CHAPTER 153 APPEARANCE TO LOWER GASTROINTESTINAL BLEEDING
HSIU-PO WANG

IMMEDIATE CONCERNS

Lower gastrointestinal (LGI) bleeding (LGIB) may be the primary cause of admission to the intensive care unit (ICU) or occurs during the course of ICU care for disorders other than LGIB. The initial approach to patients with LGIB, whether occult or acutely overt, is resuscitation to maintain organ perfusion. ICU physicians must maintain adequacy of airway and circulation, including ensuring an open airway and adequate intravenous access. Thereafter, the severity of bleeding should be graded to determine whether urgent diagnostic and interventional procedures need be initiated immediately. The consequences of LGIB should be prevented or treated, just as would an acute myocardial infarction (AMI) induced by anemia or unstable hemodynamic status. A summary approach to ICU patients is noted in Table 153.1 and Figure 153.1.

Evaluation

1. Is there true gastrointestinal (GI) bleeding (for suspicious cases with anemia in the ICU)?
2. Is the bleeding from the lower or upper GI tract?
3. Are there any adverse consequences caused by the LGIB?
4. What are the effective diagnostic and interventional tools to use for the workup of patients with LGIB?

Essential Diagnostic Tests and Procedures

1. History taking, including previous operative interventions and medications
2. Physical examination, including digital examination
3. Stool guaiac for anemic patients with suspicious GI blood loss
4. Complete blood count
5. Endoscopic procedures: sigmoidoscopy, colonoscopy, enteroscopy, and capsule endoscopy
6. Barium study
7. Bedside ultrasound (US)
8. Radiologic procedures: angiography
9. Nucellar scintigraphy: technetium-labeled red blood cell scan
10. Other examinations for affected organs due to unstable hemodynamics, such as electrocardiogram (EKG) for AMI

Treatment

1. Ensure stable hemodynamic status, using fluids and transfusion of blood components.
2. Endoscopic therapies include local injection, electrocoagulation, hemoclipping, argon plasma coagulation, and elastic banding according to the nature of the bleeding.
3. Radiologic interventions include angiography with intraarterial vasopressin infusion, embolization, and glue.
4. The adverse events caused by treatment modalities, such as perforation caused by endoscopic thermocoagulation or bowel infarction due to angiographic embolization, should be monitored and managed.
5. Surgical consultation is warranted for severe LGIB in which the patient cannot be resuscitated, or if all available therapeutic modalities fail.

OVERVIEW

Gastrointestinal diseases are often encountered in the ICU setting, either as the major cause of admission to the ICU or as a comorbid complication of another primary disease process. The consequences of LGIB in the ICU—anemia and hypovolemia—may delay the weaning or extubation of patients, which can prolong the ICU course. LGIB is defined as a bleeding source distal to the ligament of Treitz, thus involving the small bowel and colon, and accounts for an estimated 20% to 24% of all major GI bleeding (1,2). It has been estimated that the annual incidence of LGIB is approximately 0.03% in the adult population (3). Longstreth estimated the annual incidence of hospitalization for LGIB to be 20 to 30 cases per 100,000 persons (4). LGIB is more common in men than women, and the incidence increases with age, with a greater than 200-fold increase from the third to ninth decades of life (2). The true incidence of LGIB during ICU hospitalization is not precisely clear; the incidence for ICU patients with acquired hemorrhage (not primarily due to LGIB) was reported as 0.94% (5).

LGIB is clinically distinct from upper gastrointestinal (UGI) bleeding (UGIB) in epidemiology, management, and outcome. LGIB is approximately 20% to 33% as common as UGIB (2,6,7). LGIB generally has a lower mortality rate than does UGIB (8), but mortality is markedly higher in patients who begin bleeding after admission: 2.4% versus 23% (4). Most deaths are not the direct result of uncontrolled bleeding, but rather exacerbation of an underlying disorder or development of a nosocomial complication. Lin et al. (5) noted a 53%
TABLE 153.1  SUMMARY OF AN APPROACH TO ICU PATIENTS WITH LOWER GASTROINTESTINAL BLEEDING

1. Immediately assess and stabilize the patient's hemodynamic status
   The ABCs of resuscitation
2. Determine presence of lower gastrointestinal bleeding
   By history, physical examination, and sometimes nasogastric aspiration
3. Arrange appropriate diagnostic and therapeutic interventions to stop any active bleeding
4. Treat any underlying lesions, and monitor and manage the comorbid illness

ICU, intensive care unit.

mortality in 55 patients, but this outcome was attributable to LGIB in only two patients. In another study, Lin et al. noted that patients with LGIB and comorbid illness had a higher mortality rate than those without: 29.5% versus 4.3% (9). No matter whether reported in the general population or in an ICU-based study, LGIB remains a difficult diagnostic and treatment problem for several reasons:

1. Bleeding can originate from any part of the lower GI tract.
2. Blood loss is often intermittent in nature, and it is difficult to identify the source in the absence of active bleeding, especially angiographically.
3. The colon preparation before urgent colonoscopy is, obviously, needed but often incomplete (10).
4. Recurrent bleeding due to angiodysplasia (7) or diverticula (8,11) may be seen with LGIB.
5. Unlike UGIB, there are no evidence-based and effective pharmacologic therapies for LGIB.

Among the many causes of LGIB (Table 153.2), diverticular bleeding, angiodysplasia, colitis, and neoplasm have been reported to be the most frequent (4,12). While the data from most reports are mixed with both non-ICU and ICU patients, the spectrum of LGIB from ICU patients should be different from others. Data from a study limited to medical ICU patients have shown ischemic colitis and acute hemorrhagic rectal ulcers to be the most frequent causes of LGIB, followed by colitis and diverticular bleeding (5). Our unpublished data—from surgical, trauma, and medical ICUs—showed acute hemorrhagic rectal ulcers followed by ischemic colitis to be the most frequently encountered causes (Table 153.3).

Patient age is a very important factor in the differential diagnosis of GI bleeding. Patients younger than 40 years are more likely to suffer from small bowel tumors, such as lymphomas, carcinoid tumors, and adenocarcinomas; anatomic anomalies such as Meckel diverticulum and Dieulafoy lesions; genetic problems such as polyps from a hereditary polyposis syndrome; or Crohn disease and ulcerative colitis, which are common in Western countries and, recently, increasing in Asia. Patients older than 40 years are more prone to bleeding from vascular lesions and neoplasm (12). Lewis et al., while evaluating small bowel bleeding, noted that in patients between 30 and 50 years, tumors were the most common abnormalities; in patients younger than 23 years, Meckel diverticulum was the most common source of small bowel bleeding, whereas vascular ectasias predominated in the elderly (13).

Presentation

Specific groups may have specific causes of LGIB. In the immunosuppressed patient, such as those with human immunodeficiency virus (HIV) infection and renal or pancreatic transplant patients, LGIB is often caused by cytomegalovirus (CMV) ulcers. Renal failure is a well-known risk factor for angiodysplasia (14); this is also prone to occur in patients with aortic valvular stenosis (15). Radiation colitis should be considered...
Chapter 153: Approach to Lower Gastrointestinal Bleeding

### TABLE 153.2

<table>
<thead>
<tr>
<th>Causes</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute hemorrhagic rectal ulcer syndrome (AHRUS)</td>
<td>26.7</td>
</tr>
<tr>
<td>Ischemic colitis</td>
<td>17.1</td>
</tr>
<tr>
<td>Colitis other than ischemia</td>
<td>8.4</td>
</tr>
<tr>
<td>Diverticular bleeding</td>
<td>6.7</td>
</tr>
<tr>
<td>Malignancy</td>
<td>5.7</td>
</tr>
<tr>
<td>Colonic polyp</td>
<td>3.8</td>
</tr>
<tr>
<td>Solitary ulcer</td>
<td>3.8</td>
</tr>
<tr>
<td>Hemorrhoid</td>
<td>2.9</td>
</tr>
<tr>
<td>Dieulafoy lesions</td>
<td>1.9</td>
</tr>
<tr>
<td>Radiation colitis</td>
<td>1</td>
</tr>
<tr>
<td>Small bowel bleeding</td>
<td>6.7</td>
</tr>
<tr>
<td>Undetermined</td>
<td>9.4</td>
</tr>
</tbody>
</table>

**COLONIC SOURCES**
- Vascular ectasia
- Diverticulosis
- Ischemic colitis
- Acute hemorrhagic rectal ulcer syndrome
- Neoplasia
- Postpolypectomy
- Inflammatory bowel disease
- Infectious colitis and ulcer
- NSAID-induced colopathy
- Radiation colitis
- Hemorrhoids
- Dieulafoy lesions
- Colon varices
- Aortoenteric fistula

**SMALL BOWEL SOURCES**
- Vascular ectasia
- Focal active bleeding small bowel tumor:
  - Lymphoma
  - Adenocarcinoma
  - GIST
  - Other tumors
- NSAID-induced enteropathy
- Crohn disease
- Meckel diverticulum
- Vascularitis: SLE, Behçet disease, Schönlein-Henoch purpura
- Infection related ulcer: CMV, and so forth
- Small bowel varices
- Aortoenteric fistula

