THE YALE JOURNAL OF BIOLOGY AND MEDICINE 57 (1984), 135-147

Gastrointestinal Bleeding in the Pediatric Patient

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Received June 27, 1983

Gastrointestinal hemorrhage in infants and children is a catastrophic event but is not associated with significant mortality except in those with a severe primary illness. Upper gastrointestinal bleeding in infants and young children is most often associated with stress ulcers or erosions, but in older children it may also be caused by duodenal ulcer, esophagitis, and esophageal varices. Lower gastrointestinal bleeding may be caused by a variety of lesions among which are infectious colitides, Meckel's diverticulum, bleeding disorders, gastrointestinal allergy, and inflammatory bowel disease. Techniques of diagnosis and management are discussed.

Gastrointestinal hemorrhage can be one of the most catastrophic events in childhood and requires prompt assessment, diagnosis, and treatment [1-6]. *Hematemesis* refers to the vomiting of fresh red blood or of coffee-ground material and documents upper gastrointestinal bleeding from lesions proximal to the ligament of Treitz. Blood losses are often more marked in upper than in lower gastrointestinal bleeding. *Melena* refers to the passage per rectum of tarry stools and denotes bleeding from the upper gastrointestinal tract or the proximal small bowel. The tarry stools may be intermingled with maroon or red blood. *Hematochezia* is the passage of bright red blood per rectum and indicates a source of bleeding low in the gastrointestinal tract, usually in the colon. However, since blood exerts a cathartic action, massive upper gastrointestinal bleeding may occasionally present as hematochezia. Many substances ingested by children may simulate fresh or chemically changed blood. Red food coloring, as in some cereals, Jello, and Kool Aid as well as fruit juices and beets, may resemble blood, if vomited. Melena may be confused with dark- or black-colored stools due to iron supplementation, bismuth subsalicylate, dark chocolate, grape juice, spinach, cranberries, or blueberries. The presence of blood in gastric contents or in stools can be confirmed by a simple Hematest performed at the bedside. In the neonate, the Apt test will differentiate maternal from infant blood.

Before the advent of techniques to visualize the upper and lower gastrointestinal tracts directly, the source of gastrointestinal bleeding in children was not identified in nearly one-third of the patients. In infants, the cause of gastrointestinal bleeding was even less frequently identified [1-4]. In studies from the early 1960s, the prognosis for children with gastrointestinal bleeding was poor. In 1964, Spencer reported

Supported in part by the Children's Clinical Research Center Grant RR00125 and NIH Grant 00125.

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54 infants and children with peptic ulcerations of the stomach and duodenum. The overall mortality for the entire group was 65 percent and for infants under six months of age, 83 percent [2]. In 1967, a review of gastrointestinal bleeding in 94 neonates failed to identify lesion in 49, or 53 percent [5]. Typically those infants had hematemesis or rectal bleeding between one and four days of age, had normal radiologic examinations, and recovered after intensive medical support. Massive gastrointestinal hemorrhage was reported in eight neonates who had a similar presentation, and all survived without surgical intervention [4]. In our experience at the Yale–New Haven Medical Center, only 2 percent of infants with upper gastrointestinal hemorrhage seen during the last decade have required surgical intervention.

HISTORY AND PHYSICAL EXAMINATION

Pertinent items sought in the history are umbilical catheterization or sepsis in the neonatal period, previous episodes of bleeding from the gastrointestinal tract or other sites, hematologic disorders, liver disease, aspiration or other drug ingestion, or family history of peptic ulcer, telangiectasia, bleeding disorders, Ehlers-Danlos syndrome, or familial polyposis [6–13].

The physical examination should stress examination of the posterior nose and pharynx to eliminate epistaxis as the source of bleeding. Signs of liver disease or portal hypertension may be subtle in the child, but the presence of icterus, abdominal distention, prominent abdominal venus pattern, hepatosplenomegaly, cutaneous spider nevi, or ascites suggests liver disease and/or portal hypertension with esophageal varices. Splenomegaly, however, in the presence of depleted blood volume may no longer be recognized. Cutaneous petechiae, ecchymoses, purpura, or telangiectasia indicate hematologic disorders or vascular lesions. Cutaneous and oral pigmentation suggest Peutz-Jegher's syndrome, whereas soft tissue or bony tumors or bone tumors suggest Gardner's syndrome of congenital polyposts [14]. A short neck with wide-spread nipples in a female suggest intestinal vascular malformations associated with Turner's syndrome. A murmur of aortic stenosis may indicate bleeding from an associated right colonic vascular malformation.

