Objective: to present an analysis of the occurrence of gastrointestinal bleeding in children and emphasize: i) diagnostic methods; ii) the organized use of different therapeutic approaches in upper gastrointestinal bleeding; iii) the review of concepts, classifications and techniques used in endoscopy, which are important to the practice of clinical pediatrics.

Methods: literary review of chapters selected from textbooks, pertinent articles obtained through the Medline system and active search, as well as personal archives belonging to the authors.

Results: the differential diagnosis of gastrointestinal bleeding in children varies according to the age. The causes of upper gastrointestinal bleeding are subdivided into variceal and nonvariceal. Nonselective beta-blockers are recommended to prevent variceal bleeding. Vasoactive drugs, such as somatostatin, octreotide, and glypressin may be used, showing good results in both variceal and nonvariceal acute bleeding. Both sclerotherapy and variceal ligation can be used in children to achieve variceal eradication. Cyanoacrylate is effective and presents the lowest complication rate related to gastric variceal bleeding. The presence of hemorrhage stigmata, such as active bleeding and visible vessel in ulcers is indicative of a higher risk for recurrent bleeding, suggesting the need for endoscopic hemostasis. Proton pump inhibitors are more efficacious than H2-receptor antagonists to promote the peptic ulcer healing.

Conclusion: the correct etiologic diagnosis of gastrointestinal bleeding in children is fundamentally important to adopt the adequate therapeutic approach, whose main advances concern the pharmacological and the endoscopic treatment.


Introduction
Gastrointestinal bleeding constitutes an important topic, once it is a medical emergency in every age group. It is still associated with expressive morbidity and mortality indexes, as well as with high-cost hospitalizations.

The discussion about gastrointestinal bleeding in children presents differences and similarities when compared to adults. The greatest discrepancy concerns the differential diagnosis. Some pathologies, especially the ones that reflect congenital malformations, such as intestinal duplication and Meckel’s diverticulum, are the most common in childhood, while neoplasias are seen more frequently among adults. Regardless of the clinical differences, the diagnostic and therapeutic approaches are usually similar to those used among adults.1
The development of the fibroendoscope, commercially available since 1960, has revolutionized the diagnostic approach for diseases of the gastrointestinal tract. In the 70s, the first esophagogastroduodenoscopies were performed in children, and, in 1989, Tam and Saing reported their 13-year experience in pediatric endoscopy, documenting the efficacy and safety of the procedure.

Parallelly to the advances obtained with endoscopic hemostasis techniques, there were important progresses concerning the pharmacological treatment of gastrointestinal bleeding, which is defined as blood loss originating in the gastrointestinal tract and adnexa. It may be manifested as:
- Hematemesis: indicates that the bleeding origin is above the Treitz angle, i.e., that it constitutes an upper gastrointestinal bleeding (UGIB);
- Melena: in 90% of the cases, it is associated with upper gastrointestinal bleeding, but it may be originated in the small intestine or in the proximal colon;
- Hemaatochezia or enterorrhagia: evacuations with bright red blood, generally originated in the colon, rectum or anus. However, upper bleeding, either extensive or associated with intestinal transit speed, may also be manifested this way;
- Occult blood in feces: reflects the blood loss through the feces, being macroscopically imperceptible. In general, these bleedings are originated in the small intestine or in the upper segments.

Differential diagnosis

Five factors provide important information for the etiologic diagnosis: age, location of the hemorrhagic site, color and severity of the bleeding, presence or absence of pain and diarrhea. Table 1 shows the main cases for gastrointestinal bleeding in childhood, correlating the different pathologies with the age group and with the presence of other signs and symptoms.

Generally, in childhood, lower gastrointestinal bleeding is more frequent, but it is usually less severe than the upper. This study will focus specially on diagnostic and therapeutic methods used in children with UGIB.

