Digestive system Hemorrhage

Денис Овечкін
<table>
<thead>
<tr>
<th>Зміст</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Digestive system Hemorrhage</td>
<td>3</td>
</tr>
<tr>
<td>INTRODUCTION</td>
<td>3</td>
</tr>
<tr>
<td>PEPTIC ULCER DISEASE AND GASTRITIS</td>
<td>5</td>
</tr>
<tr>
<td>Primary Peptic Ulcer Disease</td>
<td>7</td>
</tr>
<tr>
<td>Secondary Peptic Ulcer Disease</td>
<td>7</td>
</tr>
<tr>
<td>PORTAL HYPERTENSION</td>
<td>9</td>
</tr>
<tr>
<td>MECKEL'S DIVERTICULUM</td>
<td>15</td>
</tr>
<tr>
<td>RECTAL BLEEDING IN INFANCY</td>
<td>18</td>
</tr>
<tr>
<td>POLYPS OF THE GASTROINTESTINAL TRACT</td>
<td>20</td>
</tr>
<tr>
<td>ANAL FISSURES</td>
<td>23</td>
</tr>
<tr>
<td>HEMORRHOIDS</td>
<td>24</td>
</tr>
</tbody>
</table>
Digestive system Hemorrhage

INTRODUCTION

Classification
Gastrointestinal bleeding is usually classified based on the anatomic relationship between the suspected site of bleeding and the ligament of Treitz.

- **Upper gastrointestinal bleeding** is defined as hemorrhage that occurs from a source proximal to the ligament of Treitz;
- **Lower gastrointestinal bleeding** occurs from a more distal source.

Occult gastrointestinal bleeding refers to an initial presentation with a positive fecal occult blood test or iron deficiency anemia without visible evidence of blood loss.

Clinical Presentation
Patients with upper gastrointestinal bleeding typically present with melena, hematemesis, or blood clots mixed with emesis.

Patients with lower gastrointestinal bleeding sometimes report bloody diarrhea, hematochezia, blood seen on toilet paper or blood streaks or clots mixed with stool.

Patients with occult gastrointestinal bleeding sometimes present with non-specific signs and symptoms including fatigue, pallor, or anemia.

Differential Diagnosis
Gastrointestinal bleeding occur in children of any age; however, many etiologies are age-specific and warrant additional distinction (Tables 4.1 and 4.2).

The patient's age and clinical presentation are the most useful pieces of information in determining the likely cause of bleeding and for directing the diagnostic and treatment algorithm.

Table 4.1 Age-based differential diagnosis of gastrointestinal bleeding (by Jay L. Grosfeld, 2006)

<table>
<thead>
<tr>
<th>Age</th>
<th>Upper GI bleeding</th>
<th>Lower GI bleeding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Newborn (&lt;1)</td>
<td>• Hemorrhagic disease of the newborn</td>
<td>• Anal fissure</td>
</tr>
<tr>
<td></td>
<td>• Swallowed maternal blood</td>
<td>• Necrotizing enterocolitis</td>
</tr>
<tr>
<td></td>
<td>• Stress gastritis</td>
<td>• Malrotation with</td>
</tr>
<tr>
<td>Diagnoses</td>
<td>Suggestive history/physical findings</td>
<td>Age groups</td>
</tr>
<tr>
<td>-----------------------------------------------</td>
<td>------------------------------------------------------------------------------------------------------</td>
<td>----------------</td>
</tr>
<tr>
<td><strong>Upper GI bleeding</strong></td>
<td>Vomiting, epigastric pain, dysphagia, indwelling NGT or gastrostomy tube, critical illness, NSAIDs, alcohol, caustic ingestion</td>
<td>All age groups</td>
</tr>
<tr>
<td>Esophagitis, gastritis or gastroduodenal ulcers</td>
<td>Vomiting, GERD, epigastric pain, dysphagia, indwelling NGT or gastrostomy tube, critical illness, NSAIDs, alcohol, caustic ingestion</td>
<td>All age groups</td>
</tr>
<tr>
<td>Mallory-Weiss tear</td>
<td>Hematemesis after forceful vomiting</td>
<td>All age groups</td>
</tr>
<tr>
<td>Varices</td>
<td>Hematemesis with hepatomegaly, splenomegaly, jaundice or ascites</td>
<td>Infancy and older</td>
</tr>
<tr>
<td><strong>Lower GI bleeding</strong></td>
<td>Painful defecation with streaks of red blood on stool</td>
<td>All age groups</td>
</tr>
<tr>
<td>Anorectal fissure</td>
<td>Painful defecation with streaks of red blood on stool</td>
<td>All age groups</td>
</tr>
<tr>
<td>Allergic colitis</td>
<td>Blood stained vomiting or diarrhea within 48 h of introducing formula</td>
<td>Neonates and infants</td>
</tr>
</tbody>
</table>

Table 4.2 Common presentation and workup of specific causes of gastrointestinal bleeding [36]
The approach to any patient with gastrointestinal bleeding should begin with an assessment of hemodynamic stability and overall clinical status followed by resuscitation, diagnosis, and therapy. After resuscitation, the level of bleeding must be established and a list of potential diagnoses generated based on the child's age and clinical presentation. A nasogastric tube lavage helps to confirm or exclude an upper GI source of bleeding (proximal to the ligament of treitz) and to remove particulate matter and clots from the stomach to facilitate endoscopy. For patients with a suspected upper GI bleed, esophagogastroduodenoscopy (EGD) helps identify the bleeding source, permits treatment of the identified bleeding lesions, and allows for stratification of the risk for rebleeding. Adjunct treatments for upper gastrointestinal bleeding may include intravenous proton pump inhibitors or octreotide. For patients with a suspected lower GI bleed, the diagnostic workup depends on the suspected diagnosis based on the patient's age and presentation.

### Peptic Ulcer Disease and Gastritis

Acid-peptic injury to the mucosa of the stomach and duodenum in the form of inflammation, erosion and ulcerations occurs infrequently in childhood. Gastroduodenal ulcers are classified as either primary or secondary (Tables 4.3).