UPPER GASTROINTESTINAL BLEEDING

The etiology, diagnostic considerations, and therapeutic approach to upper gastrointestinal bleeding vary with the age of the patient (Tables 1 and 2). In young infants under one year of age, gastrointestinal bleeding is most often related to duodenal ulcer, gastric erosions, ulceration, or esophagitis [6,15–17]. In older infants and children, drugs as well as stress contribute to bleeding from peptic ulceration and bleeding from esophageal lesions becomes more prominent. In a review of children with severe upper gastrointestinal hemorrhage, bleeding was associated with other severe, life-threatening diseases in 40 to 75 percent of patients; with aspirin ingestion in 12 to 25 percent; and with steroid therapy in 12 percent. Children under stress from major surgical procedures or those with burns or increased central nervous system pressure are at increased risk for gastric ulceration [18–20]. Recent studies suggest that the stress of major surgery in children may increase total pepsin and pepsin I concentrations as a result of vagal stimulation of gastric chief cells. Decreased blood flows, hypoxia, and defective mucous barrier have all been postulated as contributing to decreased mucosal resistance in patients with gastric ulcers or erosions.

TABLE 1

	No. Pt.	Duodenal ulcer	Duodenitis	Gastric ulcer	Gastric erosions and ulcer	Esophagitis	Varices
A: Source of UGI Bleeding in Children 1–6 Years Old							
YNHH 1978–1980	8	2	1	1	1	2	1
Cox and Ament [16]	15	1	–	6	1	3	3
Graham et al. [38]	4	1	–	2	1	–	–
TOTAL	27	5	1	9	3	5	4
		22% Duodenal		44% Gastric		33% Esophageal	
B: Source of UGI Bleeding in Children 7–18 Years Old							
YNHH 1978–1980	7	3	1	0	1	2	1
Cox and Ament [16]	26	10	0	5	4	3	4
Graham et al. [38]	8	3	0	2	1	0	1
TOTAL	41	16	1	7	6	5	6
		41% Duodenal		31% Gastric		27% Esophageal	

Duodenal ulcers are less frequently associated with stress-related states [21,22]. Liebman has shown that children, like adults with duodenal ulcers, have increased levels of serum gastrin (although these fall within normal adult levels) [23]. Gastric hypersecretion is not consistently present in children with ulcer disease [24]. Signifcant hemorrhage may occur in 24 percent of children with duodenal ulcer, although perforation is infrequent. There also appears to be a genetic tendency for childhood duodenal ulcer disease, with up to 50 percent of immediate family members having a history of peptic ulceration as compared to 10 to 15 percent for age-matched controls. Most studies in children report a higher incidence of duodenal ulcers in males.

TABLE 2
Common Causes of
Upper Gastrointestinal Bleeding

Esophagus:	Varices
	Esophagitis
	Mallory-Weiss tear
	Duplication cyst
Gastric:	Ulcer
	Erosions
	Hematoma
	Tumor
	Duplication cyst
Duodenum:	Erosions
	Ulcers
	Duodenitis
	Hemobilia
	Hematoma
Non-specific:	Swallowed blood
	Bleeding disorders

Those with gastric ulcers do not have evidence of increased acid secretion and it is postulated that the mucosal barrier to the H^+ ion is more permeable than in normals [25]. Bile acids may disrupt the mucosal barrier [26]. Salicylates and other antiprostaglandin drugs predispose to gastric ulceration by diminishing the cytoprotective effects of prostaglandins [28].

Children who bleed from esophagitis usually have a history of gastroesophageal reflux, scoliosis, or body cast application after orthopedic procedures [14,17]. Few children under three to four years of age bleed from esophageal varices unless there is a previous history of intrauterine viral infection causing liver disease or of neonatal portal vein thrombosis. Bleeding from esophageal varices does not usually occur until after four years of age, and the first and second bleeds are not lethal. Upper gastrointestinal hemorrhage in the child with cystic fibrosis may result from either duodenal ulcer or from esophageal varices which have developed secondary to occult liver disease [29–30]. As in adults, nearly half of the children with esophageal varices will bleed from other lesions such as gastric erosions or peptic ulcer. With the increasing use of endoscopy, Mallory-Weiss tears of the esophagus are now being recognized more frequently as a cause of hematemesis after episodes of forceful vomiting. Such tears usually respond well to conservative medical management.