Diagnostic and therapeutic approach in children with UGIB

Initially, false episodes of gastrointestinal bleeding have to be excluded, for they may be caused by several factors, such as: deglutition of breast milk, epistaxis, hemoptysis, earlier use of drugs and foods that may color the feces, such as iron, bismuth, berries, chocolate, beet, among others. This way, the performance of invasive and unnecessary procedures is avoided.

The diagnostic and therapeutic approach of the child with UGIB may be divided into three stages:
- Stage I: general evaluation of the patient and hemodynamic stabilization;
- Stage II: etiologic diagnosis;
- Stage III: specific treatment.

Stage I: General evaluation of the patient and hemodynamic stabilization

In this phase, the pediatrician in charge of the patient should rapidly evaluate three items: the permeability of the upper airways; the existence of active bleeding (intensity); the patient’s hemodynamic conditions. Venous access, resuscitation, adequate ventilation and control of pulse and arterial pressure are essential for the patient’s good evolution.

The estimated blood loss is obtained through the assessment of the exteriorized loss, of the arterial pressure, pulse and hematocrit. However, the initial value of the hematocrit may be deceiving, since it is only after 24 to 72 hours, with the reestablishment of the vascular space, that the hematocrit reflects the actual loss volume.

The observation of the volume and characteristics of the drained material through a nasogastric catheter provides information about the density of the bleeding. Besides monitoring the loss, the drainage through a nasogastric catheter promotes the gastric content cleaning. This way, it eases the endoscopist’s work and decreases the risk for aspiration of gastric content. For the performance of the gastric lavage, the use of common water or physiological serum at room temperature is recommended.

Stage II: Etiologic diagnosis

In this phase, the attention is directed to the elucidation of the etiologic diagnosis. UGIB is a symptom of digestive problems, and not a disease by itself. It may be the result of several heterogeneous affections, with different therapeutic peculiarities. The differential diagnosis of UGIB in childhood is showed in Table 2.

The following steps should be followed for the elucidation of the etiologic diagnosis:

a) Clinical history

The complete and detailed clinical history is greatly important, and does not have laboratorial substitutes. Chronic pain, located (epigastric) or associated with clocking (to wake up at night with pain), suggests the presence of peptic disease. Hemorrhagic vomits and acute abdominal pain after repetitive, initially non-hemorrhagic vomits suggest diagnosis of Mallory-Weiss’s syndrome. Previous history of gastroesophageal reflux corroborates the possibility of complicated esophagitis. Other important data are: previous history of umbilical catheterism, blood transfusions and use of drugs, especially steroidal and nonsteroidal anti-inflammatories.
b) Physical examination

After the general evaluation of the patient and the hemodynamic stabilization, a detailed clinical examination should be carried out. The evaluation of vomit and evacuation characteristics should be part of the physical examination of UGIB patients. Some findings are important to the final conclusion of the etiologic diagnosis: the presence of aphthae suggests diagnosis of Crohn’s disease; the presence of splenomegaly, spiders, ascites and hard consistency of the liver are compatible with the diagnosis of portal hypertension; the presence of ecchymoses in the lower members suggests Henoch-Schönlein purpura; torticollis is part of the Sandifer syndrome and is associated with reflux esophagitis, among others.

c) Upper digestive endoscopy

The patient should be referred to endoscopic evaluation after the hemodynamic and respiratory stabilization, preferably in the first 12 hours after the hemorrhagic episode, since the diagnostic index is higher, up to 95%, in endoscopies performed early. Patients with solid losses, who continue with active bleeding and hemodynamic...
instability even after reposition should be submitted to endoscopic evaluation immediately, concomitantly with the resuscitation and hemodynamic stabilization procedures, and preferably in a intensive therapy unit. It is important to remember that 70% to 80% of the patients present self-limited bleeding.

Upper digestive endoscopy acts in three stages: on the diagnosis, on the prognosis and during the treatment.