#### Tables 4.3 Classification and Causes of Gastritis and Ulcers in Children [43]

<table>
<thead>
<tr>
<th>Classification/Category</th>
<th>Etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Digestive system</td>
<td>Hemorrhage</td>
</tr>
<tr>
<td>Necrotizing enterocolitis</td>
<td>Non-specific systemic signs of toxicity with abdominal distention, tenderness, vomiting, thrombocytopenia, or diarrhea with enteral feeding</td>
</tr>
<tr>
<td>Malrotation with midgut volvulus</td>
<td>Melena with abdominal distention and bilious emesis</td>
</tr>
<tr>
<td>Hirschsprung disease</td>
<td>Delayed meconium passage (&gt;48 h) or progressive constipation with abdominal distention</td>
</tr>
<tr>
<td>Intussusception</td>
<td>Sudden onset, severe, colicky pain with vomiting and bloody mucoid stool; possible abdominal mass</td>
</tr>
<tr>
<td>Meckel's diverticulum</td>
<td>Well child with large volume painless bleed</td>
</tr>
<tr>
<td>Lymphonodular hyperplasia</td>
<td>Painless bleeding after viral illness or allergic colitis</td>
</tr>
<tr>
<td>Juvenile polyp</td>
<td>Painless rectal bleeding with blood on top of the stool</td>
</tr>
<tr>
<td>Infectious diarrhea</td>
<td>Bloody diarrhea with fever, pain, or tenesmus</td>
</tr>
<tr>
<td>Inflammatory bowel disease</td>
<td>Chronic bloody diarrhea with weight loss, anorexia, arthralgia or erythema nodosum</td>
</tr>
</tbody>
</table>
Primary ulcerations occur in the absence of any underlying systemic disease, acute medical illness, or ulcerogenic medications.

Secondary ulcers are related to prematurity, steroid use, sepsis, or major physical or thermal injury. Children in need of surgical evaluation or therapy for these disorders often present with complications such as bleeding, perforation, obstruction, or chronicity.

**Pathophysiology**

Basal acid output in children with peptic ulcer disease is not significantly different than that of control subjects. Although acid and pepsin are necessary for the development of ulcers, acid hypersecretion is only rarely the sole cause of peptic ulcer disease [14, 36, 43].

The Zollinger-Ellison syndrome (hypergastrinemia secondary to gastrinoma) is exceptionally rare in children [8].

The increased frequency of peptic ulcer disease in children with chronic renal failure is attributed to elevated gastrin levels. G-cell hyperplasia, systemic mastocytosis and hyperparathyroidism are rare conditions associated with increased hyperacidity.

The most common mechanism for ulcer formation involves a decrease in the ability of the mucosa to generate the thick mucus layer on the surface of the stomach to provide an effective barrier to acid.

**The cause of gastrointestinal ulcers** has been related to one or a combination of three factors [14, 16, 36]:

- decreased mucosal blood flow
- disruption of the protective mucosal barrier
- intraluminal acidity

Steroids, anti-inflammatory medications, or a low arterial pH of the blood supplying the stomach further impair the ability of the mucosa to protect itself. Inadequate gastric emptying may exacerbate mucosal injury by increasing the duration of exposure to acid.

Helicobacter pylori is an urease producing, spiral-shaped, Gram-negative rod whose has changed traditional concepts about the pathogenesis and treatment of peptic ulcer disease. The hydrolysis of urea to ammonia and water produces an alkaline microenvironment that shields the H. pylori from gastric acid.

Bacteria and their cytotoxins are responsible for the inflammatory process. Nearly all patients with duodenal ulcers and about 85% of patients with gastric ulcers are infected with H. pylori [36, 43].

**NB:** H. pylori gastritis is the most common cause of chronic gastritis in children [2, 3, 8, 36, 43].

In developing countries, up to 70% of children are infected with H. pylori by 15 years of age. H. pylori transmission is person to person via fecal-oral, oral-oral, or gastro-oral routes. The gastro-oral route of
transmission infers the vomitus as the contaminant in situations of overcrowding and poor sanitation.

**Primary Peptic Ulcer Disease**

The true incidence of peptic ulcer disease in children is unknown. Although the incidence in boys is 2 to 3 times higher than that in girls, the sex distribution is similar in infants and very young children.

Ulcers occur more often in patients with blood type O, who have a 30% increased risk for developing duodenal ulcers when compared with those individuals with blood types A, B, and AB [14, 43].

A strong familial tendency has been noted. 30–60% of children with ulcer disease have first- and second-degree relatives with peptic ulcer disease.

**Secondary Peptic Ulcer Disease**

Secondary peptic ulcers are usually due to noxious agents (e.g., corticosteroids and NSAIDs) or after major stresses (e.g., burns, head injury, major physical or thermal trauma, sepsis, shock, systemic illness).

Stress ulcers account for 80% of the peptic disease seen during infancy and early childhood [43]. Gastrointestinal bleeding, the predominant presenting symptom of secondary ulcers, occurs in 90% of patients younger than 6 years of age [14, 43].

Stress ulcers in children are duodenal, single, and deeply penetrating; in contrast, stress ulcers in adults are multiple and superficial [3, 4, 8].

Gastric stress ulcers are usually multiple superficial mucosal erosions found primarily in the fundus of the stomach.

**Clinical Presentation**

The clinical features of peptic ulcer disease in children can easily be confused with those of many other disorders; this similarity may inflate the actual incidence of ulcer disease in children (Tables 4.4).

The symptoms of gastritis, peptic ulcer and duodenitis are similar and nonspecific.

Tables 4.4 Clinical Findings in Primary and Secondary Gastritis and Peptic Ulcer Disease (by Jay L. Grosfeld, 2006).

<table>
<thead>
<tr>
<th>Disease Entity</th>
<th>Signs and Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary gastritis</td>
<td>• Recurrent abdominal pain (any location)</td>
</tr>
<tr>
<td></td>
<td>• Epigastric pain</td>
</tr>
<tr>
<td></td>
<td>• Water brash, heartburn (gastroesophageal reflux disease symptoms)</td>
</tr>
<tr>
<td></td>
<td>• Vomiting, nausea, anorexia</td>
</tr>
<tr>
<td></td>
<td>• Iron deficiency anemia</td>
</tr>
<tr>
<td></td>
<td>• Short stature, growth failure (?)</td>
</tr>
<tr>
<td>Secondary gastritis</td>
<td>• Abdominal pain</td>
</tr>
<tr>
<td></td>
<td>• Upper gastrointestinal blood loss – hematemesis, melena</td>
</tr>
<tr>
<td></td>
<td>• Epigastric pain localization (&quot;crampy&quot;)</td>
</tr>
<tr>
<td></td>
<td>• Irritability, change in feeding patterns</td>
</tr>
<tr>
<td></td>
<td>• Fatigue</td>
</tr>
<tr>
<td></td>
<td>• Iron deficiency anemia</td>
</tr>
<tr>
<td>Primary peptic ulcer disease</td>
<td>• Chronic, recurrent abdominal pain</td>
</tr>
<tr>
<td></td>
<td>• Episodic epigastric pain</td>
</tr>
<tr>
<td></td>
<td>• Vomiting, particularly recurrent</td>
</tr>
<tr>
<td></td>
<td>• Nocturnal awakening</td>
</tr>
<tr>
<td></td>
<td>• Anemia</td>
</tr>
<tr>
<td>Secondary peptic ulcer disease</td>
<td>• Life-threatening gastrointestinal bleeding</td>
</tr>
<tr>
<td></td>
<td>• Gastric or duodenal perforation</td>
</tr>
<tr>
<td></td>
<td>• Shock</td>
</tr>
<tr>
<td></td>
<td>• Abdominal pain (rare)</td>
</tr>
</tbody>
</table>
For children up to 2 years of age, these symptoms consist of recurrent vomiting, difficulty with feeds, growth delay and gastrointestinal bleeding. In preschool children, postprandial pain, vomiting and hemorrhage are the common presenting features.