Diagnostic Procedures

Once the assessment and stabilization of the patient have been initiated and the bleed determined to originate in the upper gastrointestinal tract [31–32], attention turns to the appropriate diagnostic procedures. The source of upper gastrointestinal bleeding in the child can be identified by conventional upper gastrointestinal contrast radiography in 50 to 75 percent of patients and has its highest yield in those with esophageal varices [16,17,33]. Double contrast studies may improve the sensitivity of this test by defining shallow gastric or esophageal erosions. Upper panendoscopy has identified the source of bleeding in 70 to 100 percent of patients, and studies have estimated that up to 27 percent of those with negative radiologic examinations will have an endoscopically recognized abnormality. Failure to identify lesions during endoscopy is due to excessive bleeding which obscures visualization or to a lesion which is situated below the reach of the endoscope.

Upper panendoscopy is clearly superior to conventional contrast radiography in determining the exact source of bleeding [33] and, in skilled hands, carries minimal risk. It is not clear, however, whether the emergent performance of this procedure will affect the outcome of most cases of acute bleeding. The one instance where endoscopy is clearly indicated is to differentiate esophageal varices from other gastrointestinal lesions. Therefore the patient who is actively bleeding and has known liver disease or portal hypertension should be endoscoped immediately. Children who present with hematemesis and a prior history of salicylate ingestion or who respond promptly to conventional medical therapy do not require immediate endoscopy.

Upper panendoscopy in the pediatric age group has become technically easier and safer during the last decade due to the technical advances in equipment. The two most frequently used endoscopes in children are the GIF P1 and P2 models manufactured by Olympus. The rare young infant who requires endoscopy with a smaller instrument may be examined with the flexible bronchoscope, which has a diameter of 5 mm. Critically ill children with coagulation disorders or thrombocytopenia are at increased risk of endoscopic trauma, and endoscopy should be avoided until bleeding

can be controlled. Infants under one year of age, because of the small size of the esophagus and danger of airway compromise, are best examined under general anesthesia or with an anesthetist standing by. The 0.8 to 1.3 percent operative mortality from general anesthesia must be weighed against the value of the procedure [34]. The procedure, however, when performed with appropriate precaution is technically feasible even in the newborn. In older children and adolescents endoscopy can be performed without general anesthesia and with moderate sedation. If the patient is hypotensive, endoscopy may be performed without any sedation. Sedation of the hemodynamically stable patient is achieved with a mixture of meperidine, 0.5 to 2 mg/kg, and diazepam, 0.1–0.5 mg/kg, given intravenously. Anesthesia of the throat is achieved through the use of a Tessilon pearl or cetocaine spray. The patient is placed in the left lateral decubitus position and the endoscope introduced into the hypopharynx and slowly through the esophagus into the stomach. Under ideal circumstances the esophagus, greater and lesser curvatures of the stomach, pylorus, and duodenum should be thoroughly examined. During the procedure, blood pressure and respiration are closely monitored, with narcan available to reverse meperidine depression.

Selective angiography is extremely valuable in the assessment of the child with portal hypertension and upper gastrointestinal bleeding [35]. Intravascular pressure measurements at the time of study should include right atrial filling pressure and free hepatic vein pressure to distinguish suprahepatic vein thrombosis from congenital anomalies such as web or thrombosis of the portal venous system. A wedged hepatic vein pressure will estimate actual portal vein pressure. The difference between this and the free hepatic vein pressure denotes the liver's contribution to the portal hypertension. Injection of contrast material into the superior mesenteric and celiac artery permits careful visualization of the splenic and renal arteries in order to assess them for possible shunt procedures. The venous phase of this injection will demonstrate the portal vein and its collaterals and permit visualization of the esophageal or gastric varices.

The use of angiography to detect the exact site of bleeding in the child is often not successful because a bleeding rate of 0.5 ml/minute is required before luminal extravasation can be visualized.

