyps — Juvenile polyps are benign hamartomas, which typically occur between the ages of 2 and 10 years, with a peak at 3 to 4 years [44]. Patients usually present with painless rectal bleeding, with or without mucus [45]; a few may have lower abdominal pain from traction on the polyp. Juvenile polyps tend to be pedunculated rather than sessile, and may autoamputate, which results in significant bleeding. On occasion, polyps in the rectum present as prolapsed tissue. Polyps usually bleed after injury by fecal passage and usually result in bright red blood on the outside of the stool. If the polyp is located proximally, the blood will be darker and found in the core of the stool. About 60 to 80 percent of these polyps are in the rectosigmoid, and some of these can be palpated on rectal examination [44,46]. In children and adolescents, about 85 percent or more of polyps are juvenile (hamartomas), 10 percent or less are adenomas, and 3 percent are hyperplastic [47]. Adenomatous polyps occur more frequently in older children and adolescents or in the setting of a polyposis syndrome, and current guidelines for evaluation, management, and follow-up should be followed [48].

Colonoscopy is the best way to diagnose polyps and permits their immediate, painless removal [49]. If possible and safe, all polyps should be removed and evaluated by microscopy to confirm that they are juvenile hamartomas rather than adenomas. More than 80 percent of children have only one or two juvenile polyps [44]. Although older series suggested that recurrence in such children was rare [48], a subsequent study reports recurrence in 17 percent of patients with a single juvenile polyp [50]. Patients with three to five or more juvenile polyps (especially those with ≥10 polyps) and those with a family history of polyps may have familial juvenile polyposis or juvenile polyposis syndrome. These patients should undergo colonoscopy and biopsy every two to three years as surveillance for colorectal neoplasia. Although the polyps themselves are not premalignant, the syndrome is associated with a higher lifetime risk of colorectal malignancy [48]. (See "Juvenile polyposis syndrome", section on 'Cancer screening'.)

Solitary rectal ulcer syndrome — Solitary rectal ulcer syndrome (SRUS) is a benign but potentially chronic ulcerative disease of the rectum that is infrequent in childhood. It tends to present with bleeding, passage of mucus, straining during defecation, and a sense of incomplete evacuation [51,52]. Treatment of associated constipation and strategies to avoid dysfunctional stooling or excessive straining may help alleviate this condition. (See "Solitary rectal ulcer syndrome".)

School-aged children and adolescents — The spectrum of disorders in school-aged children with lower gastrointestinal bleeding is similar to that of the preschool age group, although HUS and intussusception are less common:

- Anal fissures (especially around school entry) (see 'Anal fissures' above)
- IgAV (HSP) (see 'IgA vasculitis (Henoch-Schönlein purpura)' above)
- Meckel's diverticulum (see 'Meckel's diverticulum' above)
- Infectious colitis (see 'Infectious colitis' above)
- Juvenile polyps (see 'Juvenile polyps' above)
- Hemorrhoids (primarily in older adolescents)
- Inflammatory bowel disease (see 'Inflammatory bowel disease' below)
- Solitary rectal ulcer syndrome (SRUS) (see 'Solitary rectal ulcer syndrome' above)

Inflammatory bowel disease — Inflammatory bowel disease may present in preschool children and even in infancy (see 'Infantile and very early onset inflammatory bowel disease' above), but is more common in school-aged children and adolescents.

IBD is comprised of two major disorders: ulcerative colitis (UC), which affects only the colon, and Crohn disease (CD), which can involve any portion of the gastrointestinal tract. The most common presenting symptoms of IBD are abdominal pain, fever, and diarrhea (with or without blood), occurring in about 80 percent of patients with CD; about 20 percent of patients with CD and about 95 percent of patients with UC will have visible rectal bleeding. The peak incidence of IBD is in late adolescence and early adulthood, but a significant number of children present prior to adolescence and children may present even before five years of age. The diagnosis usually is suspected by the chronicity or severity of symptoms and weight loss or growth failure (especially in those with CD). Many patients have iron deficiency and/or an elevated erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP), hypoalbuminemia, and elevated fecal calprotectin. Some patients with IBD may have extraintestinal symptoms, including anorexia, arthralgia, and erythema nodosum. The diagnosis is confirmed with imaging of the small bowel, upper endoscopy, colonoscopy, and biopsy. (See "Clinical presentation and diagnosis of inflammatory bowel disease in children".)

Rare causes of lower gastrointestinal bleeding — Rare causes of LGIB in children and adolescents include:

- Vascular malformations – eg, associated with hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu syndrome), blue rubber bleb nevus syndrome [53,54]. (See "Clinical manifestations and diagnosis of hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu syndrome)".)