Older children present similar to adults.

**NB:** Six symptoms correlated significantly with acid-peptic disease: epigastric pain, nocturnal pain, postprandial pain, water brash, weight loss, and a family history of peptic ulcer disease.

**Diagnosis**
- **Noninvasive** diagnostic tests for H. pylori include:
  - Urea breath testing
  - Stool test for H. pylori antigen (enzyme-linked immunosorbent assays [ELISAs])
  - Blood tests consisting of whole blood and serological assays

- **Invasive** tests include:
  - Endoscopy with gastric biopsy

Due to multiple factors, noninvasive tests may not be optimal for the pediatric population. Because the performance of the urea breath tests requires some level of oral-pharyngeal coordination and the ability to breathe into a straw, it may be difficult to produce accurate results from young children [43].

Due to the persistence of immunoglobulin G (IgG) antibodies that may last for months and possibly years, the serologic assays cannot differentiate between active or past infection.

Finally, stool antigen tests have not shown uniform results, reducing their positive predictive values.

Due to the limitations of noninvasive tests, an EGD with biopsy remains the recommended diagnostic test [14, 16, 43].

Direct visualization and biopsies of the affected areas of the mucosa can determine the organic etiology of ulcer disease and gastritis. A histologic study, rapid urease testing, or tissue culture on biopsy can identify H. pylori.

**Medical Treatment**

The treatment of peptic ulcer disease in children is similar to that employed in adults.

Antacids and H\(_2\)-receptor antagonists (i.e., cimetidine, ranitidine, famotidine) are the mainstays of medical therapy.

The effectiveness of H\(_2\)-receptor antagonists and proton-pump inhibitors (PPI) for the prevention of stress ulcers is extremely effective.

Eradiation of H. pylori requires a combination of gastric acid antisecretory agents plus an antimicrobial agent administered for 10 to 14 days (Table 4.5).

**Table 4.5 Recommended Combination Eradication Therapies for H. pylori-Associated Disease in Children according the evidence-based guidelines from North American Society for Pediatric Gastroenterology, Hepatology and Nutrition (NASPGHAN), 2011**

<table>
<thead>
<tr>
<th>Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>PPI + amoxicillin + metronidazole</td>
</tr>
<tr>
<td>PPI + amoxicillin + clarithromycin</td>
</tr>
<tr>
<td>Bismuth subsalicylate (or subcitrate) + amoxicillin + metronidazole</td>
</tr>
<tr>
<td>PPI + amoxicillin + clarithromycin + metronidazol</td>
</tr>
</tbody>
</table>

First line regimens, each agent administered twice daily for 10-14 days
Dose: PPI (1-2 mg/kg/day); amoxicillin (50 mg/kg/day); clarithromycin (15 mg/kg/day); metronidazole (20 mg/kg/day).

Once treated and cured, children are at a low risk for recurrence.

Management following treatment failure (according to the evidence-based guidelines from NASPGHAN, 2011):

- EGD, with culture and susceptibility testing, including alternate antibiotics
- Fluorescence in situ hybridization (FISH) on previous paraffin-embedded biopsies (if clarithromycin susceptibility testing has not been performed)
- Modification of therapy (addition of an antibiotic, different antibiotics, addition of bismuth, and/or increasing dose and/or duration of therapy)

Surgical Treatment

With the advent of advanced modern pharmacological therapy, surgical intervention is generally reserved for the management of acute complications, such as perforation or bleeding.

In the majority of cases, bleeding responds well to nasogastric decompression, volume replacement and transfusion therapy.

For major or persistent bleeding, endoscopic treatment modalities include therapeutic injections (i.e., hypertonic NaCl, epinephrine, absolute ethanol) and cauterization with heater probe, bipolar coagulator, or laser (or argon).

If medical and endoscopic treatments fail, surgery is indicated.

The surgical procedures used to treat peptic ulcer disease include:

- Simple closure of a localized perforation overlaid with omental patch
- Gastrotomy or duodenostomy with over-sewing of the base of a bleeding ulcer
- Partial and subtotal gastrectomies
- Vagotomy with either pyloroplasty or antrectomy
- Proximal gastric vagotomy

Vagotomy with pyloroplasty is the traditional approach that provides good long-term results causing minimal disturbance of growth and development. The choice of the operation should be individualized, taking into account the likelihood of recurrence, the comorbid factors and the nutritional and developmental needs of the growing child.

PORTAL HYPERTENSION
The portal venous system drains blood from the stomach, pancreas, gallbladder, spleen and intestines into the liver. The portal vein arises in the embryo as the left and right vitelline veins, which form numerous anastomoses among developing hepatocytes. Following gut rotation, the left vitelline vein is obliterated and the right vitelline vein persists as the main portal vein [14, 43, 45].

Portosystemic anastomoses exist in four main areas:
1. the gastroesophageal veins via the cardiac vein and perforating esophageal veins;
2. the retroperitoneum via the pancreaticoduodenal veins and the retroperitoneal-paravertebral veins;
3. the gastrorenal-splenorenal vein;
4. the hemorrhoidal plexus.

The portal venous system lacks valves, making blood flow entirely dependent upon the pressure gradient within the system. Normal flow toward the liver is termed hepatopedal.

Normal portal venous pressure is 5-10 mm Hg greater than central venous pressure. In children, portal hypertension is defined as elevation of the portal venous-inferior vena cava pressure gradient above 10-12 mm Hg or a parenchymal spleen pressure greater than 16 mm Hg [14, 16, 43].

Classification
Portal hypertension in children can be divided into two major categories based upon the anatomic location of the increased portal resistance:
- **Extrahepatic** portal hypertension (EHPH)
- **Intrahepatic** portal hypertension (IHPH)

Etiology
EHPH is most commonly the result of portal vein obstruction due to thrombosis. Risk factors for portal vein thrombosis include umbilical vein catheterization, neonatal sepsis, blunt abdominal trauma and omphalitis; idiopathic cases are also common. The thrombosis frequently recanalizes which results in cavernous transformation of the portal vein into numerous smaller channels.

IHPH is typically associated with congenital liver or biliary diseases in children. Biliary atresia is by far the most common cause of IHPH in children, followed by cystic fibrosis.