**Diagnosis:** upper digestive endoscopy is better than radiographic studies to the localization of hemorrhagic sites.

**Prognosis:** hemorrhage stigmata, defined at Forrest’s classification, were originally described more than two decades ago and are worldwide accepted still in the present days. They provide information about the prognosis, since they present a correlation with the hemorrhagic recidivism index. This way, they orient the endoscopist and the pediatrician in terms of the adequate therapeutic conduct, hospitalization time and necessary fasting time. Forrest’s classification is specified in Table 3, and the correlation between the bleeding stigmata and the rebleeding indexes is shown in Table 4.

### Table 2 - UGIB main etiologies in childhood

<table>
<thead>
<tr>
<th>Variceal UGIB</th>
<th>Nonvariceal UGIB</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Esophagus</strong></td>
<td><em>PHG - Portal hypertensive gastropathy</em></td>
</tr>
<tr>
<td><em>PHG</em> - Portal hypertensive gastropathy</td>
<td></td>
</tr>
<tr>
<td><strong>Stomach</strong></td>
<td>Gastric varices</td>
</tr>
<tr>
<td>Gastric varices</td>
<td></td>
</tr>
<tr>
<td><strong>Duodenum</strong></td>
<td>Duodenal varices</td>
</tr>
<tr>
<td>Duodenal varices</td>
<td></td>
</tr>
<tr>
<td><strong>Variable location</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Table 3 - Forrest’s classification**

<table>
<thead>
<tr>
<th>I. Active hemorrhage</th>
<th>Nonbleeding bleeding (jet)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ib. Slow bleeding (dribbling)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>II. Recent hemorrhage</th>
<th>Nonbleeding visible vessel</th>
</tr>
</thead>
<tbody>
<tr>
<td>IIA. Adherent clot on the base of the lesion</td>
<td></td>
</tr>
<tr>
<td>IIc. Flat pigmented spots</td>
<td></td>
</tr>
</tbody>
</table>

| III. No evidence of bleeding (clean base) |

Source: Forrest

### Table 4 - Frequency of endoscopic stigmata and of rebleeding incidence

<table>
<thead>
<tr>
<th>Stigmata</th>
<th>Incidence</th>
<th>Rebleeding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bleeding in jet</td>
<td>8 to 15%</td>
<td>&gt;90%</td>
</tr>
<tr>
<td>Visible vessel (red)</td>
<td>26 to 55%</td>
<td>30 to 51%</td>
</tr>
<tr>
<td>Adherent clot</td>
<td>10 to 18%</td>
<td>25 to 41%</td>
</tr>
<tr>
<td>Slow bleeding</td>
<td>10 to 20%</td>
<td>20 to 30%</td>
</tr>
<tr>
<td>Flat clot</td>
<td>12%</td>
<td>0 to 5%</td>
</tr>
<tr>
<td>Clean base</td>
<td>36%</td>
<td>0 to 2%</td>
</tr>
</tbody>
</table>

Source: Luna

Treatment: therapeutic upper digestive endoscopy presented important advancements in the 80s, and it is considered the first option in hemostatic treatment.

d) Other diagnostic methods

These other methods may also be useful for the diagnostic elucidation:

- Intestinal transit, especially when there is a clinical suspicion of Crohn’s disease;
- Abdominal scintigraphy, examination of choice for the identification of Meckel’s diverticulum;
- Arteriography;
- Explorative laparotomy.

**Stage III: Specific treatment**

The different affections that may go along with UGIB in childhood, listed in Table 2, require specific treatments.
Variceal hemorrhage

Bleeding resulting from the rupture of gastroesophageal varices is the most serious complication of portal hypertension, and it is responsible for 10 to 15% of UGIB in childhood. The different therapeutic options used for the hemostasis of variceal UGIB are listed in Table 5 and will be discussed below.