- Adenomatous polyps – Patients with adenomatous polyps should be further evaluated for familial adenomatous polyposis or one of its variants because of a high risk of developing colorectal cancer. (See "Clinical manifestations and diagnosis of familial adenomatous polyposis" and "Familial adenomatous polyposis: Screening and management of patients and families".)

- Gastrointestinal stromal tumors (GIST) – GIST are often associated with Carney's syndrome (GIST, pulmonary chondromas, paraganglioma, adrenal cortical adenoma, and esophageal leiomyoma), and are located most frequently in the stomach, especially in the antrum in pediatric patients. GIST have been reported more frequently over the course of a patient's lifetime in patients with neurofibromatosis type 1 (NF 1), but in this condition they tend to have a more benign course and can be located in the small intestine rather than in the stomach. GIST in pediatric patients are frequently associated with metastatic disease at a higher frequency than in adults [55]. (See "Epidemiology, classification, clinical presentation, prognostic features, and diagnostic work-up of gastrointestinal stromal tumors (GIST)", section on 'Pediatric GIST'.)

- Typhlitis – Typhlitis is an enterocolitis of the ileocecal region that occurs in patients with neutropenia, most commonly in children with hematologic malignancies. It presents with fever and abdominal pain, and sometimes with bloody diarrhea. (See "Neutropenic enterocolitis (typhlitis)".)

- Malignancies – Malignancies of the GI tract occur infrequently in pediatric aged patients. They may present with rectal bleeding with or without other associated symptoms such as weight loss, abdominal pain, and symptoms of obstruction. Malignancies may be primary (eg, GIST discussed above, lymphoma, adenocarcinoma) or represent metastatic disease from a variety of primary sites. Patients with polyposis syndromes and IBD among other conditions are at higher risk of small bowel or colonic malignancy depending on the underlying condition and current screening guidelines should be followed.

DIAGNOSTIC APPROACH — The diagnostic approach to a patient with suspected lower gastrointestinal bleeding (LGIB) is structured around the following questions (algorithm 2):

- **Is the child hemodynamically stable?** – Children with evidence of hemodynamic instability (tachycardia, orthostasis, hypotension, poor peripheral perfusion) and/or altered sensorium should be managed emergently for shock. Sepsis should be considered in those with fever or conditions that predispose to infection. Laboratory tests for hemodynamically unstable patients include a complete blood count (CBC), routine chemistries, coagulation studies, and blood type and crossmatch (in case transfusion will be needed). (See "Hypovolemic shock in children: Initial evaluation and management" and "Septic shock in children: Rapid recognition and initial resuscitation (first hour)".)

- **Is it blood?** – In most cases, the clinician should examine the stool directly and test it for blood. This is because several foods and medicines may give stool a bloody appearance (table 1). (See 'Testing for blood in the stool' below.)

- **Is the blood from the lower GI tract?** – Red blood found in a child's stool is most often from the anus or lower gastrointestinal tract, but occasionally has an upper gastrointestinal source. The appearance of the bloody stool helps to distinguish between these possibilities:

    - Hematochezia (the passage of bright red blood per rectum) usually suggests LGIB, and is typically from the colon or anus. Rarely, hematochezia can be caused by upper gastrointestinal bleeding

(UGIB), especially in an infant or other individual with rapid intestinal transit time, or because of a massive UGIB in an older individual. Thus, the possibility of UGIB should be considered in an individual with hematochezia and hemodynamic compromise, or in a child with risk factors for UGIB, such as underlying liver disease.

- Melena (black, tarry stools) usually suggests UGIB. It also may be caused by blood from the nose that is swallowed, or bleeding from the proximal small bowel (see "Approach to upper gastrointestinal bleeding in children"). Black-appearing stools also may be caused by certain medications (eg, bismuth or iron), or foods (large amounts of dark green leafy vegetables). These black stools can be distinguished from melena by testing the stool for blood. (See 'Testing for blood in the stool' below.)

- **What are the most likely causes of the LGIB in this patient?** The diagnostic possibilities depend on the patient's age and individual characteristics, assessed by a focused history and physical examination, as summarized in the table (table 2). (See 'History' below and 'Physical examination' below.)

This information permits selection of specific tests to diagnose or exclude the likely disorders, which are organized above by the typical age group in which each tends to appear. (See 'Causes of bleeding' above.)