Therapy

The child presenting with upper gastrointestinal hemorrhage requires rapid assessment of bleed loss and initiation of supportive care (Table 3). Therapy is often initiated before diagnosis is established. A return of red blood or coffee-ground material through the nasogastric tube indicates a lesion above the ligament of Treitz, although its absence does not preclude bleeding distal to the pylorus [32]. If the bleeding is active and the aspirate contains clots, a large-bore Ewald tube is placed for more adequate drainage. Iced saline lavage is initiated and continued until bleeding ceases. The tube may be left in place for a short time thereafter or removed, since its presence may aggravate esophagitis or bleeding from esophageal varices. The patient should be placed on his side to prevent aspiration, and bleeding studies should be performed immediately. Rapid replacement of fluid volume is initiated with normal saline or lactate until colloid or blood becomes available. A massive bleed requiring immediate transfusion may be treated with emergency cross-matched or type-specific uncross-matched blood.

Therapy is oriented to remove or to neutralize gastric acid. In this respect, con-

TABLE 3
Causes of Rectal Bleeding

Neonate	6 Weeks–1 Year	1–12 Years
Swallowed maternal blood	Anal fissure	Anal fissure
Anal fissure	Infection	Infection
Infection	Milk or soy allergy	Intussusception
Milk allergy	Intussusception	Polyp
Meckel's diverticulum	Meckel's diverticulum	Colitis
Duplication cyst	Polyp	Meckel's diverticulum
Hemorrhagic disease	Lymphoid hyperplasia	Lymphoid hyperplasia
Arteriovenous malformation	Hematoma	Milk allergy
	Duplication cyst	Hematoma
	Arteriovenous malformation	Tumor
	Peptic ulcer	Gastric heterotopia
	Foreign body	
	Colitis of immune deficiency	
	Tumor	
	Gastric heterotopia	

tinued gastric aspiration is therapeutic and may be continued, using a smaller feeding tube. Antacids to maintain the gastric pH above 5 have been and remain the major form of therapy [36]. They are administered in dosage of 5 to 50 ml (depending upon patient size) hourly through the tube. A cold, constant-drop formula has been used successfully in the later treatment of small infants, but care must be taken to intermittently aspirate the stomach to avoid fluid accumulation and vomiting. Cimetidine, a histamine-receptor antagonist, alone has not proved effective in the management of acute upper gastrointestinal bleeding and is of only questionable value when combined with antacids, particularly in the severely stressed patient. In control studies of patients with gastric ulcer, the drug has actually seemed to increase the acute bleed [37]. Cimetidine does seem to be an effective agent, however, for the chronic treatment of duodenal ulcer and is administered four times daily in a dosage to total 20 to 30 mg/kg per day. Ranitidine, a currently released drug, is administered twice daily and has none of the reported side effects of Cimetidine. Experience with its use in children, however, is not available. Patients with esophagitis are treated similarly, but are placed in a moderately erect position, and the nasogastric tube is removed as soon as hemostasis is achieved. If there is evidence of bile reflux in the esophagus at the time of endoscopy, antacids of the aluminum hydroxide type are given because of their bile acid binding properties. Bleeding esophageal varices are treated by intravenous pitressin in dosage of 2.5 milliunits/kg/minute [14–38]. Frequent side effects of vasopressin are vasoconstriction, myocardial ischemia, and necrosis of tissue. Recently there has been increasing experience with the procedure of selective arterial embolization after the site of bleeding has been localized by angiography. The development of an inflatable balloon in the size appropriate for use in children has allowed this technique to be applied. A 10 kg child whose life was threatened by hemorrhage from the gastroduodenal artery was successfully treated using such a balloon [39,40]. Experience in the pediatric age group is yet limited and this technique cannot be recommended except in the exceptional patient where necessary angiographic studies can be performed and embolization performed by skilled physicians [41].

A child who is bleeding so rapidly that adequately central venous pressure cannot

be maintained or who has been transfused 85 ml/kg or more (equaling the total blood volume replacement) is a candidate for immediate surgical intervention [42].

If the patient has stabilized and the nasogastric aspirate has remained clear for 12 or more hours, the tube may be removed and antacids administered orally. Milk and clear non-acidic liquids may be offered after 48 hours. If bleeding recurs and a gastric or duodenal ulcer has been identified, the likelihood of erosion into a vessel is present and surgical intervention is indicated.

Prognosis

The majority of infants and young children tolerate upper gastrointestinal bleeds extremely well. The first bleed from esophageal varices due to extrahepatic portal hypertension is usually not fatal and not associated with intrahepatic disease and is usually controlled by medical management. Acute gastric or duodenal ulcers in young infants and children usually heal without recurrence. Duodenal ulcer in older children, and particularly when associated with a positive family history for ulcer disease, may be expected to become chronic as in the adult. Deaths which may occur during or after an acute gastrointestinal bleed in infancy or childhood are usually related to the underlying disease rather than to the bleed itself.