Each of the causes of elevated portal pressure shares the common mechanism of increased resistance to blood flow from the splanchnic portal circulation to the right atrium (Table 4.6). In children, the location of this increased vascular resistance can be:
- **pre-hepatic**, usually within the portal vein and its primary feeding branches;
- **intra-hepatic**, most commonly related to intrinsic liver disease, but may be secondary to presinusoidal obstruction (congenital hepatic fibrosis or schistosomiasis);
- **post-hepatic**, secondary to hepatic vein outflow obstruction.

Table 4.6 Pediatric diseases associated with portal hypertension [43]

<table>
<thead>
<tr>
<th>Pre-hepatic</th>
<th>Post-hepatic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extrahepatic portal vein thrombosis</td>
<td>Cavernous transformation of the portal vein</td>
</tr>
<tr>
<td>Splenic vein thrombosis</td>
<td></td>
</tr>
<tr>
<td>Location</td>
<td>Causes</td>
</tr>
<tr>
<td>------------------</td>
<td>------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Pre-hepatic</td>
<td>Causes: Congenital portal vein malformation (web or diaphragm), Extrinsic portal vein compression</td>
</tr>
<tr>
<td>Intra-hepatic</td>
<td>Causes: Hepatocellular disease, Biliary tract disease, Congenital hepatic fibrosis, Schistosomiasis, Sinusoidal veno-occlusive disease</td>
</tr>
<tr>
<td>Post-hepatic</td>
<td>Causes: Budd-Chiari syndrome, Inferior vena cava obstructions (web), Chronic congestive heart failure, Veno-occlusive disease (s/p bone marrow transplantation), Postoperative hepatic vein stenosis, Prothrombotic disease</td>
</tr>
<tr>
<td>Hyperkinetic</td>
<td>Causes: (high flow) Arteriovenous fistula (congenital or acquired)</td>
</tr>
</tbody>
</table>

**Incidence**

Liver disease in children is relatively rare, with an incidence of 1 in 5000-7000. EHPH is approximately twice as common as IHPH in children. The most common cause of IHPH, biliary atresia, occurs at a rate of 1 in 15,000 live births [14].

This distribution is distinctly contrary to that seen in adults, who more commonly develop IPPH as a result of alcoholic cirrhosis.

**Clinical Presentation**

Children with IHPH usually present between several months to one year of life with severe hepatic dysfunction, manifested by jaundice, hepatic encephalopathy and malnutrition complicated by poor growth and increased susceptibility to infections.

EHPH most commonly presents in the first decade of life with gastrointestinal hemorrhage from esophageal varices.

Bleeding is often precipitated by a respiratory or gastrointestinal febrile illness and aspirin is frequently implicated. Increased portal pressure causes splenic congestion, resulting in splenomegaly and hypersplenism.

**NB:** Portal hypertension should be suspected in any child with splenomegaly, unexplained thrombocytopenia, leukopenia, ascites or gastrointestinal hemorrhage [36, 45].

**Diagnosis**

**Abdominal ultrasonography (with Doppler ultrasound)** - the presence of portal vein thrombosis, the extent of collateral formation and the direction of portal vein flow is established by this noninvasive and relatively inexpensive diagnostic examination [5, 36].

**Upper endoscopy (esophagogastroduodenoscopy)** is used to identify and quantify esophageal varices. This procedure is ideally performed under general anesthesia to provide a controlled environment for a thorough study and possible therapeutic intervention (i.e., sclerotherapy or banding).
Identification of other potential sites of bleeding is also important, as many children with portal hypertension also have gastric varices, peptic ulcer disease, esophagitis, or a portal hypertensive gastropathy or enteropathy.

**Angiography** is another, less used diagnostic and potentially therapeutic modality used in certain cases of portal hypertension.

**Treatment**

The most common, clinically significant complication of portal hypertension is gastrointestinal bleeding from esophageal varices.

In the acute setting, hemorrhage is managed with intensive care monitoring. Volume resuscitation should be rapidly instituted with crystalloid and red blood cells as necessary. Fresh frozen plasma, platelets and vitamin K may be indicated in the presence of coagulopathy. A nasogastric (or orogastric) tube should be placed for lavage and monitoring. Intubation and sedation may be required for airway protection and to minimize the agitation that may increase variceal pressure.

**Medical treatment** of portal hypertension is aimed at relieving the pressure in the portal system. \( \beta \)-blockade, if tolerated, reduces cardiac output and therefore portal venous pressure. **Vasopressin** decreases portal blood flow by increasing splanchnic vascular tone. Vasopressin is initially bolused at 0.33 U/kg over 20 minutes and is then infused continuously at the same dose on an hourly basis or as an infusion of 0.2 U/1.73 m\(^2\)/min. Vasopressin has a half-life of approximately 30 minutes [3, 16, 36, 45].

Side effects of vasopressin use are secondary to cardiac or visceral vasoconstriction and may be ameliorated by transdermal nitroglycerin administration [14].

Infusion of **Octreotide**, a long-acting somatostatin analog, has also been shown to decrease splanchnic blood flow [3, 16, 36, 45].

**Endoscopic variceal banding or sclerotherapy** is used in cases where hemorrhage does not resolve with supportive care [1, 6, 8].
Once the patient has stabilized, endoscopy with sclerotherapy or banding is employed to prevent repeat episodes of hemorrhage. In bleeding that is refractory to both pharmacologic and endoscopic interventions, placement of a Sengstaken-Blakemore tube may be warranted (Image 4.1). Clinicians should be cognizant of the significant rates of complications with its use, including recurrent bleeding, pulmonary aspiration and gastroesophageal perforation.

Since there is a normally functioning liver, gastrointestinal hemorrhage in children with extrahepatic portal hypertension tends to be less severe than bleeding that occurs in children with IHPH. In addition to coagulation abnormalities, many patients with IHPH also have significant malnutrition that contributes to the greater morbidity and mortality of gastrointestinal bleeds in this group.

**Surgical treatment** of portal hypertension can be either direct, which involves ligation of the varices themselves (esophagogastric devascularization with or without splenic artery ligation) or indirect, in which the portal venous system is decompressed via a portosystemic shunt.

Surgical options for shunt procedures are based on the primary etiology of portal hypertension (Table 4.7).

**Table 4.7 Portal hypertension surgical options [36]**

<table>
<thead>
<tr>
<th>Primary etiology</th>
<th>Preferred surgical therapies</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Pre-hepatic obstruction</td>
<td></td>
</tr>
<tr>
<td>• Extrahepatic portal vein thrombosis</td>
<td></td>
</tr>
<tr>
<td>• Cavernous transformation of the portal vein</td>
<td>- Rex shunt construction</td>
</tr>
</tbody>
</table>
Nonselective portosystemic shunts divert the majority of portal blood to the caval system, which may result in a higher incidence of hepatic encephalopathy. Examples include portocaval, mesocaval and central splenorenal shunts [16, 43].