**ALL JPG**

### CAUSES OF LOWER GASTROINTESTINAL BLEEDING IN SURGICAL, TRAUMA, AND MEDICAL ICUS

<table>
<thead>
<tr>
<th>Causes</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute hemorrhagic rectal ulcer syndrome (AHRUS)</td>
<td>26.7</td>
</tr>
<tr>
<td>Ischemic colitis</td>
<td>17.1</td>
</tr>
<tr>
<td>Colitis other than ischemia</td>
<td>8.4</td>
</tr>
<tr>
<td>Diverticular bleeding</td>
<td>6.7</td>
</tr>
<tr>
<td>Malignancy</td>
<td>5.7</td>
</tr>
<tr>
<td>Colonic polyp</td>
<td>3.8</td>
</tr>
<tr>
<td>Solitary ulcer</td>
<td>3.8</td>
</tr>
<tr>
<td>Hemorrhoid</td>
<td>2.9</td>
</tr>
<tr>
<td>Dieulafoy lesions</td>
<td>1.9</td>
</tr>
<tr>
<td>Radiation colitis</td>
<td>1</td>
</tr>
<tr>
<td>Small bowel bleeding</td>
<td>6.7</td>
</tr>
<tr>
<td>Undetermined</td>
<td>9.4</td>
</tr>
</tbody>
</table>


in patients with LGB and a history of radiation therapy for cervical or prostate malignancy. Although some lesions (i.e., diverticula) are common in the left colon, most bleeding sites are noted in the right colon. In approximately 76% of patients, a definitive site of bleeding will be diagnosed (16). Bleeding in most patients with LGB will stop spontaneously, although continued or recurrent bleeding during an acute episode occurs in 10% to 40% of patients (17–19). Finally, 2% to 15% of patients with presumed LGB will have UGIB (20); the cause of overt GI bleeding remains undetermined in 4% to 15% of cases, even after upper GI endoscopy and colonoscopy (21).

LGI B may present in multiple ways, including occult fecal blood, iron deficiency anemia; melena; intermittent scant hematochezia; or acute, massive, and overt bleeding (22). Patients with chronic LGB may only present with anemia, whereas those with acute LGB may complain of passing bright red blood per rectum, dark blood with clots, or, less commonly, melena. Patients with brown or infrequent stools are unlikely to have brisk bleeding, and those with frequent passage of red or maroon stool may have aggressive ongoing bleeding (23). Pallor, fatigue, chest pain, palpitations, dyspepsia, tachypnea, tachycardia, posture-related dizziness, and syncope are suggestive of hemodynamic compromise and demand aggressive care. The severity of LGB may be underestimated due to compensatory mechanisms that delay the onset of hypotension. Careful history taking and physical examination are essential to the care of LGB patients.

Modern management of LGB encompasses emergency medicine, intensive care medicine, gastroenterology, interventional radiology, and surgery. The approach to LGB patients is initially aimed at immediate assessment and stabilization of the hemodynamic status, followed by identifying the source of bleeding, stopping any active bleeding, treating any underlying lesions, monitoring and managing comorbid illness, and preventing recurrent bleeding.

The first phase of management adheres to the ABCs of resuscitation, governed by the same priorities that apply to all acutely ill patients. Of course, resuscitation and diagnosis must proceed simultaneously. To identify the source of bleeding, endoscopy, mesenteric arteriography, and radionuclide scintigraphy are the major tools, in addition to history taking and physical examination. Endoscopy, the gold-standard procedure for LGB, includes:
- Stigmnsoscopy
- Colonoscopy
- Capsule endoscopy
- Push enteroscopy
- Double-balloon enteroscopy

Endoscopic local hemostasis can be performed at the same time the diagnosis is made. Mesenteric arteriography and radionuclide scintigraphy may be used in difficult LGB cases, with the former providing the ability for embolization or delivering pharmacotherapy. Advances in endoscopic and radiologic hemostasis techniques appear to decrease the rates of rebleeding and surgical intervention (24,25). Transabdominal ultrasound (US) has become popular as a first-line diagnostic tool in a GI emergency. Details of these procedures are noted below. The limited pharmacologic treatments available for LGB—estrogen and octreotide for angiodysplasia, and topical formalin for intractable bleeding from radiation colitis—are strongly supported (26,27). Even with the advances in localization
ANATOMIC CONSIDERATIONS OF LOWER GASTROINTESTINAL BLEEDING

LGB is anatomically defined as bleeding located from the ligation of Treitz to the anus, and may include the jejunum, ileum, ileocecal valve, colon (ascending, transverse, descending, sigmoid, rectum), and anus. Given the length of lower GI tract (the small bowel averages 6.7 meters), as well as the special anatomic problem for the small bowel, such as its free intraperitoneal location, multiple overlying loops, and active contractility, there are several management issues for LGB; these include choice of tests for fecal occult blood, the necessity of nasogastric (NG) tube placement, and the application of endoscopy, radiologic procedures, and US.

Fecal Occult Blood

This is the most common form of GI bleeding. In the ICU, patients suspected of having GI blood loss, secondary to findings of persistent anemia, will have a fecal occult blood study as the first investigative step. While there are numerous types of fecal occult blood tests, the test used determines the location in the GI tract where blood is likely to be detected (29).

Guaiac-based Tests

Guaiac-based tests, the classic fecal occult blood study, utilizes hemoglobin's pseudoperoxidase activity. Guaiac turns blue after oxidation by oxidants or peroxidases in the presence of an oxygen donor such as hydrogen peroxide. Because hemoglobin is degraded in the GI tract, guaiac-based tests are more sensitive for detecting bleeding in the lower than upper GI tract (21). However, the characteristics of specific guaiac-based tests from different companies vary. Whether a guaiac-based test will be positive or not is related to the quantity of blood present in the stool, which is related to the size and location of the bleeding lesion (30,31). Bleeding colonic lesions are more likely to lead to undegraded blood and heme in the stool, guaiac-based tests are best at detecting these distal lesions (29). Various factors influence guaiac test results; for example, fecal rehydration affects the reactivity of guaiac-based tests, and may raise sensitivity but reduce specificity (32). Additionally, foods that contain peroxidases or animal hemoglobins can cause false-positive guaiac test results. False-negative guaiac-based tests may be seen with hemoglobin degradation, sample storage, and vitamin C ingestion (21). Orally administered iron, even in large amounts, does not cause a positive guaiac reaction. Guaiac-based tests are rapid bedside studies.

Immunochromatographic Fecal Occult Blood Tests

Immunochromatographic fecal occult blood tests detect human globin epitopes, and are highly sensitive for the detection of stool blood (34). These tests do not detect UGBL because globin molecules are degraded by UGI tract enzymes. Theoretically, these tests have a higher specificity for the detection of colonic lesions than guaiac-based tests. However, they are limited by technical problems and the need for more intensive laboratory processing. A false negative may occur from hemoglobin degradation and sample storage.

Heme-porphyrin Test

The heme-porphyrin test provides a highly accurate determination of total stool hemoglobin based on a spectrofluorometric method that measures porphyrin derived from heme. The heme-porphyrin test is the most sensitive method of detecting occult blood loss of either the upper or lower GI tract. The results of the heme-porphyrin test are neither affected by intraluminal degradation of hemoglobin nor by the interference of peroxide-producing substances. False positives result from animal hemoglobin and red meats, which contain myoglobin, a heme-containing protein; false negatives are a consequence of sample storage. In summary, guaiac-based tests and immunochromatographic fecal occult blood tests focus on LGB, especially from the colon. The heme-porphyrin tests cannot discriminate between bleeding from the upper and lower GI tract.

In the cardiac ICU, warfarin or low-dose aspirin is used frequently. Neither of these alone appears to cause positive guaiac-based, fecal occult blood tests (35). A positive fecal occult blood test in this setting should raise the possibility of a GI tract abnormality, and requires appropriate evaluation. Jaffin et al., prospectively evaluating the GI tract in antiocoagulated patients with positive guaiac-based fecal occult blood tests, showed that 20% of these results were associated with malignancy (36).

Nasogastric Tube Placement

The necessity of NG tube placement and gastric lavage for acute LGB to exclude a UGI source has not been studied prospectively. Theoretically, blood originating from the LGB tract will not retropulse across the ligament of Treitz to the duodenum or stomach, and there should be no blood content in the nasogastric aspirate. Nonetheless, approximately 2% to 15% of patients presenting with acute severe hematochezia have an upper GI source of bleeding identified on upper GI endoscopy (20). Therefore, some favor the routine use of the NG tube to exclude the possibility of a UGI bleed, although this is controversial and, overall, nasogastric aspiration localizes bleeding accurately only in 66% of attempts (37). In the American Society for Gastrointestinal Endoscopy (ASGE) guideline, NG tube placement to rule out a UGI source of bleeding may be considered if the source is not identified on colonoscopy, particularly if there is a history of UGI symptoms or anemia (38). Patients with hemodynamic compromise and hematochezia should have an NG tube placed (39). The absence of blood in NG aspirate is not sufficient to exclude UGI bleeding—16% of patients with bleeding secondary to a duodenal ulcer have a negative NG tube lavage. The presence of bile without blood is considered as the most reassuring result of a negative NG aspirate, and indicates the absence of active UGB (40).