Pharmacological treatment

Prophylactic treatment

Propranolol is used as a prophylactic medicine for the prevention of either the first hemorrhagic episode or the hemorrhagic recidivism. In general, it is well tolerated by the pediatric population, with minimal side effects. The dosage should be adjusted so that it reduces the original cardiac frequency at 25%. It is contraindicated for asthmatic patients and for those with cardiac blockade.

Treatment of the acute phase

Vasoactive drugs, such as vasopressin, somatostatin, octreotide and glypressin (terlipressin) are effective in controlling variceal hemorrhage. Vasopressin, widely used in the past, has been substituted for somatostatin or for its synthetic analog, octreotide, which, for acting in a selective way in the splanchnic vasoconstriction, present high efficacy in the control of acute bleeding and minor side effects. Octreotide presents a longer average life than somatostatin. The preconized dosages are shown in Table 6. The octreotide infusion should be maintained until the control of the bleeding, for 48 hours on average, and the dosage reduction should be started 24 hours after the bleeding interruption. The weaning should be progressive, reducing half a dosage every 12 hours. Higher dosages may be used in selected cases, and the increase should be progressive (every 8 hours). Side effects are more frequent in high-dosage infusions. During the use of the drug, even during weaning, glucemia should be controlled. Since octreotide presents a longer average life, it may be administrated subcutaneously every 8 hours. In the department of Pediatric Gastroenterology, at Hospital de Base do Distrito Federal (Brasília, DF, Brazil), we started the continuous infusion with 0.25 mg/kg/hour, and increased it every 2 hours, progressively (0.25 mg/kg/hour, 0.50 mg/kg/hour, 0.75 mg/kg/hour, 1.00 mg/kg/hour). This scheme is also used by the Pediatric Gastroenterology and Nutrition Division, at the Children’s Hospital Medical Center, Cincinnati, EUA.

Endoscopic treatment

Endoscopic hemostasis is indicated for the control of acute hemorrhage and also for the prevention of hemorrhagic recidivism.

Sclerotherapy is not usually indicated for the prevention of the first episode of variceal hemorrhage. However, a study performed by the North Italian Endoscopic Club (NIEC) established prognostic criteria for the risk for bleeding, with an estimated variation of 6.8 to 68.9%. The three variables with prognostic significance were: Child’s classification, the caliber of the varices, and the presence of red stains on their surface. Based on these criteria, some authors preconize the prophylactic endoscopic treatment for the obliteration of esophageal varices in patients with a high risk for bleeding. Although this index is not ideal for the pediatric population, it is still the most accepted one.

Endoscopic sclerotherapy of esophagogastric varices

In the present days, several types of sclerosants are available, and the ethanolamine oleate is the most commonly used for the sclerosis of esophageal varices, with intra and paravasal injections, as shown in Figure 1. Esophageal varices are more common and present a better response to sclerotherapy. Gastric varices present a higher caliber, bleed more abundantly and are associated with a higher mortality rate. It is worthwhile to stand out that the control of hemorrhage resulting from the rupture of gastric varices is more efficacious and associated with a lower index of complications when cyanoacrylate is used.

In children, the sclerotherapy of esophageal varices is greatly successfully, presenting low incidence of complications and low mortality rate. It is usually considered the first option in hemostatic treatment.

Elastic ligature

The elastic ligature of esophageal varices (Figure 2) uses the same technique of the treatment of hemorrhoids, and it was proposed as an alternative for the eradication of

<table>
<thead>
<tr>
<th>Table 5 - Therapeutic methods used in variceal UGIB</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pharmacetics</td>
</tr>
<tr>
<td>Propanolol</td>
</tr>
<tr>
<td>Vasopressin</td>
</tr>
<tr>
<td>Somatostatin</td>
</tr>
<tr>
<td>Octreotide</td>
</tr>
<tr>
<td>Glypressin</td>
</tr>
</tbody>
</table>

*TIPS - Transjugular intrahepatic portosystemic shunt
esophageal varices in 1986. Its utilization was more accepted after the development of multiple league applicators, which allow the collocation of up to 10 leagues in one only passage of the endoscope. Good results are obtained with the ligature in children.