Testing for blood in the stool — In most cases of suspected gastrointestinal bleeding, the clinician should examine the stool directly and test it for blood. This is because several foods and medicines may give stool a bloody appearance that may be misinterpreted by the child or their parent(s) (table 1) [56]. Red-colored stools resembling hematochezia may be caused by foods with red dyes (eg, Kool-Aid or fruit punch), beets, red licorice, or rifampin. Black-colored stools resembling melena may be caused by ingestion of bismuth, activated charcoal, or iron, and occasionally by consumption of large quantities of chocolate, blueberries, or dark green foods. Testing is particularly important in children with equivocal symptoms, such as a well-appearing child with small amounts of red material in the stool.

The most common assays can be performed at the bedside and are guaiac-based. This test identifies hemoglobin by the presence of a peroxidase reaction that turns guaiac-impregnated paper blue [57]. Occasionally, false-positive results may occur if the patient has ingested rare red meat or peroxidase-containing vegetables (such as turnips, horseradish, broccoli, cauliflower, and cantaloupe). Whether iron supplements cause false-positive results is controversial [10]. False negative results can be obtained if the patient is ingesting large doses of ascorbic acid (vitamin C) or if intestinal bacteria have degraded the hemoglobin to porphyrin.

Patients with positive guaiac tests that are thought to be possible false-positives can be further evaluated with non-guaiac-based tests, which are based on measurement of fecal hemoglobin or fecal alpha-1 antitrypsin. The sensitivity of the test for fecal hemoglobin is limited because of degradation of hemoglobin by fecal bacteria, and the overall sensitivity and specificity are similar to the guaiac test [10]. Fecal alpha-1 antitrypsin is used primarily to diagnose enteric protein loss, but it is also elevated in patients with gastrointestinal blood loss (upper or lower), in whom it has a sensitivity and specificity of 88 and 90 percent, compared with 68 and 73 percent, respectively, of the guaiac test [10]. The fecal alpha-1 antitrypsin test can be used in patients with recurrent red stools that are guaiac-negative or in those with a possible false-positive guaiac test result.

History — If the presenting complaint is rectal bleeding, details about the symptom should be sought:

- Duration and amount of bleeding – The patient or family can estimate the amount of blood, but this is often inaccurate. Asking whether there is enough blood to color the toilet water, if so how red, and if clots are present, can help to convey the amount of bleeding observed. Note that very small amounts of blood, even a few drops, will color the toilet water.

- Color of the blood – Bright red blood is most consistent with a lower gastrointestinal source, hemorrhoid, or anal fissure. However, UGIB should be considered in infants or any child with evidence of hemodynamic instability or with risk factors for UGI bleeding. (See 'Diagnostic approach' above.)

- Consistency of accompanying stool – Accompanying diarrhea suggests the possibility of colitis (eg, food protein-induced colitis in an infant, hemolytic-uremic syndrome (HUS) or immunoglobulin A vasculitis (IgAV; Henoch-Schönlein purpura [HSP]) in a young child, inflammatory bowel disease (IBD) in older children and adolescents, or infectious colitis in all age groups). Acute or intermittent constipation suggests the possibility of anal fissures or solitary rectal ulcer syndrome (SRUS). The degree of constipation is often under-estimated by children and their caretakers, so it is valuable to seek additional details about constipation-related symptoms such as pain while defecating, time spent on the toilet, withholding behavior, size of stools, and whether they plug the toilet. (See "Constipation in infants and children: Evaluation".)

- Distal bleeding – Several characteristics suggest that the blood is most likely from an anorectal source such as an anal fissure, hemorrhoid, or polyp. These include blood that is primarily on the outside of a formed stool, blood that is mostly seen on the toilet paper rather than in the stool, or that drips into the toilet after the bowel movement (sometimes called "terminal bleeding").

A history of associated symptoms should be specifically sought, even if the child is generally well-appearing. The following symptoms provide clues to the underlying diagnosis (table 2):

- Well infant or child – Suggests the following possibilities:
    - Anal fissure (all age groups). Typical features are small recurrent rectal bleeding, often with constipation and/or anal symptoms.
    - Milk- or soy protein induced colitis (infants). Typical features are small to moderate bleeding, often with mucous in stool, with or without abdominal pain and diarrhea.
    - Juvenile polyps (preschool aged children and older). Typical features are normal appearing stool, often with recurrent bleeding.
    - Meckel's diverticulum (especially in children younger than two years). Consider if above diagnoses are excluded, particularly if bleeding is large volume bleeding and/or recurrent.
- Diarrhea with abdominal pain – Suggests colitis (milk- or soy-induced in an infant; infectious colitis or IBD in older children), or HUS.
- Fever – Suggests infectious colitis, possibly Hirschsprung-associated enterocolitis in a young infant, or occasionally IBD.
- Weight loss and fatigue, delayed puberty – Suggest underlying systemic disease, especially IBD.
- History of abnormal non-gastrointestinal bleeding – Suggests an underlying coagulopathy such as hemophilia or HUS; or IgAV (HSP) if purpura is present.
- History of epistaxis (nosebleeds), especially if large volume or recent – If a large amount of blood is swallowed during a nosebleed, patients may present with melena, or occasionally with hematochezia (especially in infants or other individuals with rapid transit time).
- Recent use of nonsteroidal antiinflammatory drugs (NSAIDs) or any other medications – NSAIDs can cause UGIB due to peptic ulcers, or exacerbate LGIB due to anti-platelet effects.
- Underlying disease – Many underlying systemic disorders alter the differential diagnosis of LGIB. For example, a child with an underlying immunodeficiency is particularly at risk for infectious colitis or