In spite of the length of the LGB tract, there are anatomic fixed portions, such as the ascending and descending colon and rectum, which makes a US survey of part of the LGB tract possible. The proximal jejunum and terminal ileum can be traced from the fixed duodenum and cecum, respectively (41). US may
Chapter 153: Approach to Lower Gastrointestinal Bleeding

Resuscitation

Approximately 85% of GI bleeding episodes stop spontaneously, whereas the remainder require aggressive resuscitation, diagnostic modalities, and often intense medical and/or surgical management. The first management step for a patient presenting with overt LGIB is the ABCs of resuscitation. Resuscitation is imperative to restore euvolemia and prevent complications of blood loss in the cardiac, pulmonary, renal, or neurologic systems. This takes place in parallel with the initial evaluation of the patient; resuscitation must not be withheld or delayed for diagnostic procedures. The patient’s respiratory and heart rates, and blood pressure, including orthostatic measurements, should be assessed. Attention to the airway is important when the LGIB is caused by an obstructive lesion, which may lead to vomiting with the consequent high risk of aspiration. Postural hemodynamic changes, chest pain, palpitations, syncope, pallor, dyspnea, and tachycardia suggest hemodynamic compromise (43); the severity of bleeding is easy to underestimate due to compensatory mechanisms. An orthostatic decrease in systolic blood pressure greater than 10 mm Hg or an increase in heart rate greater than 10 beats/minute indicates an acute loss of at least 15% of blood volume (44). With hemodynamic compromise, two 16-gauge or larger intravenous (IV) catheters should be secured immediately; central venous access can be established in unstable patients. Packed red blood cells should be utilized in hemodynamically unstable patients, with the goal of maintaining a hematocrit of approximately 30% in the elderly and in those with heart disease or who are otherwise compromised physiologically; and 20% to 25% in younger patients (45). The initial hematocrit may not be the true value, requiring up to 72 hours for equilibration with the intravascular space (46). The presence of coagulopathy, international normalized ratio [INR] greater than 1.5 or thrombocytopenia (less than 50,000 cells/μL) should be corrected with fresh frozen plasma or platelet transfusions, respectively. Oxygen should be administered to keep the SpO₂ between 91% and 95% at a minimum, and vital signs and urine output should be closely monitored. In the elderly or those with a history of cardiac disease, an ECG and cardiac enzyme analysis should be considered. Approaches for LGIB with hemodynamic instability and hemodynamically stable LGIB are noted, respectively, in Figures 153.2 and 153.3.

Determination of Bleeding Site: Noninstrumental

History

Careful history taking is helpful for determining the level of bleeding from the GI tract and to work out a differential diagnosis of the LGIB. Included should be the duration, frequency, and color of the stool; related symptoms of the GI tract such as constipation, fever, or location of pain; medical or surgical history; and history of medications. Except for asymptomatic patients with only anemia due to GI bleeding, the color of the stool is always queried. The patient presenting with acute LGIB may complain of passing bright red blood per rectum, dark blood with clots, or, less commonly, melena. The stool appearance, largely dependent on blood transit time, may be suggestive of the location of bleeding. Blood that has been in the GI tract for >5 hours is usually red, whereas blood present for <24 hours is usually melanin. Upper GI, small bowel bleeding, or slow oozing from the right colon usually produces melena, whereas patients with hematochezia typically have left colonic or rectal lesions (47). If the blood coats the surface of stool, the left colonic—especially the rectum and anus—is more likely to be the bleeding source. Blood dripping into the toilet may occur with hemorrhoidal bleeding. If the stool is mixed with blood, the source may be in the right colon. Of course, massive UGIB can occasionally masquerade as lower GI bleeding in 10% to 15% of patients presenting with severe hematochezia (20). This may happen when massive bleeding originates from esophageal or gastric varices; insertion of an NG tube may be helpful in this situation if the patient does not have hematemesis.

The duration and frequency of bleeding may help in determining the severity of the problem. Related GI tract symptoms may lead to the diagnosis of LGB. Severe constipation should prompt an investigation for colon cancer or a stercoral ulcer; diarrhea may indicate enterocolitis; and fever or local pain may indicate an inflammation-related bleeding source, such as infectious colitis or inflammatory bowel disease. The medical or surgical history may provide clues to the causes of LGB. A previous history of colonic polyps, diverticulosis, or colonic tumor should be considered as a possible source of LGB during the initial evaluation. Renal failure is a well-known risk factor for angiodysplasia or arteriovenous malformation (AVM) (48), as is aortic stenosis (15). Note should be made of patients with renal impairment or who are being dialyzed, as these patients may have platelet abnormalities, resulting in a tendency to bleed if a lesion is present. Ischemic bowel may be present
when severe abdominal pain and bloody stool occur in patients with severe atherosclerotic vascular disease, atrial fibrillation, or hypotension. Radiation therapy for prostate or pelvic cancer induces inflammatory changes of the rectum, and can produce radiographic perforation, presenting months or even years after the radiation exposure. A history of recent colonoscopy with polypectomy indicates post-polypectomy bleeding as the likely source. In patients who have undergone aortic reconstructive surgery, the frequency of significant postoperative colonic ischemia ranges between 1% and 7% (49–51).

Medication history is also important. Medications that can damage the GI mucosa or exacerbate bleeding include non-steroidal anti-inflammatory drugs (NSAIDs), alendronate, potassium chloride, and anticoagulants. Patients admitted with GI bleeding were more likely to be taking selective serotonin reuptake inhibitors (SSRIs) than controls; this association exists for LGIB as well as UGIB (52). Concurrent anticoagulation or use of NSAIDs may be important cofactors in potentiating bleeding (53). The use of aspirin or NSAIDs is strongly associated with both LGB—chiefly from diverticula—and UGIB (54,55). NSAID enteropathy has been increasingly reported, and can be a potential cause of LGB (56,57). A family history of colon cancer increases the likelihood of a colorectal neoplasm, and generally calls for a complete colonic examination in patients with hematochezia (58,59).

**Physical Examination**

A thorough physical examination is essential to assess loss of blood volume, a possible bleeding source, and comorbid conditions (especially for ICU patients). The comorbid conditions may affect the suitability for interventions, such as urgent colonoscopy. The physical examination should also include complete vital signs and heart, lung, and abdominal assessment, as well as an examination of the conjunctiva and...
Overt/acute hematochezia

Hemodynamically stable after resuscitation*

Stable

Bleeding stops

Colonoscopy

Unstable

Still bleeding

Colonoscopy

(-)

Capsule endoscopy

PE/DBE

(+)

Tx of individual lesions

Bladder above ileocecal valve or source unidentified due to much blood in colon

Colonoscopy

(+)

Capsule endoscopy

PE/DBE

(-)

Tx of individual lesions

Helical CT

SBFT/enteroclysis

RBC scan/capsule endoscopy/angiography*

(+) (+)

Repeated endoscopy

and Tx of individual lesions

Observation

and conservative Tx

PE for proximal jejunal lesions

(-)

Repeat colonoscopy if lesions are suggested at colon

DBE for inconclusive studies or deep small bowel lesions

Figure 153.3. Approach for hemodynamically stable lower gastrointestinal bleeding. PE, push-type enteroscopy; DBE, double-balloon enteroscopy; Tx, treatment; SBFT, small bowel follow-through.

Determination of the Bleeding Site: Diagnostic Approach

Endoscopic Approach: General

Safety of Endoscopic Procedures for Critically Ill Patients.

Colonoscopy remains the procedure of choice for evaluating patients with acute LGIB, and enteroscopy is considered for proximal small bowel bleeding. However, for ICU patients with acquired bleeding and hemodynamic instability, questions remain due to the possibility of severe comorbid illness, including skin. Pale conjunctiva indicate anemia. Cutaneous manifestations may suggest disorders causing GI bleeding; those caused by celiac sprue may indicate dermatitis herpetiformis. The polyposis syndromes (Peutz-Jeghers syndrome, Gardner syndrome, and Cronkhite-Canada syndrome) often have cutaneous abnormalities, such as lip pigmentation in Peutz-Jeghers syndrome. Orthostatic vital signs are an important complement to standard monitoring in a patient with severe bleeding but without overt hemodynamic instability. Abdominal tenderness on examination may indicate an inflammatory process, such as ischemic colitis or inflammatory bowel disease. The rectal examination serves to identify anorectal lesions and confirm the stool color described by the patient. In addition, approximately 40% of rectal carcinomas are palpable during a digital rectal examination. Regardless of the presenting features and findings on physical examination, most patients with LGIB still warrant a full examination of the colon.

Laboratory Examination

Initial laboratory studies should include a complete blood count, serum urea nitrogen, creatinine, coagulation profile (prothrombin time, INR, and partial thromboplastin time), liver tests, blood type and cross-match, and electrolytes. As discussed above, positive fecal occult tests (guaiac-based tests and immunochemical fecal occult blood tests) may favor LGIB. The blood urea nitrogen-to-creatinine ratio has been used as a noninvasive test to help distinguish UGIB from colonic sources of bleeding. In the study of Chalasani et al., a ratio of 33 or higher had a sensitivity of 96% for UGIB, although overlap was observed with LGIB, especially in patients with UGIB without hematemesis.