Cost and availability
Sclerotherapy is the most used technique, since it offers easy execution, low cost and availability in several centers. The elastic ligature is associated with a lower index of complications, but its availability is more restrict, and its cost is more elevated.

Independently on the method used, the long-term survival depends especially on the hepatopathy stage, that is, on the hepatocellular dysfunction level. In children presenting portal hypertension without hepatocellular dysfunction (example: thrombosis of the portal vein), there is a tendency of reduction of the bleeding after adolescence, as a consequence of the recanalization of the portal vein and of the development of collateral portosystemic in other areas.

Additional measures
After the sclerotherapy sessions, the use of H2 blockers or sucralfate is indicated, aiming to decrease the incidence of complications, such as ulcers and stenoses. Antibiotics are indicated for the prophylaxis of bacterial endocarditis in risk patients. The use of omeprazole promotes the efficacious cicatrization of the post-sclerosis ulcer.

Combined pharmacological and endoscopic treatment
Vasoactive drugs, indicated for the treatment of the acute phase, may be administrated in UGIB cases right after the patient’s admission, still in the emergency room, even before the endoscopic evaluation. The co-operating treatment (endoscopic and pharmacological) is more effective. The decrease of the hemorrhagic flux eases the visualization of the lesion, favors the endoscopic treatment, reduces the need for blood transfusion, and decreases the risk for aspiration of hemorrhagic gastric content during endoscopy.

Mechanical treatment: Sengstaken-Blakemore balloon
Indications
Still in the present days, the temporary tamponade is useful. It is obtained with the installation of the Sengstaken-Blakemore balloon, shown in Figure 3. The main indications are: failure of the endoscopic treatment or impossibility of visualization at the bleeding area (severe bleeding).

Installation
After the introduction of the balloon catheter, certify that the gastric balloon is placed with one end in the stomach. Then, the partial insufflation of the gastric probe should be done, and right after this, a radiograph of the upper abdomen should be performed, in order to confirm the position of the gastric balloon (below the diaphragm). If the gastric balloon is well placed, its insufflation should be completed, and the balloon catheter should be pulled and fixed, so that the gastric balloon gets adjusted to the gastroesophageal junction. Then, the esophageal probe may be insufflated in order to maintain the pressure at 30 to 40 mmHg. The cleanness of the gastric cavity and the intermittent aspiration of secretions from the hypopharynx and esophagus are important to avoid aspiration. The esophageal balloon should not remain insufflated for more than 24 hours, due to the risk for ischemia of the esophageal mucosa. After the fixation of the gastric balloon, it is possible to leave it only insufflated for a period of 4 to 6 hours, since only this measure may be sufficient for the hemorrhage control, with the decrease of the flux to the

Figure 1 - Endoscopic sclerotherapy of esophageal varices. A. Intravasal injection. B. Paravasal injection
Source: Terblanche

Figure 2 - Scheme of elastic ligatures in esophageal varices
Source: Stiegmann
esophageal varices. The use of the balloon in children usually requires sedation.\textsuperscript{29,40,44}

Withdrawal

The probe is removed after the balloon deinsufflation; initially, the esophageal balloon is deflated, and 24 hours later, the gastric one.\textsuperscript{29,44}

Results and complications

The use of the balloon is effective in the control of acute hemorrhage, with good results in children as well\textsuperscript{40}. The main limitations of its use are: high index of complications, especially lesions of the esophageal mucosa and acute respiratory insufficiency, due to the aspiration or even migration of the balloon. Besides, it does not act in the prevention of rebleeding.\textsuperscript{29,44}

Transjugular intrahepatic portosystemic shunt (TIPS)