neutropenic enterocolitis (typhlitis). A child with underlying liver disease may develop variceal bleeding (causing UGIB or LGIB) or coagulopathy.

Physical examination — The first step is to evaluate the child's hemodynamic stability, by measuring vital signs and assessing for signs of shock, including tachycardia, hypotension or orthostasis, poor capillary refill and altered sensorium. Sepsis should be considered in those with fever or conditions that predispose to infection. Bowel obstruction is suggested by marked abdominal pain and distension and/or bilious or nonbilious vomiting. Children with evidence of shock, sepsis, or abdominal obstruction should be treated promptly with fluid resuscitation and other measures, as described separately. (See "Hypovolemic shock in children: Initial evaluation and management" and "Septic shock in children: Rapid recognition and initial resuscitation (first hour)" and "Emergency evaluation of the child with acute abdominal pain", section on 'Signs of obstruction or peritoneal irritation'.)

For stable patients, the physical examination should investigate potential sites of bleeding. In particular, the anus should be carefully inspected for fissures in all age groups by very gently spreading each fold of the anus. Other features to note in the perianal area include large skin tags or fistulas, both of which suggest the possibility of IBD (Crohn disease [CD]) (see 'Inflammatory bowel disease' above). A rectal examination is important to evaluate for rectal polyps (but does not rule out this condition) and to obtain stool for guaiac testing. In young infants or children with a history of constipation (especially in those without associated encopresis), the possibility of Hirschsprung disease is suggested by an explosive expulsion of gas and stool after the digital rectal examination (squirt sign or blast sign). (See 'Hirschsprung disease' above.)

The nasopharynx and oropharynx should be inspected. Recent nosebleeds can mimic gastrointestinal bleeding. CD is often associated with aphthous ulcers in the mouth. (See "Evaluation of epistaxis in children" and 'Inflammatory bowel disease' above.)

The skin should be inspected for bruising, petechiae or other rashes, which suggest abnormal hemostasis, as may be seen in HUS. Palpable purpura suggests a vasculitis, such as IgAV (HSP). The presence of cutaneous hemangiomas (especially five or more) in an infant suggests the possibility of gastrointestinal hemangiomatosis. However, up to 50 percent of infants with visceral hemangiomas do not have cutaneous hemangiomas [58]. Jaundice suggests hepatic failure or biliary obstruction, especially if significant. The abdominal examination should evaluate for hepatomegaly, splenomegaly, or other signs of portal hypertension (ascites, prominent superficial veins), masses, tenderness, abnormal bowel sounds, and for evidence of underlying peritonitis. (See "Infantile hemangiomas: Evaluation and diagnosis".)

Laboratory studies and imaging — Infants and children presenting with significant rectal bleeding should be evaluated for anemia. Patients with identifiable sources of minor bleeding such as a fissure or viral gastroenteritis may not require laboratory testing.

More extensive laboratory testing is appropriate for those who are not well-appearing, or who have symptoms or signs suggestive of a particular disorder. As an example, for children with suspected colitis (infectious colitis or IBD), the evaluation includes an erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP), albumin and complete blood count (CBC), and stool testing for Clostridium difficile (C. difficile), enteric pathogens, ova and parasites (O&P), and possibly fecal lactoferrin and fecal calprotectin. (see 'Infectious colitis' above and 'Inflammatory bowel disease' above and "Clinical presentation and diagnosis of inflammatory bowel disease in children"). Children with evidence of bleeding from non-GI sources (eg, petechiae or bruising) should be evaluated for a bleeding disorder (see "Approach to the child with bleeding symptoms"). Patients with concern for HUS should also undergo serial evaluation of CBC and renal function. (See 'Hemolytic-uremic syndrome' above.)