* intervention with IA vasopressin or embolization

Repeat colonoscopy if lesions are suggested at colon

DBE for inconclusive studies or deep small bowel lesions

Repeat colonoscopy

Colonoscopy

Capsule endoscopy

PE/DBE

Hillicial CT

SBFT/enteroclysis

RBC scan/capsule endoscopy/angiography*

PE for proximal jejunal lesions

Colonoscopy

(+) (+)

Repeated endoscopy

and Tx of individual lesions

Observation

and conservative Tx

PE for proximal jejunal lesions

Colonoscopy

Stable

Bleeding stops

Colonoscopy

(-)

Capsule endoscopy

PE/DBE

(+)

Tx of individual lesions

Bladder above ileocecal valve or source unidentified due to much blood in colon

Colonoscopy

(+)

Capsule endoscopy

PE/DBE

(-)

Tx of individual lesions

Helical CT

SBFT/enteroclysis

RBC scan/capsule endoscopy/angiography*

(+) (+)

Repeated endoscopy

and Tx of individual lesions

Observation

and conservative Tx

PE for proximal jejunal lesions

(-)

Repeat colonoscopy if lesions are suggested at colon

DBE for inconclusive studies or deep small bowel lesions

Figure 153.3. Approach for hemodynamically stable lower gastrointestinal bleeding. PE, push-type enteroscopy; DBE, double-balloon enteroscopy; Tx, treatment; SBFT, small bowel follow-through.
respiratory distress, aortic aneurysm, life-threatening dysrhythmias, AML, history of GI tract perforation or operation, severe lower GI obstruction, and bleeding tendency, to list only a few conditions. Colonoscopy complication rates in LGIB of the general population or patients admitted to the ICU primarily for hemorrhage are low, and the bowel preparation itself appears to be safe (65–67). Zuckermand and Prakash reviewed 13 studies and found an overall complication rate of 1.3% (68). A study of 55 ICU patients with acquired LGIB demonstrated an acceptable diagnostic rate of 67% (37 of 55 cases) with out procedure-related complications, suggesting that bedside colonoscopy after preparation is safe for the critically ill (5).

Preparation before Endoscopic Procedures. In patients with massive active bleeding, colonoscopy is often frustrating and nonproductive, and sometimes dangerous. However, some clinicians still consider it the first diagnostic maneuver, even in the patient with severe ongoing bleeding (20,69). Traditionally, colonoscopy for LGIB was delayed because of the need for bowel preparation and the fear of increased procedural complications. Indeed, urgent colonoscopy in an unprepared colon can be challenging, or even dangerous. Good bowel preparation is important for an adequate and sensitive colonoscopy. Studies of urgent colonoscopies performed in an unprepared colon to evaluate for LGIB revealed completion rates as low as 35% (70,71). Jensen et al. studied patients with severe diverticular hemorrhage, noting that completion rates may reach up to 100% if aggressive bowel preparation is performed before urgent colonoscopy (24). Because of variable comborbid conditions, such as decreased bowel motility, obstruction, and elevated risk of diverticular bleeding, colon preparation in ICU patients may not be as thorough as in the general population. There are limited studies of the effectiveness of colon preparation for LGIB in ICU patients. Lin et al. (5) noted that for patients with LGIB occurring after admission to ICU, the reach rate of the cecum with a colonoscope was 58% after enemas or an oral polyethylene glycol solution were administered. In spite of providing a relatively feces-free colon, old methods for colon preparation with clear liquids, laxatives, and enemas or peroral gut lavage 48 to 72 hours before colonoscopy are time consuming, uncomfortable, and inconvenient for patients. For those in the ICU or with active LGIB, these methods are clearly not useful. Peroral gut lavage with saline or balanced electrolyte solutions have been proposed and were found to provide rapid, effective cleansing of the colon; however, the method is not tolerated in 11% of patients due to the high fluid volume—7 to 12 L—and might cause fluid and electrolyte disturbances (72). Recently, development of osmotically balanced solutions may provide minimal water absorption or secretion into the bowel lumen. Generally speaking, there are isoosmotic solutions may provide minimal water absorption or secretion (73). Two liters of PEG-ELS plus bisacodyl or magnesium citrate have also been suggested for colon preparation (74). The studies comparing a standard 4-L PEG-ELS with 2 PEG-ELS with either a magnesium citrate or bisacodyl preparation have shown equal efficacy for colon cleansing (75–77). With PEG-ELS, 5% to 15% of patients have difficulty drinking the large amounts of fluid, or they develop symptoms such as nausea, vomiting, abdominal fullness, and cramps, leading to incomplete colon preparation (78,79). Hyperosmotic preparations, including monobasic and dibasic sodium phosphate (NaP) and magnesium citrate, draw plasma water into the bowel lumen to promote evacuation. In addition to hyperosmotic action, magnesium citrate also stimulates fluid secretion and intestinal motility through the action of cholera toxin. Fleet Phospho-Soda (C. B. Fleet Co., Lynchburg, VA) is a poorly absorbed salt that produces intestinal fluid retention due to the osmotic load, and causes fluid evacuation from the bowel. Patients often prefer NaP to PEG-ELS because there is much less fluid to drink. Liquid and tablet forms of NaP are currently available. Stimulant laxatives include bisacodyl and senna extract. The former is a poorly absorbed difluoromethane that stimulates colonic peristalsis, and the latter contains anthracene derivatives that may be metabolized by colonic bacteria into substances that enhance colonic motility. The timing of colon preparation still varies among institutions. Early methods required preparation 48 to 72 hours before the procedure; of course, this is not ideal for ICU patients who need urgent interventions. Some institutions administer the solution on the day of the examination, even though the manufacturer recommends taking the medication the day before the procedure (74). For the critically ill patient with LGIB, there are no data on the “proper timing” of colon preparation. Further, preparation may be incomplete due to poor motility or ileus. Chiu et al. have suggested that PEG-ELS on the day of procedure may be adequate (74).

The safety of colon preparation is important for critically ill patients with comorbid illness; of particular concern are fluid overload and electrolyte disturbances. PEG-ELS causes no significant change in weight, vital signs, serum electrolytes, or complete blood count (80–82). It is a relatively safe colon preparation solution for patients with electrolyte imbalance, advanced liver disease, poorly compensated congestive heart failure, or renal failure, although in the study of Cranberry et al., exacerbation of congestive heart failure after PEG-ELS administration was noted (83). NaP, a hyperosmotic preparation, may cause alterations in serum electrolytes and extracellular fluid status (84,85). Asymptomatic hyperphosphatemia is seen in up to 40% of patients, but clinically significant hyperphosphatemia is rare and usually limited to patients with renal failure (86–88). Twenty percent of patients had abnormally low serum potassium levels after bowel preparation with NaP (84). It is suggested that NaP is contraindicated in patients with renal failure, acute myocardial infarction or unstable angina, congestive heart failure, ileus, intestinal malabsorption, and significant ascites. Gremse et al. reported that the degree of asymptomatic hyperphosphatemia in children was greater than in adults, and recommended avoiding NaP in children with renal failure, congestive heart failure, ileus, and ascites (88).

Colonoscopy for Critically Ill Patients

Three primary diagnostic tools for LGIB are colonoscopy, radionuclide scintigraphy, and mesenteric arteriography.
Advances in endoscopic technology have brought colonoscopy to the forefront of the management of LGIB. Colonoscopy as the first choice for occult or stable LGIB is not in dispute. However, for brisk LGIB or LGB with hemodynamic compromise, whether the attending physicians consider colonoscopy as the first diagnostic maneuver for brisk LGIB or LGB with hemodynamic compromise is controversial. Colonoscopy in patients with severe hematochezia is impractical because of inadequate visualization caused by brisk blood loss (2,68). Some are reluctant to perform colonoscopy in hemodynamically unstable patients with ongoing bleeding, suggesting that these patients are best served by urgent angiography, perhaps in conjunction with surgical consultation (73,89). However, there is a reason to favor colonoscopy for acute LGIB. In addition to affording a rapid diagnosis, colonoscopy may indicate specific therapy when (and if) the bleeders are found [24,90–92], although the rate for intervention in an ICU study was low [3]. Rapid endoscopic identification of a bleeding source, regardless of whether therapy is administered, may contribute to the clinical management of recurrent bleeding, if it occurs. Finally, compared with angiography, urgent colonoscopy has a higher diagnostic yield and a lower complication rate. In a retrospective study of 107 patients with severe LGIB, colonoscopy was diagnostic in 90% of patients and angiography in 48%; the former was therapeutic in 12% versus 22% for the latter (19). In another study, the diagnostic yield of colonoscopy was 82% versus 12% for angiography (20). Interestingly, most patients undergoing radiographic evaluation for LGIB—regardless of findings and interventions—will subsequently require a colonoscopy to establish the cause of bleeding. In addition to diagnosis, occasional therapy, and management planning, earlier colonoscopy does contribute to a shorter length of hospital stay (93). Hemodynamic instability, higher comorbidity, performance of a tagged red blood cell nuclear scan, and surgery for hemostasis were significantly associated with a decreased likelihood of discharge (93).

While most episodes of LGIB will stop spontaneously, 10% to 15% of patients undergoing urgent colonoscopy received endoscopic therapy. The lesions most amenable to colonoscopic treatment of LGIB, in most studies, are usually angiodysplasia or diverticulosis. Once it is identified as the source of bleeding, angiodysplasia is usually coagulated by methods including the following (5,24,94,95): injection therapy (epinephrine, saline, or ethanol); heater probe; monopolar and bipolar electrocautery; argon plasma coagulation; hemoclips; band ligation.