LOWER GASTROINTESTINAL BLEEDING

Bleeding distal to the ligamet of Treitz is defined as lower gastrointestinal bleeding and can present as either malena or hematochezia, depending on the exact site and volume of the bleeding. The initial assessment and institution of supportive care is similar to that for a child with upper gastrointestinal bleeding for whom it may be difficult to determine the exact site of the lesion. Therefore, examination of a gastric aspirate in any patient with significant lower gastrointestinal bleeding without an identifiable colonic lesion is of paramount importance.

Etiology

The causes of lower gastrointestinal bleeding vary significantly with the age of the patient, as noted in Table 3 [43–60]. Advances in diagnostic procedures have markedly diminished the role of exploratory laparotomy in children. Massive lower intestinal bleeding is rarely seen in the newborn but may occur. Rectal bleeding can be associated with hemorrhagic disease of the newborn, infectious diarrhea, necrotizing enterocolitis, or enterocolitis of Hirschsprung's disease. Probably the most common cause of blood in the stool is the passage of ingested maternal blood swallowed during delivery or from a fissured maternal breast during feeding. Milk or soy protein-induced colitis may cause significant blood loss during infancy and may suddenly appear in an infant who is otherwise healthy. Congenital duplications of the intestinal tract may cause significant hemorrhage either through ulceration of the mucosa or intussusception.

During infancy and preschool years, Meckel's diverticulum and intussusception are the most common causes of bleeding. Juvenile polyps may be associated with blood streaking of the stool or the passage of blood at the end of a stool, but rarely cause significant hemorrhage. The role of nodular lymphoid hyperplasia in lower gastrointestinal bleeding remains controversial, but there is increasing evidence to suggest that this may be a common finding in this age group. It cannot be dismissed, however, since it has been a finding upon barium enema examinations performed largely for the evaluation of rectal bleeding. Henoch-Schönlein purpura and

hemolytic-uremic syndromes, as well as vascular lesions of the bowel, may also present with lower gastrointestinal bleeding. Vascular lesions of the right colon, associated with Turner's syndrome or aortic stenosis, must always be considered.

In the child over six years of age, the most common cause of significant lower gastrointestinal bleeding is an inflammatory colitis. Children of any age are vulnerable to the infectious colitides such as Salmonella, Shigella, Yersinia, and Campylobacter. Recently, rectal bleeding has been associated with infection by *Clostridium difficile* following a course of antibiotic therapy. Ulcerative colitis and Crohn's disease are being seen with increased frequency and may present with either intermittent rectal bleeding or fulminant hemorrhage.

Pathology and Pathophysiology

Milk colitis is postulated to be a hypersensitivity phenomenon and presents with findings identical to those of ulcerative colitis in older patients. The onset is usually between three to four months of age, although it has been noted in small infants by three to four weeks [14]. Sigmoidoscopy shows a friable, red mucosa which reverts to normal within 72 hours after elimination of milk protein from the diet.

A Meckel's diverticulum is usually located in the distal ileum and represents a remnant of the omphalo-mesenteric duct. It is present in approximately 22 percent of the population, with the vast majority being asymptomatic. The male-to-female ratio is 2:1. Approximately half the diverticula contain ectopic gastric mucosa and become symptomatic through bleeding within the diverticulum or through ulceration of the normal ileum adjacent to it. A few diverticula contain pancreatic tissue. In addition to intestinal bleeding, these diverticula may serve as the lead point of an intussusception or may cause volvulus or intestinal obstruction. It has been estimated that 4 percent of patients with a Meckel's diverticulum will have some complication during their lifetime.

Henoch-Schönlein purpura is a systemic vasculitis, the intestinal manifestations of which often precede the skin lesions. Massive intestinal hemorrhage occurs in approximately 10 percent of patients. The most frequent intestinal complaint is abdominal pain. The hemolytic-uremic syndrome is a systemic disease consisting of thrombocytopenia, hemolysis, and renal failure. An acute colitis occurs in approximately 50 percent of cases and may precede the renal manifestations.