Infants and children with an abnormal abdominal examination (distension, tenderness, or abnormal bowel sounds) or who are ill-appearing should be evaluated with an abdominal plain film, at a minimum. Patients with suspected intussusception should be evaluated with ultrasonography (and/or with an air or water-soluble contrast enema), depending on institutional protocols. (See 'Intussusception' above.)

Selection of other tests depends on the age group of the patient and the suspected disorder, and may include laboratory tests, imaging studies (eg, tagged red blood cell [RBC] scan, angiography, or computed tomography [CT] angiography), and endoscopic procedures (colonoscopy, upper endoscopy or capsule endoscopy), as discussed in the summaries above or the linked topic reviews.

SOCIETY GUIDELINE LINKS — Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Hemophilia, von Willebrand disease, and other coagulation disorders" and "Society guideline links: Gastrointestinal bleeding in children".)

INFORMATION FOR PATIENTS — UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Meckel's diverticulum (The Basics)" and "Patient education: Anal fissure (The Basics)" and "Patient education: Ulcerative colitis in children (The Basics)" and "Patient education: Crohn disease in children (The Basics)")

- Beyond the Basics topics (see "Patient education: Bloody stools in children (Beyond the Basics)" and "Patient education: Anal fissure (Beyond the Basics)")

SUMMARY AND RECOMMENDATIONS

- Lower gastrointestinal bleeding (LGIB) typically presents with bright red blood per rectum (hematochezia). In most cases, the clinician should examine the stool directly and test it for blood. This is because several foods and medicines may give stool a bloody appearance that may be misinterpreted by the child's parents (table 1). Testing the stool for blood is particularly important in children with equivocal symptoms, such as a well-appearing child with small amounts of red material in the stool. (See 'Testing for blood in the stool' above.)

- The diagnostic possibilities depend on the patient's age group and are then narrowed using a focused history and physical examination (algorithm 2 and table 2). A complete blood count (CBC) or hemoglobin should be measured in patients who are unwell or who have more than minimal bleeding. Other laboratory tests or imaging are added to diagnose or exclude specific causes. (See 'Diagnostic approach' above.)

- Anal fissures are a common cause of low-volume rectal bleeding in all age groups. They are often triggered by constipation, which is more common during periods of dietary change (introduction of solid food or cow's milk), toilet training, or around school entry. In older children, anal fissures also may be associated with inflammatory bowel disease (IBD). (See 'Anal fissures' above.)

- Milk or soy-induced colitis is a common cause of bloody stools in infants and usually resolves by 12 months of age. It can occur in infants who are formula-fed, or in breastfed infants because of cow's milk in the mother's diet. Affected infants have loose stools, often with occult or gross blood, but are usually otherwise healthy. It should be differentiated from lymphoid nodular hyperplasia, a benign self-resolving condition. (See 'Milk- or soy-induced colitis' above.)

- Meckel's diverticulum is usually asymptomatic, but may cause painless rectal bleeding, which may be chronic and insidious, or acute and massive. It can present at any age, but is most common in children under two years of age. (See 'Meckel's diverticulum' above.)

- Causes of rectal bleeding that present as acute abdominal crisis include malrotation with midgut volvulus (usually in newborn infants but may be seen in children of any age); Hirschsprung disease complicated by obstruction (usually in newborns) or by Hirschsprung disease-associated enterocolitis (usually in infants or those with known Hirschsprung disease); or intussusception (usually in infants or preschool aged children). (See 'Malrotation with midgut volvulus' above and 'Hirschsprung disease' above and 'Intussusception' above.)

- Juvenile polyps are benign hamartomas, which typically occur between the ages of 2 and 10 years, with a peak at 3 to 4 years. Patients usually present with painless rectal bleeding, although a few may have lower abdominal pain from traction on the polyp. Most polyps in children are solitary and benign, but children with multiple polyps may have a polyposis syndrome and require surveillance. (See 'Juvenile polyps' above.)

- Inflammatory bowel disease (IBD), which includes ulcerative colitis (UC) and Crohn disease (CD), most commonly presents with abdominal pain, fever, weight loss, growth failure, and bloody or nonbloody diarrhea. The peak incidence of IBD is in late adolescence and early adulthood, but a significant number of children present prior to adolescence, and IBD is occasionally diagnosed in very young children. The diagnosis usually is suspected by the chronicity of symptoms and weight loss or growth failure, especially in those with CD. Many patients have iron deficiency and/or an elevated erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP). (See 'Inflammatory bowel disease' above.)

ACKNOWLEDGMENT — The editorial staff at UpToDate would like to acknowledge Chris Ramsook, MD, and Erin E Endom, MD, who contributed to an earlier version of this topic review.

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Topic 5904 Version 31.0