Selective portosystemic shunts divert a portion of portal blood into the systemic circulation, with the distal splenorenal (Warren) shunt being the most common [14].

Recently, selected children with EHPH due to portal vein thrombosis have been successfully treated by surgical creation of a mesenterico-left portal venous bypass (Rex shunt) [36, 43].

In many centers, transjugular intrahepatic portosystemic shunt (TIPS) therapy has become first line treatment for refractory bleeding or hypersplenism [14].

Recurrent bleeding, shunt thrombosis and hepatic encephalopathy are the most common complications of surgical shunting.

Treatment of IHPH focuses on the primary liver disease. In biliary atresia, surgery to decompress the biliary tract is ideally performed within the first 3 months of life.

With the Kasai procedure, the atretic segments of the extrahepatic bile ducts are excised and a Roux loop is anastomosed to the porta hepatis. If performed early, before significant liver injury and cirrhosis, the Kasai procedure can delay liver failure and the need for transplantation in as many as two-thirds of patients.

For advanced cirrhosis and other intrahepatic sources of portal hypertension, liver transplantation represents definitive treatment.
Outcome
For patients with EPH, sclerotherapy is effective in the treatment of acute variceal bleeding in up to 75% of patients. However, several follow-up sessions are necessary to obliterate the varices and a rebleeding rate of 5-25% is expected [14].
Selective shunts have proven successful for the control of bleeding, thrombocytopenia and leukopenia, without creating great risk of encephalopathy.

MECKEL’S DIVERTICULUM
For the most part, Meckel's diverticulum is clinically silent and is most often an incidental finding at laparotomy.
The incidental finding of an asymptomatic Meckel's diverticulum shows no gender predilection, although this is not the case with symptomatic lesions.
Presence of a Meckel's diverticulum and problems associated with it are known as the "disease of twos". Persistence of the diverticulum in the general population is 2%; it is 2 times more prevalent in males when symptoms exist. It is located 2 feet from the ileocecal valve, is 2 cm wide and 2 inches long. There are common clinical presentations (bleeding, obstruction, diverticulitis) in children around the age of two.

Embryology
By the third week of gestation, the midgut of the fetus is connected to the yolk sac via the vitelline duct. The vitelline duct progressively narrows during development until the third month of gestation, when it disappears along with resolution of umbilical herniation. Meckel's diverticula, as well as other omphalomesenteric defects, arise from incomplete regression of the omphalomesenteric (vitelline) duct [14].
Which anomaly will be manifested is dependent upon the stage at which this regression is arrested (Image 4.2):

A. Meckel's diverticulum;  
B. Meckel's diverticulum with ulceration and hemorrhage.  
C. The vitelline duct normally regresses between the 5th and 7th weeks of fetal life. When failed regression results in a fibrous band, the midgut may volvulate around it.  
D. The fibrous band, which also produce abnormal peritoneal spaces through which an internal hernia may result.  
E. The omphalointestinal fistula. If a patent connection persists between the intestine and the umbilicus, the entity is recognized as an omphalointestinal fistula.  
F. Omphalomesenteric sinus and cyst.

Image 4.2 Diagram depicting possible complications associated with different omphalomesenteric remnants. Meckel's diverticulum are symptomatic in 4-35% of patients. Infants and young children are more likely to present with symptoms.

Anatomy

Digestive system Hemorrhage 16
Meckel's diverticulum always occurs on the antimesenteric border of the ileum. Approximately 75% will have the distal tip free in the abdomen; while 25% will be tethered to the anterior abdominal wall [14, 45]. When a persistent fibrous cord exists, the bowel is predisposed to intestinal volvulus leading to obstruction. Obliteration of the proximal and distal duct with patency of the mid portion leads to the formation of a vitelline duct cyst.

When the duct remains patent, a fistula exists between the ileum and umbilicus. The blood supply to a Meckel's diverticulum is a remnant of the vitelline arteries, which in utero provided circulation to the yolk sac. The right vitelline artery becomes the superior mesenteric artery, while the left usually obliterates. In the case of Meckel's diverticula, the blood supply is from a persistent left vitelline artery and will arise from the ileal, ileocolic or mesenteric arteries.

**Pathophysiology**

Meckel's diverticulum is a **true diverticulum**, consisting of mucosal, submucosal and muscularis propria layers. As a result of the pluripotential cells lining the vitelline duct, the presence of ectopic mucosa is common in Meckel's diverticula (over 60%). Approximately 50% will contain ectopic gastric mucosa. These comprise 75% of symptomatic Meckel's diverticula. Pancreatic tissue is contained in 5%, while an additional 5% will contain both gastric and pancreatic mucosa [14, 16, 43, 45].

Although studies have demonstrated the presence of Helicobacter pylori within Meckel's diverticula, there is no evidence of a correlation between colonization and symptomatology or ulceration. This is in contrast to similar pathology in the stomach and duodenum [14].

**Clinical Presentation**

**Asymptomatic Meckel's diverticulum** - the vast majority of Meckel's diverticulum are asymptomatic. Complications are most likely to occur when the diverticulum contains heterotypic tissue. This is most often gastric, but may also be pancreatic, jejunal or colonic mucosa. The lifetime risk of developing a complication that requires surgery is thought to be 4-6%.

**Symptomatic Meckel's diverticulum** - present in a variety of ways. The three most common are rectal bleeding (40%), obstruction (25-35%) and diverticulitis (14-17%) [1, 14].

Bleeding is a result of ulceration from exposure to gastric acid secreted by parietal cells in the ectopic gastric mucosa. Histologically this ulceration usually occurs at the border between the ectopic and normal ileal mucosa and very rarely on ileal mucosa more remote from the diverticulum. In less severe cases, children may present with melena and mild anemia.

Obstruction is usually due to intussusception (about 50% of those cases that present with obstructive symptoms) with the Meckel's diverticulum serving as the lead point. Patients present with classic signs of obstruction including vomiting, abdominal pain, distension and often a palpable abdominal mass. Other causes of obstruction associated with a Meckel's diverticulum include volvulus, internal herniation, inguinal herniation (Littre's hernia) and kinking of the bowel at the diverticulum.

**NB:** A Littre's hernia is one of the rarest forms of hernia; it is an abdominal wall hernia that involves the Meckel's diverticulum.