In a study of ICU patients with nonprimary LGIB, spontaneous cessation occurred in 53% of patients; 29% achieved hemostasis with angiography, but had a higher rate (19%) of recurrent bleeding (5). Other studies, not limited to ICU patients, have reported rebleeding rates of 13% to 33%, and many patients may require more than one treatment (84,95).

Timing of Colonoscopy

The use of colonoscopy is controversial for critically ill patients with LGIB. Some believe that colonoscopy is best utilized in patients whose bleeding has stopped or slowed down (73,89), and others agree with early colonoscopy for LGIB. Evidence suggests that earlier intervention leads to more diagnostic and therapeutic opportunities (24,96). It has further been noted that early colonoscopy reduces the length of hospital stay, and therefore should decrease treatment costs (97,98). Urgent colonoscopy after bowel preparation with endoscopic treatment of patients with active diverticular bleeding or stigmata of bleeding has been shown to be highly effective in decreasing the need for surgical intervention (24,73).

In conclusion, prompt intervention may decrease the need for surgical exploration, as well as the rate of recurrent bleeding and length of hospital stay. Who needs urgent colonoscopy? Early identification of high-risk patients would allow the more selective delivery of urgent therapeutic interventions to those who will benefit. Clinical high-risk predictors in the first hour of evaluation in patients with severe LGIB have been proposed, and included an initial hematocrit of no more than 35%, the presence of abnormal vital signs 1 hour after initial medical evaluation, and gross blood on initial rectal examination (99). How early should the urgent colonoscopy be performed? The definition of urgent colonoscopy varies widely in the literature—from within 8 hours to 24 hours of presentation (5,20,24,100–104). Most definitions consider the procedure within 12 to 24 hours; more recently, the literature defines urgent colonoscopy as within 12 hours (20,24).

Sigmoidoscopy or Colonoscopy?

In studies of the general population with LGIB, diverticular and angiodysplastic bleeding are the most frequent events. Although anatomically prone to be located in the left colon, bleeding most often occurs in the right colon; thus, most endoscopists prefer a total colonoscopy. Flexible sigmoidoscopy can be performed in the initial evaluation of patients with LGIB, but the diagnostic yield of flexible sigmoidoscopy in LGIB is low, ranging from 9% to 38% (10). Regardless of presentation, flexible sigmoidoscopy may miss serious proximal pathology (103). However, in the study of ICU patients with acquired bleeding, 78% of responsible lesions were in the left colon (5). If the critical situation precludes use of total colonoscopy, sigmoidoscopy as the first maneuver may be acceptable for this patient group. Nonetheless, unless a definite and compatible bleeding source is identified with flexible sigmoidoscopy, the study of LGIB should proceed to a full colonoscopy in most patients.

In 2005, the ASGE offered the following guidelines regarding LGIB (38): 1. Colonoscopy is effective in the diagnosis and treatment of LGIB (prospective controls). 2. Colonoscopy is recommended in the early evaluation of severe acute LGIB (prospective controls). 3. Thermal contact modalities, including heat probe, and bipolar/multipolar coagulation and/or epinephrine injection can be used in the treatment of bleeding diverticula, vascular ectasia, or postpolypectomy bleeding (prospective controls).

Enteroscopy and Capsule Endoscopy for Critically Ill Patients

The small bowel has traditionally been a problematic area to evaluate because of the long length, looping, free intraperitoneal location, active contractility, and limits of standard endoscopy. It is estimated that 10% to 25% of LGIB originates...
in the small bowel and can pose a diagnostic dilemma for clini-
cians (106–108). Small bowel sources account for 0.9% to 7% of
cases presenting with blood per rectum (19,20,109), and
comprises approximately 5% of obscure GI bleeding (110). Up-
per endoscopy and colonoscopy appear to have limited roles in
the investigation of small bowel bleeding, and are only useful
when the bleeding source is the duodenum or the most dis-
tal segment of the small intestine (terminal ileum), respectively
(53). Some clinicians have utilized peroral intubation with a
standard colonoscope, reaching a point 20 to 60 cm distal to the
ligament of Treitz; this may increase the diagnostic yield
of GI bleeding of obscure origin by 17% to 46%—not an in-
significant proportion (13,111–113). Small bowel enteroscopy
is currently the best endoscopic investigative modality. Indeed,
it has become the cornerstone of management in patients with
obscure GI bleeding. Current tools for ruling out small bowel
diseases include push-type enteroscopy (PE), double-balloon
enteroscopy (DBE), intraoperative enteroscopy, and capsule en-
teroscope.

Small bowel evaluation may be performed with PE, which
allows endoscopic evaluation of the proximal 60 cm of the
jejunum or 150 cm distal to the pylorus. PE is probably the
most commonly performed small bowel procedure today, and
is often pursued when upper GI endoscopy and colonoscopy
have failed to find the source for blood loss. PE may be per-
formed using an enteroscope or pediatric colonoscope. The
obvious limitation of push enteroscopy is the inability to reach
lesions distal to the middle jejunum. PE can only examine a
relatively short portion of the small bowel, even under fluo-
rosCOPY, and the true depth of insertion is unreliable. Some
experts consider it insufficient to capsule endoscopy, and
believe that it should be performed only if capsule endoscopy or
other modalities are positive for a proximal small bowel lesion.
The use of an overtube to prevent looping of the instrument
in the stomach increases the insertion depth by 10 to 25 cm
(114). Prototype, variable stiffness enteroscopes are emerging in
an attempt to achieve maximal insertion depth without the use of an overtube (115,116). Although PE cannot investigate
the entire small bowel, an important benefit of PE is the abil-
ity to provide diagnostic and therapeutic capabilities with one
procedure if the bleeder can be found.

The diagnostic yield of PE is between 38% and 65% of pa-
tients in whom upper and lower endoscopy are negative (117).
Multivariate analysis in a retrospective, two-center study by
Lepère et al. showed that melena and chronic renal failure in-
crease the diagnostic yield of PE in patients with unexplained
GI bleeding (123). The positive findings noted with PE for pa-
tients with renal failure were most often in the distal duodenum
or jejunum, including ulcers (17%) and AVMs (41%). Others
have shown similar results in that most small bowel lesions
diagnosed by PE are vascular in nature (angiodysplasia/AVM
(119–121), followed by ulcerations and malignancies (122). A
delay between the bleeding and PE (less than or more than 4
days) and a history of recurrent intestinal bleedings before
PE were not associated with positive findings; thus, rapid per-
formance of PE may not be necessary, except in patients with
continuous active bleeding (123). Another benefit of PE is that
it provides a “second look” for lesions that may have been
missed on original endoscopy. Interestingly, 25% to 40% of
lesions found on PE are within reach of a standard upper en-
teroscopy (124,125). Complications of PE are infrequent, occur-
ing in less than 1% of cases. Most complications, including
bleeding and perforation, are related to the use of an overtube
(126,127). The data of effectiveness and safety of PE for ICU
patients are limited.

Even when PE reaches a maximal depth of insertion of 160
cm below the ligament of Treitz, there is still over 250 cm of
small intestine remaining unexamined. It is possible to close
this gap with a newly developed DBE system (128). The newest
modality for imaging the small bowel was introduced in 2001,
yet Yamamoto et al. reported their results using a double-
balloon method in four patients (129). The DBE represents
the first successful provision of both diagnostic and therapeu-
tic intervention to the entire small bowel. The goal is to reach
the ileocecal valve, but this often is not possible. The total
inspection of the small bowel is usually attainable with the
peroral approach and retrograde approach per rectum. On av-
erage, approximately 250 cm is achieved via the oral route and
130 cm via the anal route, with a mean examination time of
75 min (130). If total enteroscopy is necessary, it can be
achieved in 60% to 86% of cases, depending on the experi-
ence of the endoscopist (131,132). Yamamoto et al. performed
50% of the studies in an antegrade fashion, with the remain-
der being retrograde. The bleeding source was found in 76%,
and hemostasis using electrocautery was performed success-
fully in 18% (131). Multicenter experience with DBE in the
United States showed that the mean procedure time was 115
minutes, and the yield of DBE for a GI bleed ranged between
32% and 75% (130,133). Oral DBE requires no specific prepa-
ration other than a 6- to 8-hour fast before the procedure. If
a retrograde (anal) approach is undertaken, standard colonic
preparation is necessary. Conscious sedation or general anes-
thesia may be utilized. Endoscopic hemostasis using injection
therapy, argon plasma coagulation, electrocautery, and hemo-
clipping may be used as is done in routine upper GI endoscopy
and colonoscopy. Complications of DBE are noted in 1.1% to
8.5% (131,133), and include aspiration pneumonia, abdom-
inal pain, perforation, and acute pancreatitis (130,131,133–
135). The effectiveness and safety of DBE for critically ill pa-
tients are unknown.