The TIPS is a percutaneous, non-surgical shunt. A prosthesis, placed through the hepatic parenchyma, unites the hepatic vein and the portal vein, as shown in Figure 4. The TIPS are effective in the control of the variceal hemorrhage, even in case of gastric varices.\textsuperscript{46} However, they are associated with an increased risk for encephalo-pathy.\textsuperscript{47} TIPS may be used in children, with good results, for the treatment of the portal hypertension complications.\textsuperscript{48}

Surgery

The surgical treatment is indicated in cases of failure of the already mentioned therapeutic alternatives. The surgical options are: portosystemic shunts (nonselective and selective), transection and esophageal devascularization, and hepatic transplantation.\textsuperscript{49}
Nonvariceal hemorrhage

Ulcers

Ulcers are classified according to their location (esophageal, gastric or duodenal) and etiology (primary or secondary). Secondary ulcers, associated with systemic diseases or ulcerogenic drugs, are usually silent until the arousal of complications, such as UGIB, with a higher index of morbidity and mortality than in primary ulcers.

Deep gastric ulcers, located in the lesser curvature, and duodenal ulcers, located at the posterior-inferior wall of the bulb, present a higher risk for massive bleeding, due to their proximity with the large vessels.

The different therapeutic options used for nonvariceal UGIB hemostasis are now going to be discussed.

Pharmacological treatment

The use of proton pump blockers, indicated in the treatment of hemorrhagic ulcer, is associated with the decrease of the rebleeding rate, of the need for blood transfusion, as well as of the surgical treatment. It should be associated with endoscopic hemostasis in cases of active bleeding. The omeprazole dosage for children is described in Table 6. Vasoactive drugs, such as somatostatin and octreotide, are efficacious in the control of nonvariceal digestive bleeding.

Endoscopic treatment

The need for endoscopic hemostasis in hemorrhagic ulcerous lesions depends on the aspect of the ulcer base, since the bleeding stigmata are especially important for the prediction of the hemorrhagic recidivism, as shown in Table 4. The hemostatic endoscopic treatment is indicated in lesions that present endoscopic signs associated with a high index of hemorrhagic recidivism, such as active bleeding and visible vessel. Hemostasis is not indicated in ulcers with a clean base or flat pigmented spots, due to the low frequency of rebleeding. The conduct in lesions with an adherent clot is no longer a consensus in literature, since the removal of the clot may cause digestive bleeding again. In services where thermal methods are not available, an injection of adrenaline should be carried out on the base of the lesion, and then, the clot should be removed, and the hemostatic therapy should be performed according to the aspect of the base of the lesion.

### Table 6 - Drugs used in the treatment of the digestive hemorrhage

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ranitidina</td>
<td>4 a 6 mg/kg/dia, 2x/dia, VO*/EV†</td>
</tr>
<tr>
<td>(Rodgers²³)</td>
<td>Infusão contínua (EV†): 0,10 a 0,25 mg/kg/hora</td>
</tr>
<tr>
<td>Ranitidine</td>
<td>4 to 6 mg/kg/day, twice a day, O*/E†</td>
</tr>
<tr>
<td>Rodgers²³</td>
<td>Continuous infusion (E†): 0.10 to 0.25 mg/kg/hour</td>
</tr>
<tr>
<td>Omeprazole</td>
<td>0.7 to 3.3 mg/kg/day, O*/EV†</td>
</tr>
<tr>
<td>Israel⁵³</td>
<td></td>
</tr>
<tr>
<td>Propanolol</td>
<td>1 mg/kg/day, three times a day, O*</td>
</tr>
<tr>
<td>Shashidhar¹⁹</td>
<td>(progressive increase up to ↓ 25% of the original CF†)</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>Bolus (E†): 0.33 U/kg (20 minutes)</td>
</tr>
<tr>
<td>Mowat²⁴</td>
<td>Infusion (E†): 0.33 U/kg/hour</td>
</tr>
<tr>
<td>Somatostatin</td>
<td>Bolus (E†): 1 to 2 µg/kg (2 to 5 minutes)</td>
</tr>
<tr>
<td>Rodgers²³</td>
<td>Infusion (E†): 1 to 2 µg/kg/hour</td>
</tr>
<tr>
<td>Octreotide</td>
<td>Teenagers and adults:</td>
</tr>
<tr>
<td>Siafakas²²</td>
<td>Bolus (E†): 50 mg (5 minutes)</td>
</tr>
<tr>
<td></td>
<td>Infusion (E†): 50 µg/hour</td>
</tr>
<tr>
<td></td>
<td>Younger children:</td>
</tr>
<tr>
<td></td>
<td>Bolus (E†): 1 µg/kg (5 minutes)</td>
</tr>
<tr>
<td></td>
<td>Infusion (E†): 1 µg/kg/hour</td>
</tr>
</tbody>
</table>