Of the infectious colitides, Shigella causes a more classical left-sided dysentery, and Salmonella, involving ileum and right colon, may also involve the left or entire colon. Both may present with sigmoidoscopic and radiologic findings compatible with ulcerative colitis or Crohn's disease. Other causes of infectious colitis are Campylobacter and Yersinia, which require special cultured media and laboratory awareness. Although a pseudomembrane is considered typical for the diagnosis of *Clostridium difficile* enterocolitis, it is not always necessarily present, and again the sigmoidoscopy may reveal only an acutely inflamed, friable mucosa.

Polyps, which often cause rectal bleeding in children, rarely cause significant hemorrhage. The most common type is the juvenile polyp which may often lie within reach of the sigmoidoscope and is usually solitary. These are hamartomatous polyps which frequently autoamputate and slough within one year of diagnosis. Juvenile polyposis coli in which large numbers of juvenile polyps lie throughout the colon may be confused with familial adenomatous polyposis, and indeed there may be some overlap between these syndromes. Other types of familial polyposis such as Peutz-Jegher or Gardner's syndrome are rare and usually demonstrate chronic blood

loss and anemia rather than acute hemorrhage. Ulcerative colitis and Crohn's disease are now recognized to occur in children as young as two to three years of age. Both are idiopathic diseases with the current hypothesis of their etiology centering around immunologic or infectious agents. Ulcerative colitis may involve the colon to varying degrees, although beginning in or limited to disease within the rectum. Histologically, there are superficial mucosal ulcerations and crypt abscesses. Crohn's disease, although occasionally resembling ulcerative colitis by sigmoidoscopy, is histologically a transmural process, and granulomas may be seen deep within the tissue. Crohn's disease may involve any area of the gastrointestinal tract from pharynx to anus, with approximately two-thirds of patients having involvement of the ileum and colon and 10 to 15 percent of the colon alone. Either form of inflammatory bowel disease may present with fulminant hemorrhage and/or the development of toxic megacolon. The patient with Crohn's disease presenting with this complication is more likely to have had his disease only a short time, whereas the patient with ulcerative colitis may develop this complication at any time during the course of his disease.

Diagnosis

A large series of adults with acute intestinal bleeding had the differentiation of upper from lower gastrointestinal bleeding determined with 99 percent accuracy by the placement of a nasogastric tube and aspiration of gastric contents [31,32]. The few patients with negative gastric aspirates for blood and upper gastrointestinal bleeding had duodenal ulcers, although the vast majority of bleeding duodenal ulcer patients had positive gastric aspirates.

Once the determination has been made that the child has rectal bleeding a sigmoidoscopy is required [61]. Inflamed mucosa can be differentiated from blood-covered mucosa by swabbing the area with cotton. This maneuver also allows one to determine if the mucosa is friable. If the mucosa is erythematous, granular, ulcerated, or friable, this is evidence of an active colitis. In the face of significant blood loss and active colitis, an emergency flat film of the abdomen is necessary to rule out the presence of toxic megacolon, which is a surgical emergency. A pseudomembrane seen on sigmoidoscopy may suggest the presence of *Clostridium difficile* toxin, but in most instances the presence of colitis does not point to a specific etiologic agent.

The infectious colitides such as Salmonella or Shigella may be detected by routine stool culture. Isolation of Campylobacter requires special media to inhibit the growth of other organisms, and Yersinia requires selective media with hypothermia in a phosphate-buffered saline and two to three weeks to grow. *Clostridium difficile* may cause lower gastrointestinal bleeding; usually bleeding occurs during or follows antibiotic therapy for an upper respiratory infection. Clindamycin was first implicated, but this disease has now been reported to follow usage of nearly every common antibiotic. Diagnosis is made by assay of *Clostridium difficile* toxin or by culture of the organism. Amoeba can be documented by examination of stools for ova and parasites and by serologic testing. Crohn's disease and ulcerative colitis will be suggested by appropriate radiologic studies and other systemic manifestations. A diagnostic plan is outlined in Fig. 1.