Before the advent of capsule endoscopy and DBE, intraop-
erative endoscopy was the only way to detect and treat lesions
beyond the reach of push enteroscopy. Now the role of di-
agnostic intraoperative enteroscopy is likely to decrease with
introduction of the less invasive capsule endoscopy and DBE;
however, it still plays a role in specific clinical situations. In-
teroperative endoscopy is performed in conjunction with a sur-
geon in the operating room, and with the patient under general
anesthesia. Intubation may be achieved transorally, transanally,
or through an operative enterotomy, depending on the clinical
circumstance and physicians’ preference.

During laparoscopy combined with intraoperative en-
teroscopy, the endoscopist carefully inspects the intestinal lu-
mens with a push enteroscope, while the surgeon slowly guides
the bowel over the endoscope using the air-trapping technique
and examines the external wall with palpation and transul-
mination (136). Lesions can be treated endoscopically or
marked with a tattoo for surgical resection. The terminal ileum
is reached in more than 90% of patients (137). The yield in de-
tecting bleeding lesions reaches 70% to 100% (133,138), mak-
ing intraoperative endoscopy the most sensitive method of di-
agnosing small bowel disorders. However, the high sensitivity
comes at the cost of extreme invasiveness, making it a proce-
dure of last resort. The complication rate is estimated at about
Capsule endoscopy is a safe and promising diagnostic tool for GI bleeding of unknown origin, focusing especially on the small bowel; it may obviate the need for angiography in some difficult patients. The idea of wireless imaging of the small intestine was conceived simultaneously by Paul Swain, a British gastroenterologist, and Gavriel Iddan, an Israeli scientist. They merged research efforts in 1998 and soon developed a pill-sized camera with sufficient battery life to image the entire small intestine (139). Capsule endoscopy was introduced into clinical practice in 2001 and made it possible for the first time to visualize intraluminal conditions throughout the entire small bowel. The first commercially available video capsule (Given) is composed of three main subsystems: an ingestible capsule endoscope, a data recorder, and a workstation. The PillCam SB capsule measures 11 × 26 mm in size, and weighs less than 4 g, with a miniaturized image-capturing system, battery, light source, and transmitter. After an overnight fast, the patient swallows the capsule, which travels through the GI tract by means of the actions of normal peristalsis. The capsule device captures two images per second and has a battery life of approximately 6 to 8 hours. Captured images are transmitted by a digital radio-frequency communication channel to an external data recorder unit.

Studies comparing capsule endoscopy with other diagnostic procedures, including enteroclysis, PE, computed tomography (CT) scan, and intraoperative enteroscopy, showed that capsule endoscopy was clearly superior in the diagnosis of occult/obscure overt small bowel bleeding (127,140–149). Capsule endoscopy has proven superior to enteroclysis (144–147) and PE (140,142,143). In a series of 60 patients comparing the wireless capsule to PE, Saurin et al. showed that the use of the capsule raised the diagnostic yield from 38% to 69% (143). However, one study did not show the superiority of capsule endoscopy over PE. Van Gossuin et al. noted that, with obscure GI bleeding, no significant difference in diagnostic yield was found between push and wireless-capsule endoscopy (150). Capsule endoscopy, compared to CT scanning, is reported to be superior in detecting small bowel lesions (144,146,147). In a study of 42 patients with obscure GI bleeding, capsule endoscopy sensitivity and intraoperative enteroscopy was not significantly different. Furthermore, no additional diagnoses were made with intraoperative enteroscopy (149).

The overall diagnostic yield rate of capsule endoscopy in patients with GI bleeding ranges from 45% to 66% (141,144,151,152,153). The timing of capsule endoscopy has been addressed in two studies and appears related to diagnostic yield and intraoperative enteroscopy was not significantly different. When administered to patients with ongoing overt bleeding, the diagnostic yield is higher—87% to 92%—than in those with previous overt bleeding or iron deficiency anemia—46% to 36% (151,154). Hartmann et al. noted that capsule endoscopy identified lesions in 100% of patients with ongoing overt bleeding, 67% of patients with previous overt bleeding, and 67% of patients with obscure/occult bleeding (148). To increase the diagnostic yield of capsule endoscopy, prokinetic agents prompt the passage of the capsule and prevent the exhaustion of batteries before study completion. Bowel preparation with oral sodium phosphate has been suggested to offer better visualization than overnight fasting alone, and is associated with fewer disturbances by intraluminal turbid fluid (156). However, there are concerns that increasing bowel motility may result in missing a lesion. Freeman et al. showed that erythromycin markedly reduced gastric emptying time and had a negative effect on the small bowel images. Preparation of elderly subjects with PEG or sodium phosphate also had a negative effect on small bowel transit time (157). PEG increased the visibility of the proximal small bowel in one study, but had no effect in a second investigation (158,159). Presently, bowel preparation is preferred by most practitioners. Erythromycin leads to faster gastric emptying at the expense of small bowel transit time and poorer visualization (160). Bowel preparation with simethicone, which can decrease intraluminal gas bubbles, resulted in significantly better visibility (161). Simethicone may be added to the routine preparation for capsule endoscopy to improve visualization of the small bowel mucosa. A delay in bowel transit time may result in an incomplete study due to capsule battery drainage.

Despite the higher diagnostic yield, capsule endoscopy limitations are evident: biopsy specimens cannot be obtained, therapeutic intervention cannot be performed, and localization of some lesions is imprecise (162). Capsule endoscopy, however, is regarded as a low-risk procedure that is well tolerated (163). The primary risk with capsule endoscopy is capsule entrapment within the GI tract; this occurs in 0.75% to 3% of cases. Most entrapment occurs in the small intestine, although case studies report impaction at the cricopharyngeus, tracheal aspiration, and retention in diverticula (10). Risk factors for entrapment include NSAID-induced strictures, prior abdominal radiation, Crohn enteritis, prior major abdominal surgery, and known diverticula. A trapped capsule may be retrieved endoscopically or surgically. In cases where the colon is not visualized on capsule endoscopy and the patient does not see the capsule pass, an abdominal radiograph should be obtained to document passage. Absolute contraindications to its use include GI obstruction and pseudo-obstruction—meaning ileus. Relative contraindications include lesions that limit visualization, such as gastroparesis; history of intestinal strictures or fistulae; pregnancy; history of multiple small bowel diverticula; history of Zenker diverticulum; history of abdominal surgeries or radiation; and an active swallowing disorder or dysphagia. Although there is concern about the use of capsule endoscopy in patients with pacemakers, new evidence suggests that capsule endoscopy may be safely utilized in these patients (164).

Capsule endoscopy has been reported to change patient management in up to 75% of cases (142), although the studies focused solely on small bowel lesions. Colonic bleeding is difficult to evaluate via capsule endoscopy because of retained stool, limited battery life, and poor visual field due to the colon’s large diameter. A recently developed PillCam Colon capsule endoscopy appears promising for colonic evaluation (165), although in comparison to conventional colonoscopy, false-positive findings were recorded in 33% cases (166). The use of capsule endoscopy in critically ill patients has been limited. These patients often cannot ingest the capsule by themselves, especially if endotracheally intubated; an endoscopic technique of capsule placement has been described for such patients (167). In addition to swallowing problems, bowel transit time may be delayed due to sepsis, electrolyte imbalance, medication use, and anatomic changes due to surgery. Abnormal bowel transit time affects the diagnostic yield.
of capsule endoscopy. The utilization of capsule endoscopy for small bowel or obscure bleeding must be made on a case-by-case basis.

Nonendoscopic Approach

Nuclear Medicine

As mentioned previously, for LGIB, endoscopy, nuclear medicine, and mesenteric angiography are three main diagnostic modalities. The two techniques of radionuclide scanning commonly use either $^{99m}$Tc-labeled red blood cells or $^{99m}$technetium sulfur colloid. A $^{99m}$Tc-labeled red blood cell scan is the preferred technique, with images that can be detected for up to 12 to 24 hours after injection. If the rate of bleeding is insufficient to give an immediate positive test, or if the bleeding is intermittent, the labeled red blood cells can sometimes accumulate to detect the site of bleeding—when rescanned—up to 24 hours after injection. This technique can detect bleeding at a rate as low as 0.1 to 0.5 mL/minute, and is thought to be a sensitive diagnostic tool for LGIB (168). $^{99m}$Technetium sulfur colloid is rapidly cleared by the reticuloendothelial system after injection, with a half-life of only 2 to 3 minutes. Therefore, if there is no active bleeding when administered, the $^{99m}$technetium sulfur colloid is quickly cleared, with a resultant nondiagnostic test.

Timing the Use of Radionuclide Scanning. Radionuclide scanning, often performed repeatedly during a hospital course, may be used as the screening test, followed by angiography, small bowel enteroscopy, or surgery to definitively localize and treat the bleeding lesion. Because of its high sensitivity—it has the ability to detect bleeding as low as 0.1 to 0.5 mL/minute—radionuclide scanning has been utilized as a guide for surgical resection, and as a screening test prior to angiography when colonoscopy fails to find the LGIB bleeder. As a guide for surgical resection, localization of the bleeding site is essential. The literature suggests that the localization accuracy of radionuclide scanning is quite variable, ranging from 24% to 94% (169-171). Contrarily, a review by Hunter and Pezim suggested that a localization rate with the red blood cell scan was estimated to be 23% to 71% (172). In the same study, nearly half of patients studied (42%) underwent an incorrect surgical procedure based on red blood cell scan results (172); other studies have noted that radionuclide scanning did not alter surgical management in any manner (173-175). Thus, most clinicians use radionuclide scanning as a guide for further diagnostic studies, such as enteroscopy/colonoscopy, rather than for surgical intervention (176).