* Orally † Intravenously ‡ Cardiac frequency

### Table 7 - Therapeutic methods used in nonvariceal UGIB

<table>
<thead>
<tr>
<th>Injection</th>
<th>Nonthermal</th>
<th>Thermal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenaline</td>
<td>Mechanical</td>
<td>Thermal</td>
</tr>
<tr>
<td>- Pure (1:10,000</td>
<td>Elastic ligature</td>
<td>With contact</td>
</tr>
<tr>
<td>or 20,000)</td>
<td>Hemoclip</td>
<td>Coagulation factors</td>
</tr>
<tr>
<td>- With NaCl</td>
<td>Sutures</td>
<td>Vasoconstrictors</td>
</tr>
<tr>
<td>- With sclerosants</td>
<td>Balloon</td>
<td></td>
</tr>
<tr>
<td>- Associated with</td>
<td>Endoloop</td>
<td></td>
</tr>
<tr>
<td>thermal methods</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absolute alcohol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ethanolamine (1 to</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Polidocanol (1%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thrombin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fibrin glue</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cyanoacrylate</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
The several modalities of endoscopic hemostasis available in the present days for the control on nonvariceal hemorrhage are shown in Table 7. The injection therapy is still the most commonly used, since it offers efficacy, low cost, easy transportation, and handling.55,56

Dismissal
Patients that carry lesions associated with a low risk for rebleeding may be dismissed on the same day, while high-risk patients should stay at the hospital for at least 72 hours, period in which the hemorrhagic recidivism is more frequent.57

Fasting
Fasting should be maintained for at least 48 hours in children with a high risk for hemorrhagic recidivism, because of the reasons mentioned before.58

Esophagitis
Hemorrhage is considered a complication of esophagitis, which in childhood is usually associated with gastroesophageal reflux (GER). So, in most cases, the treatment should be directed both to esophagitis and GER. In hemorrhagic esophagitis, proton-pump blockers are more efficacious than H2-blockers to promote the lesion cicatrization.53

Mallory-Weiss syndrome
In the Mallory-Weiss syndrome, the UGIB results from the laceration of the esophageal mucosa. The clinical treatment includes: volemic replacement, antacids, antiemetics, and, if necessary, blood transfusion. Endoscopic hemostasis is indicated in cases of active bleeding or visible vessel close to the laceration. Other therapeutic options are: vasoconstrictor pharmaceutics, such as octreotide; embolization of the left gastric artery; or surgical correction (gastronomy with suture of the fissure or fundoplication).59

Gastritis and gastropathy
Different types of gastritis and gastropathy may accompany UGIB in childhood. The classification of gastritis through Sydney’s system,60 used in adults, has a limited value for the pediatric population, since it focuses especially on the severity of chronic gastritis, atrophy, and intestinal metaplasia, more common in adults. It does not classify noninflammatory lesions either. A proposal to classify gastritis and gastropathy in children was published in 1999,61 and is described in Table 8.

In the section of Pediatric Gastroenterology, at Hospital de Base do Distrito Federal, we indicate the use of pump blockers in the treatment of hemorrhagic gastritis. However, H2 blockers may also be used. In the presence of Crohn disease, celiac disease, and others, specific measures should be instituted.