Meckel's diverticulitis usually occurs prior to 10 years of age and is very similar to appendicitis in presentation. As such, it is often diagnosed during an appendectomy. The location of this pain can be more variable with Meckel's diverticulum as the position of the diverticulum within the abdomen is less fixed than that of the appendix. Additionally, free air in the abdomen is much more common in perforated Meckel's diverticula secondary to its lack of retroperitoneal attachments and free-floating position in the abdomen.

Meckel's diverticulum can also be associated with malignancy, the most common being carcinoid tumors. Other documented neoplasms include adenocarcinoma, leiomyoma and lymphoma [16, 43].

**Diagnosis**

If Meckel's diverticulum is suspected in a case of rectal bleeding (painless blood loss in a child somewhere around two years of age), a **Meckel's scan** (Technetium pertechnetate scintigraphy) is ordered [1, 3, 6, 8, 36].

This nuclear imaging study involves intravenous injection of Tc-99m (Technetium pertechnetate) which concentrates within gastric mucosa. When seen on a nuclear medicine scan within the terminal ileum, this "bright spot" is diagnostic for Meckel's diverticulum (Image 4.3).
Image 4.3 Concentration of the Tc-99m (Technetium pertechnetate) in ectopic gastric mucosa, presumed to be within a Meckel’s diverticulum (arrow). Also note uptake in gastric mucosa of the stomach and excreted radionucleotide in the bladder.

**Treatment**

Complications such as haemorrhage, diverticulitis, intestinal obstruction and umbilico-ileal fistulas are absolute indications for resection. [1, 3, 36].

After appropriate resuscitation, exploration through a right-lower quadrant incision or laparoscopically is performed. Interventions range from simple diverticulectomy to wedge resection, or ileal resection with primary anastomosis.

Video 1:

Video 2:
Incidental finding
When a Meckel's diverticulum is an incidental finding during laparotomy or laparoscope the indications for surgical intervention are more controversial [14, 36, 45]. Some feel numbers needed to treat do not justify the risk, as mortality rates are low. Other authors suggest the increased risk of malignancy justifies aggressive treatment. In asymptomatic individuals, some British authors advice resection of a diverticulum discovered incidentally should be considered for those presenting a higher risk of complication, such as:

- Patients aged younger than 40.
- Diverticula longer than 2 cm.
- Diverticula with narrow necks.
- Diverticula with fibrous bands.
- Suspected ectopic gastric tissue.
- Inflamed, thickened diverticula.

For a symptomatic Meckel's diverticulum, laparoscopic resection has been shown to be safer, less invasive and more cost-effective than laparotomy [36].

Postoperative complications
These are not uncommon. Complications of surgery are reported in 1-8% of asymptomatic patients. Complications include ileus, suture line or intestinal anastomotic leak, intra-abdominal abscess or pulmonary embolism. Late postoperative complications include intestinal adhesions leading to small bowel obstruction.

RECTAL BLEEDING IN INFANCY
Because rectal bleeding may result from several different diagnoses, its exact occurrence is difficult to quantitate. Blood loss is usually minor and self-limited; massive rectal hemorrhage is uncommon. Allergic colitis and anorectal fissure are increasingly common diagnoses in children younger than one year-old [43, 45].

Etiology
Age is an important consideration when evaluating a patient with rectal bleeding [3, 14, 36, 43]. A common cause of rectal bleeding in the newborn period is related to swallowing maternal blood at the time of birth. Rectal bleeding in newborns can also be caused by hemorrhagic diseases of the newborn, hypoprothombinemia and thrombocytopenia. Certain diagnoses such as necrotizing enterocolitis and allergic colitis are unique to neonates and younger infants. Juvenile polyps are occasionally the cause of rectal bleeding in older infants but are a more common cause in early childhood.
Rectal bleeding in infancy can occur as a result of hemorrhage at upper or lower gastrointestinal sites. Upper intestinal bleeding can present as rectal bleeding in young infants due to their faster intestinal transit time. Although many common etiologies of rectal bleeding in infancy have been described (Table 4.8), clinical diagnosis is illusive in nearly one-half of these children.

Table 4.8 Causes of rectal bleeding in infancy [14]

<table>
<thead>
<tr>
<th>Location of Bleeding Source</th>
<th>Age</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Neonate &lt; 1 month</td>
</tr>
<tr>
<td><strong>Upper Gastrointestinal:</strong></td>
<td></td>
</tr>
<tr>
<td>Hemorrhagic disease</td>
<td>+</td>
</tr>
<tr>
<td>Swallowed maternal blood</td>
<td>+</td>
</tr>
<tr>
<td>Esophagitis or gastritis</td>
<td>+</td>
</tr>
<tr>
<td>Peptic ulcer disease</td>
<td>+</td>
</tr>
<tr>
<td>Gastric teratoma</td>
<td></td>
</tr>
<tr>
<td>Esophageal or gastric varices</td>
<td>+</td>
</tr>
<tr>
<td><strong>Lower Gastrointestinal:</strong></td>
<td></td>
</tr>
<tr>
<td>Anal fissure</td>
<td>+</td>
</tr>
<tr>
<td>Necrotizing enterocolitis</td>
<td>+</td>
</tr>
<tr>
<td>Gangrenous bowel</td>
<td></td>
</tr>
<tr>
<td>Malrotation with midgut volvulus</td>
<td>+</td>
</tr>
<tr>
<td>Hirschsprung’s disease with enterocolitis</td>
<td>+</td>
</tr>
<tr>
<td>Allergic proctocolitis</td>
<td></td>
</tr>
<tr>
<td>Intussusception</td>
<td>+</td>
</tr>
<tr>
<td>Prolapse</td>
<td></td>
</tr>
<tr>
<td>Polyps</td>
<td>+</td>
</tr>
<tr>
<td>Meckel’s diverticulum</td>
<td></td>
</tr>
<tr>
<td>Lymphonodular hyperplasia</td>
<td></td>
</tr>
<tr>
<td>Enteritis (i.e., Campylobacter, Yersinia, Salmonella)</td>
<td>+</td>
</tr>
<tr>
<td>Inflammatory bowel disease</td>
<td></td>
</tr>
<tr>
<td>Intestinal duplication</td>
<td></td>
</tr>
<tr>
<td>GI vascular malformation</td>
<td></td>
</tr>
<tr>
<td>+++ most common cause, ++ more common cause, + relatively more common.</td>
<td></td>
</tr>
</tbody>
</table>

**Clinical Presentation**

Clinical presentation depends almost exclusively upon the underlying etiology.

Rectal bleeding may be in the form of hematochezia, melena, or occult blood. Hematochezia, although usually from a distal gastrointestinal lesion, can occur from either upper or lower gastrointestinal sources. Melena or tarry stools only occur when the bleeding is from a lesion proximal to the ligament of Treitz.