Reportedly requiring 10-fold less hemorrhage to achieve a positive study than angiography, the sensitivity of a radionuclide scan for active bleeding has been noted to be greater than 90%, and is superior to that of angiography (177-179). Penney et al. showed that radionuclide scans increased the yield of angiography from 22% to 53% (180). Other studies have had contrary results (180-182), and radionuclide scanning may potentially delay therapeutic interventions (183). Therefore, although there is no strong evidence supporting radionuclide scanning prior to mesenteric angiography, it may demonstrate low-flow bleeders, leading to better management.

Another role for radionuclide scanning is in the evaluation for Meckel diverticulum, especially in young patients presenting with LGIB (184). The Meckel scan uses a technetium pertechnetate tracer, which has affinity to accumulate in the gastric mucosa. It is quite useful in the pediatric population, with sensitivity as high as 81% to 90% (185,186). Due to insufficient gastric mucosa in the diverticulum, the sensitivity of the Meckel scan is much lower in the adult population, estimated to be approximately 62% (187,188). Several techniques that were reported to increase the diagnostic yield of the Meckel scan, administered before the study, include pentagastrin, histamine blockers, and saline lavage of the stomach and bladder (189-192).

Angiography

Angiography, first employed in the diagnosis of GI bleeding more than 40 years ago, provides imaging of the entire mesenteric system, localizes the sites of hemorrhage, and affords the opportunity for transcatheter interventions. It now holds an established place in dealing with difficult GI bleeding, both for diagnosis and treatment. Mesenteric angiography is more invasive than technetium-labeled red blood cell scanning, and requires a bleeding rate of at least 0.5 to 1.0 mL/minute to detect bleeding (193). Unfortunately, bleeding is frequently intermittent and may occur at a much lower rate, resulting in the inability to detect the causative lesion (194). Angiography is usually undertaken when patients have clinical indicators of severe bleeding (e.g., tachycardia and/or syncope). Although colonoscopy is the diagnostic modality of first choice for LGIB, many endoscopists are reluctant to perform colonoscopy in hemodynamically unstable patients with ongoing bleeding; these patients usually undergo radiographic studies. In addition, the bleeding of colonic lesions, such as vascular abnormalities, can be too massive for colonoscopic visualization, thus precluding the procedure. In some centers, a radionuclide scan is requested before mesenteric angiography, because a negative radionuclide scan is unlikely to have a positive angiogram. Angiography has been reported to be especially useful in patients presenting with postoperative GI hemorrhage (195).

Angiography will localize the site of bleeding in 40% to 86% of patients with LGIB (118,183,196,197). Even if a bleeding site is identified on angiography, localizing the site intraoperatively can be difficult; angiography has a specificity of 100% but a sensitivity of only 30% to 47% (198). Diverticula and angiodysplasia are the most common findings when angiography is positive, with 50% to 80% of the bowel bleeding sites being supplied by the superior mesenteric artery (199). Diverticular hemorrhage is most likely to produce extravasation on angiography (200). Following the injection of contrast media, bleeding and nonbleeding angiodysplastic lesions are characteristically seen as ectatic slowly emptying veins, vascular tufts, or small veins, with early filling in the arterial phase (201). Angiography is more sensitive than colonoscopy for detecting angiodysplasia (202), and when angiography identifies a bleeding site, treatment with embolization therapy or directed infusion of vasopressin may be performed. The overall rate of complication for mesenteric angiography is similar to most selective angiography, and is acceptable at less than 5% (203). Complications include hematoma or bleeding at the catheter site; access site thrombosis; contrast reactions; injury to the target vessels, including dissection and distal embolization; and...
Pharmacologic vasoconstriction for LGIB involves an intraarterial infusion of vasopressin, started at a rate of 0.2 units/minute. If the bleeding continues, the rate of infusion can be increased up to maximal dose of 0.4 units/minute. A repeat angiogram should be performed after 20 to 30 minutes to assess whether the bleeding is continuing or slowing down. If the bleeding seems to stop, infusion continues at the same rate for 12 hours, and subsequently, the dose of vasopressin is decreased by 50% provided that no bleeding recurs. After 12 hours of only saline infusion, the catheters are removed (169). Success rates for hemostasis are variable, with some reports as low as 36% and others as high as 100% (183,196,215,216).

Other Modalities

Small bowel follow-through (SBFT) and enteroclysis are used to detect small lesions, while barium enema is the image study for the colon. All of these are less sensitive for superficial lesions such as angiodysplasia, a common bleeder of LGIB. SBFT is of little use in evaluating obscure GI bleeding, with a diagnostic yield that may be as low as 0% (226). For patients with a high

renal failure (204). The injured vessels usually involve the SMA, IMA, and celiac artery.

In addition to its diagnostic role, angiography offers therapeutic possibilities via pharmacologic vasoconstriction or selective embolization (transcatheter arterial embolization [TAE]), and therefore may reduce the need for surgical resection. Once the bleeder is confirmed with contrast injection, embolization of the vessel is performed, usually with one of three embolic agents: microcoils, polyvinyl alcohol sponge particles, or gelatin sponge particles, alone or in combination (205). Pharmacologic vasoconstriction is achieved with intra-arterial vasopressin infusion. TAE may be a more definitive means of controlling bleeding, but is associated with a risk of intestinal infarction. Selective embolization initially controls bleeding in up to 100% of patients, but rebleeding rates have been reported to be 15% to 40% (206,207). The major complication rate was 10% to 20% and included dysrhythmias, pulmonary edema, hypertension, and ischemia (206,207). The bowel infarction or colonic necrosis rate from embolization ranged from 10% to 20% (208–210). Superselective TAE may decrease the incidence of ischemia and rebleeding (209,211,212). A literature review of 144 cases by Kuo et al. showed a minor complication rate of 9% and 0% for major complications (205).

In the past, embolization has been reserved for treatment of UGIB, whereas LGB has been controlled with vasopressin infusion. The reason is based on reports in the older literature in which infarction frequently occurred after LGI embolization. With advances in superselective embolization techniques, clinically significant bowel ischemia has become an uncommon complication (213,214). Although the efficacies of vasopressin and embolization are reasonably comparable, embolization allows more rapid completion of therapy and a decreased likelihood of systemic complications. Embolization should be considered a primary option for LGIB, although vasopressin is still preferable for diffuse lesions and cases in which superselective catheterization is not technologically possible.

Pharmacologic vasoconstriction for LGIB involves an intraarterial infusion of vasopressin, started at a rate of 0.2 units/minute. If the bleeding continues, the rate of infusion can be increased up to maximal dose of 0.4 units/minute. A repeat angiogram can be performed after 20 to 30 minutes to assess whether the bleeding is continuing or slowing down. If the bleeding seems to stop, infusion continues at the same rate for 12 hours, and subsequently, the dose of vasopressin is decreased by 50% provided that no bleeding recurs. After 12 hours of only saline infusion, the catheters are removed (169). Success rates for hemostasis are variable, with some reports as low as 36% and others as high as 100% (183,196,215,216). Bleeding recurrence is high, and may occur in up to 50% of patients after cessation of the infusion (183). Vasopressin should not be used in patients with significant coronary artery disease or peripheral vascular disease; mesenteric thrombosis, intestinal infarction, and death have been reported with its use (196). During the vasopressin infusion, patients need to be in an ICU setting where they can be monitored for myocardial, bowel, and peripheral ischemia; hypertension; dysrhythmias; and hypotenstion. Nitroglycerin reverses the vasopressin-induced coronary vasoconstriction without affecting the therapeutic vasoconstriction of the mesenteric artery (217). If standard angiography is negative, provocative angiography has been suggested with anticoagulants, vasodilators, and thrombolytics; of course, their use may cause bleeding (122) and is not routine. The use of provocative angiography should be reserved for selected patients at competent centers with well-trained radiologists.

For critically ill patients, ensuring the adequacy of intravenous fluid volume is very important before mesenteric angiography. Dehydration may exacerbate the nephrotoxicity of the contrast medium.

Computed Tomography Scan and Magnetic Resonance Imaging

CT scans are not usually considered diagnostic tools for LGB, except in the context of bleeding bowel tumors. Several recent reports suggest that helical CT scans may be useful (218–220) in that this mode of scanning has the potential to detect hemorrhage rates of 0.5 mL/minute or less (221,222)—between 72% and 79% (218,219). In the evaluation of colonic vascular lesions, helical CT has a sensitivity of 70% and specificity of 100% compared to colonoscopy and conventional angiography (223).

Helical CT angiogram is a modified form of angiography. A rapid-acquisition CT scan is performed 30 seconds after contrast is injected into the abdominal aorta. The images are taken in 10-mm slices, 3 mm apart. A positive result is seen as extravasation of contrast medium into the intestinal lumen. Helical CT angiography reports a 70% sensitivity in the diagnosis of colonic angiodysplasia through the demonstration of vessel accumulation in the colon wall, early filling vein, and enlarged supplying artery (223). Currently, there is no role for the use of magnetic resonance imaging (MRI) in the evaluation of LGB.