Radionuclide scintiscanning is playing an increasingly important role in the diagnosis of lower intestinal bleeding [62–64]. The injection of 99 mTc sodium pertechnetate, which is taken up by the parietal cells of the gastric mucosa, will

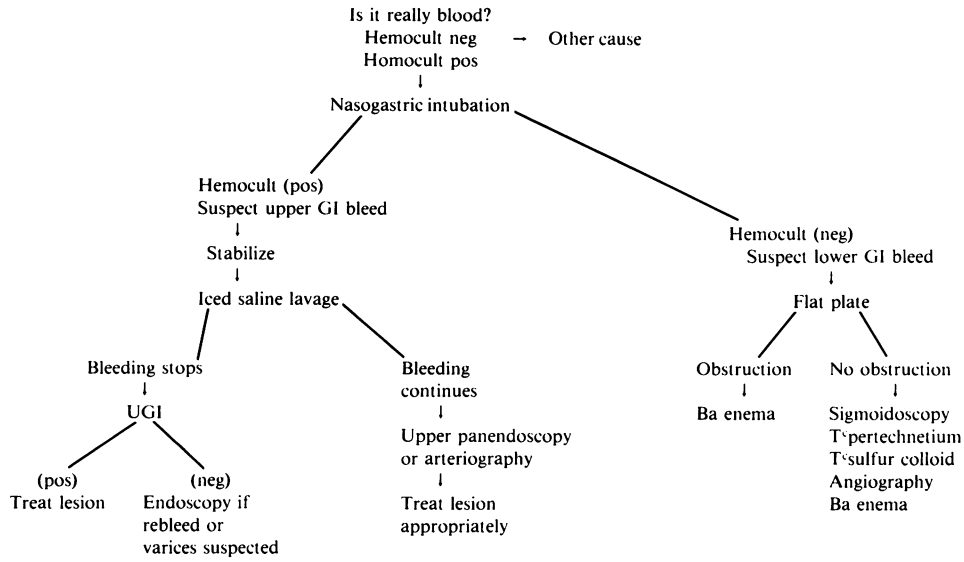

FIG. 1. Diagnostic Algorithm for Intestinal Blood Loss.

enable the visualization of ectopic gastric mucosa that is present in symptomatic Meckel's diverticulum. Other radiopharmaceuticals such as 99mTechnetium sulfur colloid, which binds to the red blood cell, have been used to localize the site of bleeding in order to make selective angiography more valuable. After injection, the isotope extravasates into the lumen, and if sufficient accumulation occurs scintiscanning will then be able to localize the site of bleeding.

The child with significant lower intestinal bleeding, a normal sigmoidoscopy, and negative Meckel's scan is candidate for selective arteriography. The bleeding must occur at a rate of 0.5–1.5 ml/kg in order to be detected by angiography.

Colonoscopy which can be valuable in the older child or the adult with severe lower gastrointestinal bleeding is not as easily performed in the small child. The size of the patient and the inaccessibility of the most common etiology to visualization (i.e., Meckel's diverticulum) make this procedure much less valuable in the pediatric age group.

A barium enema is most valuable in detection of an intussusception, polyps, or inflammatory bowel disease. Extreme caution should be used in performance of a barium enema in a patient with significant hemorrhage who is suspected of having an intussusception, since perforation is a possibility. Air contrast barium enemas are superior to standard full column barium enemas in delineating mucosal detail in order to identify polyps or evidence of superificial ulcerations of inflammatory bowel disease.

Therapy

Therapy of a massive lower gastrointestinal bleed, as in upper gastrointestinal bleeding, should initially be aimed at resuscitating the patient, localizing the site of bleeding, and finally deciding on a treatment plan to stop the hemorrhage.

A Meckel's diverticulum, duplication, arteriovenous malformation, or a polyp in a patient with massive hemorrhage should be excised operatively. The intraoperative localization of an arteriovenous malformation can be difficult and may be aided by

transillumination or Doppler determinations [65]. A life-threatening hemorrhage from inflammatory bowel disease is usually an indication for surgical intervention.

In the child in whom a bleeding source cannot be localized and who continues to have massive bleeding, the use of vasopressin intravenously at a dose of 2.5 milliunits/kg/minute may be helpful.

Prognosis

Because of the spectrum of lesions, the prognosis must be individualized for each.

CONCLUSIONS

The etiology of upper gastrointestinal bleeding is most often gastric or duodenal erosions, ulcer, or esophagitis. Esophageal varices are unusual in the child under 18 months of age, but constitute an increasing source of bleeding in the older patient with liver disease. The causes of lower gastrointestinal bleeding are numerous and vary with age. Anal fissure, intussusception, juvenile polyps, and Meckel's diverticulum are the most common lesions. Medical therapy is usually successful in terminating the bleed, but identification of the source by history and diagnostic studies is imperative if bleeding does not cease or recurs.

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