Perhaps the most common presentation of rectal bleeding in infancy is an otherwise healthy-appearing infant noted by the parents to have a small amount of bright red blood on the diaper or on the outside of stool. An anal fissure is the most likely etiology in this scenario and is often identifiable as a small tear or ulceration at the anal verge usually located posteriorly. Children also have spotty bleeding on stool, mixed with stools, or present on toilet paper as their complaint.

Necrotizing enterocolitis (NEC) may also produce rectal bleeding in infancy (usually in premature infants), although rectal bleeding is seldom the primary symptom. The diagnosis is suggested by history which may include prolonged gastric emptying, feeding intolerance, apnea, jaundice, abdominal distension, vomiting, thrombocytopenia, leukocytosis, or other signs of sepsis. Recurrent rectal bleeding after recovery from NEC suggests recurrent NEC or a post-NEC gastrointestinal stricture [14, 36].

Intussusception occurs most commonly in infants between 6 and 18 months of age. Rectal bleeding associated with intussusception is classically described as having a “currant-jelly” appearance, which is probably only apparent in about a third of cases. Intermittent abdominal pain is the usual distinguishing symptom. A palpable sausage-shaped abdominal mass helps establish the diagnosis. A pneumatic enema is performed to both confirm and reduce the intussusception.
An acute onset of melena and bilious emesis in an otherwise healthy baby suggests malrotation with midgut volvulus. At onset, the physical examination may be unremarkable [16, 36, 45]. With time, the abdomen will become progressively distended and tender to palpation. An upper gastrointestinal contrast study should be obtained immediately to confirm the diagnosis. Emergent laparotomy is indicated.

Rectal bleeding may also be a presenting sign of Meckel's diverticulum with ectopic gastric mucosa.

**Diagnosis**

The exact origin of gastrointestinal hemorrhage remains undiagnosed in about 30-50% of neonates and infants with rectal bleeding. For most of these infants, the blood loss is minor, self-limited and seldom recurs [14, 43].

Diagnosis begins with a thorough **history** and **physical examination**. Diagnostic evaluation of significant or recurrent hemorrhage may include **upper and/or lower gastrointestinal endoscopy** and **radiographic procedures** including contrast enema, tagged red cell studies, arteriography, etc.

**NB:** Colonoscopy is the preferred diagnostic modality for rectal bleeding [1, 3, 8].

**Treatment**

The treatment of rectal bleeding in infants depends upon accurate identification of the bleeding source.

**POLYPS OF THE GASTROINTESTINAL TRACT**

Intestinal polyps are much less common in children than in adults, but their association with syndromic clusters is very common. Malignant transformation, except in the syndromic cases, is less than in adults. Approximately 1% of children may have asymptomatic intestinal juvenile polyps which are benign [43]. Other types of polyps are much rarer.

**Etiology and Pathology**

The etiology of polyps in children is multifactorial and depends on the type of polyp. Etiologies and pathologic features will be discussed individually in the classification section.

**Clinical Presentation**

**Bleeding.** Lower intestinal bleeding is the hallmark presentation of most polypoid conditions. The bleeding is frequently associated with crampy abdominal pain.

The blood is usually **red**, indicating its origin in the lower gastrointestinal tract and small in quantity, unlike bleeding from duplications or Meckel's diverticula with peptic ulceration.

If the bleeding is from polyps in the small bowel, the **blood will appear darker**. In the rare cases of duodenal or gastric polyps, rectal bleeding may appear black (i.e., melena).

**Pain.** Crampy abdominal pain is a frequent symptom along with bleeding. The pain does not necessarily occur with the bleeding [6, 14].

**Intussusception.** Traction on a polyp may cause intussusception anywhere it occurs. However, jejuna or ileal polyps as a cause of intussusception are quite rare, especially for the most common form of intussusception, the ileocolonic type. Colocolonic intussusception may occur when a colonic polyp serves as a lead point. The symptoms include crampy, intermittent pain, bleeding from venous engorgement of the mucosa and signs of intestinal obstruction (i.e., vomiting, distension and obstipation) [14].

Unlike idiopathic intussusception, intussusception from a polyp occurs in older children and may not be reduced by contrast enema [3, 36].

**Diagnosis**

The diagnosis of polypoid lesions depends primarily on two modalities: **endoscopy** and **intestinal contrast studies**.

Endoscopy is advantageous as it can be both diagnostic and therapeutic. Endoscopy is also excellent for studying the colon, stomach and duodenum. It is clearly more limited when the polyps are small bowel in origin. Contrast studies for colonic polyps can be very accurate and can be useful to follow polyps for changes in number and size.

Small bowel polyps may be difficult to image. Small bowel polyps are notoriously difficult to diagnose and thankfully, occur only very rarely. Diagnosis is most often made at the time of laparotomy when bleeding or obstructive symptoms have prompted an operation.
Classification of Polyps

Benign

- **Isolated Juvenile Polyps**

These are the most common polypoid lesions of infancy and childhood. The peak age of incidence is **between 3 and 10 years** [14].

As with most polyps, crampy abdominal pain and bleeding with bowel movements are the presenting symptoms.

Juvenile polyps are hamartomatous excrescences of the intestinal mucosa. They appear to lengthen from traction caused by peristalsis and the flow of intestinal contents. Juvenile polyps naturally can be autoamputated if given enough time.

**75% of juvenile polyps occur in the rectum and sigmoid colon**, but juvenile polyps may occur in the right colon as well.

A full colonoscopy has been advocated by some when a child presents with symptoms as up to 50% of children may have additional polyps identified in the right and/or transverse colon [14].

- **Peutz-Jeghers Syndrome**

This well-known syndrome causes multiple polyps predominantly in the jejunum and duodenum. Its hallmark feature is the pigmented lesions observed on the buccal mucosa and lips of these patients. Malignant degeneration within the polyp can occur, especially with gastric and duodenal polyps, so lifelong surveillance is necessary. There is also an 18-fold increased risk for extra-intestinal cancers (uterine, breast and ovarian in females; testicular and head/neck cancers in males).

- **Adenomatous Polyps**

This lesion is rare but known to occur in childhood. Malignant degeneration can occur as in the adult-type lesion.

Malignant

- **Juvenile Polyposis and Familial Adenomatous Polyposis (FAP)**

Juvenile polyposis is an autosomal dominant disorder which causes polyps predominantly in the colon (often 50-200 seen at colonoscopy) and small bowel.

It is considered a premalignant condition and as many as 50% of these children will eventually develop gastrointestinal malignancy.

Infantile juvenile polyposis occurring in children under 2 years of age is associated with multiple large polyps, rectal bleeding, protein-losing diarrhea and failure to thrive and may require a more aggressive surgical approach.

FAP is characterized by hundreds of adenomatous polyps in the rectum and colon causing diarrhea and bleeding.