<table>
<thead>
<tr>
<th>Table 8 - Classification of the gastrites and gastropathies in children</th>
</tr>
</thead>
<tbody>
<tr>
<td>Erosive and/or hemorrhagic gastrites or gastropathies</td>
</tr>
<tr>
<td>Stress gastropathy</td>
</tr>
<tr>
<td>Neonatal gastropathy</td>
</tr>
<tr>
<td>Traumatic gastropathy</td>
</tr>
<tr>
<td>Gastropathy due to aspirin and other NSAI* drugs</td>
</tr>
<tr>
<td>Portal hypertensive gastropathy</td>
</tr>
<tr>
<td>Uremic gastropathy</td>
</tr>
<tr>
<td>Varioiliform chronic gastritis</td>
</tr>
<tr>
<td>Biliary gastropathy</td>
</tr>
<tr>
<td>Gastropathy: Henoch-Schönlein purpura</td>
</tr>
<tr>
<td>Corrosive gastropathy</td>
</tr>
<tr>
<td>Gastropathy or exercise gastritis</td>
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<tr>
<td>Radiation gastropathy</td>
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<tr>
<td>Other granulomatous gastrites</td>
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<td>Other infectious gastrites</td>
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* NSA - Nonsteroidal anti-inflammatory  Source: Dohil61
Portal hypertensive gastropathy

Portal hypertensive gastropathy is responsible for 10 to 50% of the UGIB cases in patients with portal hypertension. The classic therapy for gastritis does not improve the bleeding significantly. During the acute phase of the bleeding, vasoconstrictor drugs, such as somatostatin, octreotide, or glypressin, are indicated. Propanolol, as a prophylactic, is also indicated.63

Dieulafoy lesion

The Dieulafoy lesion deserves to be mentioned, even though it is not one of the most common causes of UGIB in childhood, since it is responsible for episodes of massive hemorrhage. Early upper digestive endoscopy favors the etiologic diagnosis. The bleeding occurs through a punctiform, nonulcerous lesion, usually located at the upper part of the stomach fundus, corresponding to rupture of the caliber artery, which goes in an anomalous way up to the submucosa, probably consisting of a congenital variation. The methods of endoscopic hemostasis already described may be used. Surgical treatment is indicated only in refractory cases.59,64

Duodenitis

Duodenitis may be:
- peptic;
- parasitic (Ascaris lumbricoides, Ancylostoma duodenale, Giardia lamblia, Strongyloides stercoralis and Schistosoma mansoni);
- associated with Crohn disease;
- associated with granulomatous diseases (tuberculosis);
- associated with the immunoproliferative small intestine disease.

For the etiologic diagnosis, the description of the macroscopic aspect and the collection of material are important for the study: duodenal liquid for the investigation of larvae, and biopsy for histological and histochemical studies.65 The treatment of hemorrhagic duodinitis is similar to that described for hemorrhagic gastritis.

Prevention of UGIB in childhood

It is important to avoid the abusive use of anti-inflammatories, frequently used without a precise indication. Up to the present moment, there are no evidences that the nonhormonal anti-inflammatories reduces the inflammatory process when associated with acute respiratory diseases. They are the pharmaceutics most frequently used in the world, generating an excessive index of hospitalization, high cost, and important rates of morbidity and mortality.66-68

The treatment of H. pylori in patients that carry primary gastric or duodenal ulcers prevents rebleeding.52,69

Another preventive measure is the performance of elucidation campaigns, in order to prevent the abusive use of alcohol among teenagers.

Finally, regarding the prophylaxis of stress ulcers, respect to the serious patient’s pain and stress is important. At this point, special scope should be given to humanization in pediatric intensive therapies, as well as to analgesia and gastric alkalinization. The use of sucralfate or ranitidine is efficacious for the prevention of UGIB resulting from stress gastropathy.62,70,71

References


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