Malignant degeneration in one or more polyps is virtually certain before the age of 20 years and proctocolectomy with restorative ileoanal reconstruction is advocated before age 15-20 [14, 43].

Patients with FAP are also at increased risk for other neoplasms including desmoid tumors, epidermoid cysts, osteomas, hypertrophy of retinal pigment epithelium and upper gastrointestinal polyps and/or malignancy [45].

Children with a family history of FAP also have an 850 times greater risk of developing hepatoblastoma [14].
Treatment
The treatment of polyps of the GI tract in children can vary from simple observation of benign lesions to wide excision and chemotherapy for malignant ones.

Once diagnosed, polyps should be removed endoscopically [1, 3, 6].

Resection of segments of bowel should be reserved only for cases where cancer has developed and invaded the submucosa, or in areas where polyps are particularly dense and causing severe symptoms.

Duodenal polyposis may be a particularly difficult problem. Although most polyps can be excised endoscopically or surgically, diffuse duodenal involvement with bleeding has been treated with pancreaticoduodenectomy.

Treatment of children with FAP involves not only the individual involved but should also extend to others in the family. Family members should be screened and referred for genetic counseling and genetic analysis. The surgical treatment of FAP requires planning the process with the family and the patient. It is customary to do a complete colonic and rectal removal with a sphincter saving operation in early adolescence. A Soave type operation (endorectal pullthrough) is the one most commonly used [14].

Outcome
The prognosis when the disease is treated in a timely fashion is excellent [2, 4, 7, 18].
ANAL FISSURES

Anal fissure is a small laceration of the mucocutaneous junction of the anus. It is an acquired lesion secondary to the forceful passage of a hard stool.

Anal fissure may be suspected in children with constipation, extreme pain with defecation, rectal bleeding, or blood in the stool [2, 3, 6].

There is no clear cause for anal fissure, but it can be associated with constipation, and hypertonicity of the anal sphincter. This results in a vicious cycle of pain with bowel movements, and subsequent reluctance to have bowel movements and constipation [14].

NB: Anal fissures are usually due to anal trauma from passing hard stool, though rarely they can be a harbinger of Crohn's disease.

Anal fissures heal slowly and are reinjured by the passage of subsequent hard stools. Relief of the constipation cures the condition in the vast majority of patients.

Diagnosis
- The diagnosis of anal fissure is made on clinical examination, with a linear disruption of the anoderm on visual inspection.
- Digital rectal examination is very painful if an anal fissure is present, but should be attempted if the diagnosis is not clear, to rule out other possibilities for anal pathology. If the diagnosis is clear on visual evaluation, omitting the digital examination is advisable [14, 43].

Treatment
- Surgical treatment is almost never necessary [3, 7, 8, 16, 36, 43].
- The treatment for anal fissure should be aimed at interrupting the vicious cycle of painful bowel movements, subsequent reluctance to have bowel movements, and the resultant constipation [3, 8, 14].

The goal of easy daily bowel movements will allow for gradual healing of the fissure.
- Initial therapy usually involves dietary changes including increasing fiber to add bulk to bowel movements and decrease straining with defecation.
- Soaking in the bathtub or sitz baths (2-3 times a day) can provide some pain relief, and topical local anesthetic creams are sometimes also useful [3, 14, 16].

In the past, failure of initial medical management for anal fissures has resulted in surgical treatment such as internal anal sphincterotomy or anal dilations under anesthesia to relieve the hypertonicity of the anal sphincter and supposedly permit healing. These procedures are associated with a significant risk of
incontinence. More recently in adults, nitroglycerin ointment has been used to reduce anal sphincter hypertonicity and is gradually replacing anal sphincterotomy as the preferred treatment for anal fissure.

Prospective, blinded, placebo-controlled trials of treatment with 0.2% topical nitroglycerin ointment, local anesthetic cream, and placebo carried out in children have suggested that topical nitroglycerin ointment results in faster complete healing of the anal fissure and resolution of symptoms [14, 43].

Regardless of the treatment chosen, it will likely take several weeks for symptoms to subside, and this must be shared with the family at the outset of treatment.

HEMORRHOIDS

Etiology

Hemorrhoids in children are unusual [3, 8]. The incidence is less than 4%, with one-third of those affected requiring treatment [14].

Hemorrhoids are seen with increased frequency in children with portal hypertension and inflammatory bowel disease.

## Anal Disorders

**Clinical presentation**

The clinical presentation is variable with thrombosis occurring most frequently in teenagers. Externally, hemorrhoids are covered with squamous epithelium and are innervated by cutaneous nerves. Patients usually present with pain, bleeding, or a rectal mass.

Internally, hemorrhoids contain columnar epithelium and lack sensory innervation. Patients tend to complain of painless rectal bleeding, prolapse, or pruritus ani.

**Common symptoms of hemorrhoids include:**

- Painless bleeding during bowel movements
- Itching or irritation in the anal area
- Pain or discomfort
- Swelling around the anus
- Lump near the anus
Diagnosis
- Physical examination and history are usually adequate to establish the diagnosis.
- Digital rectal examination may identify polyps, masses, or areas of ulceration.
- Anoscopy is the study of choice to see and evaluate internal hemorrhoids.

Treatment
Conservative treatment alleviates the majority of symptoms [3, 7, 8, 16, 36, 43].
Common recommendations include:

- Hemorrhoid creams with lidocaine to help reduce pain
- Over-the-counter hemorrhoid cream with corticosteroid or anti-inflammatory creams
- Sitz baths (soak the anal area in plain warm water for 10 to 15 minutes 2-3 times a day)
- Avoid toilet paper (washing the anal area with warm water)
- Decreased straining and time spent on the toilet
- To prevent constipation:
  - Drink plenty of fluids daily
  - Eat a high-fiber foods such as fruits, vegetables and whole grains
  - Use stool softeners to prevent straining

In children with thrombosed hemorrhoids, therapy depends on timing of presentation. If seen within the first 48-72 hours of symptoms, incision and clot removal provides immediate relief from pain. After 72 hours, spontaneous resolution is underway. Rest, analgesics, stool softeners and sitz baths are then the treatment of choice [14, 16, 43].

For bleeding and pain that doesn’t stop, a minimally invasive procedure may be necessary:

- Rubber band ligation - small band is applied to the base of the hemorrhoid, stopping the blood supply to the hemorrhoidal mass. The hemorrhoid will shrink and die within a few days with shriveled hemorrhoidal tissue and band will falling off during normal bowel movements - likely without the patient noticing.
- Sclerotherapy - injection of a chemical to shrink the hemorrhoid
- Coagulation using laser or infrared light or heat

Hemorrhoidectomy (surgical hemorrhoid removal) may be necessary if these minimally invasive procedures are not successful.