Ultrasound

Ultrasonography is a convenient, noninvasive, nonradiation-emitting, and easily available diagnostic tool in the emergency department and ICU. Data regarding the use of US for LGB have been limited. Yamaguchi et al. noted that the colonic bleeding site was localized by US in 59 of the 90 (66%) patients compared with 81% by colonoscopy. When the bleeding site was in the rectum, the US detection rate was only 30% (10 of 33 patients), but the US detection rate was 82% to 100% when the bleeding site was elsewhere. These clinicians concluded that rectal and diverticular bleeding were difficult to diagnose by US, but for the other diseases, diagnosis by US was possible in 91% to 100% of cases (224). In our experience, angiodysplasia cannot be detected by US, while diverticulitis—but not diverticular bleeding—can. Other causes of bleeding, especially due to tumors, enteropathy, and colitis, can also be detected by US. In contrast to Yamaguchi’s study (224), we think rectal lesions may be visualized via US through a urinary bladder window. We have proposed the “ultrasonographic bisection approximation method” to localize and detect GI obstructive lesions. The accuracy of US in predicting obstructive levels in the gastric outlet and duodenum, the jejunum and ileum, and the colon were 100%, 74%, and 98%, respectively (225).

Other Modalities

Small bowel follow-through (SBFT) and enteroclysis are used to detect small lesions, while barium enema is the image study for the colon. All of these are less sensitive for superficial lesions such as angiodysplasia, a common bleeder of LGB. SBFT is of little use in evaluating obscure GI bleeding, with a diagnostic yield that may be as low as 0% (226). For patients with a high
index of suspicion for the presence of small bowel diseases, such as small bowel tumor or Crohn disease, the diagnostic yield will be higher; SBFT detected 83% of small bowel tumors. In patients with suspected Crohn disease, SBFT may have a sensitivity of over 90% (227, 228).

Endoscopy is a modified form of SBFT in which a 10 French catheter is inserted into the distal duodenum or proximal jejunum under fluoroscopy, followed by the infusion under high pressure of a double-contrast solution with barium and air, water, or methylcellulose. This rapid rate of infusion allows better distention and visualization of the small bowel. Studies revealed that endoscopy seems superior to SBFT for evaluation of the small bowel (229–233). Small bowel tumors seen to be the most common diagnosis made by endoscopy, followed by Meckel diverticulum and Crohn disease of the terminal ileum (232, 233). Angiodysplasia is not detected by endoscopy. With the advent of capsule endoscopy, the use of SBFT or endoscopy for GI bleeding has declined.

Barium enema cannot detect superficial lesions or confirm a definitive bleeding source of the colon. Furthermore, it may complicate subsequent colonoscopy or angiography, and is less useful for critically ill patients with LGB.

**TREATMENT OF LOWER GASTROINTESTINAL BLEEDING**

**Pharmacologic Therapy**

Unlike pharmacologic therapies for UGB, there are no medications with a strong evidence base for LGB. The medications for the different causes of LGB include estrogen/progesterone compounds, octreotide, aminocaproic acid (an antifibrinolytic), and tranexamic acid (an antifibrinolytic, marketed as Cyklokapron in the United States and as Transamin in Asia). Hormonal therapy with estrogen/progesterone compounds, previously used to treat bleeding associated with hereditary hemorrhagic telangiectasia, has been tried in patients with GI bleeding from angiodysplasia. For diffuse ectasias or angiodysplasia refractory to conservative and endoscopic therapy, estrogen/progesterone compound use is controversial, and has been noted to be ineffective in recent studies (234–237). Although the true mechanism is unknown, estrogen/progesterone compounds are thought to improve coagulation, alter microvascular circulation, and improve endothelial integrity. Adverse effects include breast tenderness and vaginal bleeding in women, gynecomastia and loss of libido in men, fluid retention, and stroke (238).

Octreotide has been used in patients with bleeding from diffuse vascular ectasia (238–240). At a dose of 0.05 to 1 mg/day subcutaneously, it was reported to be effective and without adverse effects (238, 239). Nairdone et al. noted that octreotide may lead to decreased transfusion requirements (239), but unfortunately, carefully controlled trials are not available. Other agents, including aminocaproic acid and tranexamic acid, may be helpful, but studies with controlled data are not forthcoming.

Steroids, 5-aminosalicylic acid compounds, and sucralfate (per mouth or per rectum) have been used to treat radiation proctitis, but there are little data supporting their effectiveness (241–244).

Ulcereative colitis (UC) and Crohn disease can cause severe LGIB (245). A recent review of acute major GI hemorrhage in inflammatory bowel disease suggests that bleeding is much more common in Crohn disease than UC (245). Bleeding from inflammatory diseases is usually self-limited and responds to medical therapy. An endoscopically treatable lesion is uncommon. Steroid and 5-aminosalicylic acid compounds are frequently used for active lesions. Infliximab, known as a “chimeric monoclonal antibody,” reduces the amount of active tumor necrosis factor-alpha (TNF-α) in the body. It has been used successfully to avoid emergency surgery in Crohn patients with severe bleeding (246, 247).

**Endoscopic Procedures**

Endoscopic therapy has been the major modality for LGB, including the small bowel and colon, as long as the endoscopes can reach the lesions. Endoscopic therapy for LGB includes injection therapy (epinephrine, saline, or ethanol), heater probe, monopolar and multipolar electrocoagulation, argon plasma coagulation (APC), hemoclips, and band ligation (Table 153.4). An alternative treatment for hemorrhagic radiation-induced proctitis, by topical application of formalin, was first described by Rubinstein et al. in 1986 (248). Topical application of formalin for hemorrhagic radiation-induced proctitis can be performed with or without endoscopy in the operating room. It is simple, effective, inexpensive, and without major systemic

**TABLE 153.4**

<table>
<thead>
<tr>
<th>LGB lesions</th>
<th>Endoscopic procedures</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ulcer</td>
<td>Injection therapy, Heaoter probe, Electrocoagulation</td>
</tr>
<tr>
<td>Angiodysplasia</td>
<td>APC, Heaoter probe, Electrocoagulation</td>
</tr>
<tr>
<td>Diverticulum</td>
<td>Hemoclips, APC, Injection therapy, Heaoter probe, Electrocoagulation</td>
</tr>
<tr>
<td>Radiation proctitis</td>
<td>APC, Topical formalin</td>
</tr>
<tr>
<td>Postpolypectomy bleeding</td>
<td>Hemoclips, Band ligation, APC, Injection therapy, Heaoter probe</td>
</tr>
</tbody>
</table>

LGB, lower gastrointestinal bleeding.

*APC, argon plasma coagulation. Not recommended for ulcers with big exposed vessels.
Most cases of colonic ischemia resolve with conservative treatment. However, 15% to 20% of patients who develop infarction will require surgical intervention, with a substantial risk of death (253). Until recently, surgery was the only effective management for Dushalay lesions in up to 5% of patients (254). Surgery is usually not recommended on the basis of nuclear red blood cell scans alone because of variable accuracy of nuclear red blood cell scans.

### ASSESSMENT OF SEVERITY IN LOWER GASTROINTESTINAL BLEEDING

Acute LGIB ceases spontaneously in 80% to 85% of patients (68), but the overall mortality rate may be as high as 12% (255). A reliable predictive or scoring system can accurately and quickly forecast the severity of an episode of acute LGIB, risk of recurrent bleeding, need for therapeutic intervention, and related mortality. Moreover, a scoring system is potentially of great benefit to the clinician at the point that initial triage is performed to ensure appropriate levels of care. Unfortunately, in contrast to acute UGIB, there are few scoring systems developed and validated to predict the outcome of patients with acute LGIB.

The BLEED classification system was proposed for evaluation of acute UGIB and LGIB by Kollef et al. (256) utilizing five items, including ongoing bleeding, systolic blood pressure <100 mm Hg, prothrombin time greater than 1.2 times control, altered mental status, and unstable comorbid conditions. The BLEED classification system triages patients with acute LGIB into those at high and low risk of adverse in-hospital outcomes—defined as recurrent hemorrhage, need for surgery for control of hemorrhage, and death. A second study by the same group found that it could predict outcome in patients hospitalized with acute LGIB when the BLEED classification system was applied at the point of initial evaluation in the emergency department (257). In a retrospective chart review of 252 consecutive, hospitalized patients, Strate et al. analyzed 34 clinical, nonendoscopic variables that were available within 4 hours of medical evaluation (60). They identified seven independent clinical risk factors for severe, acute LGIB: tachycardia, low systolic blood pressure, syncope, nontender abdominal examination, bleeding per rectum within the first 4 hours of medical assessment, use of aspirin, and more than two active coexisting conditions. The investigators speculated that such clinical data may be used to risk-stratify patients with acute LGIB who may benefit from urgent intervention. Based on these factors, patients were stratified into three risk groups: (a) those with more than three risk factors—an 84% risk of severe bleeding; (b) those with one to three risk factors—a 43% risk; and (c) those with no risk factors—a 9% risk. Das et al. developed and validated artificial neural networks (ANNs), computer-based decision support systems, for the prediction of recurrent bleeding, need for intervention, and death with LGIB (258). ANNs performed well in predicting death (97%), recurrent bleeding (93%), and the need for intervention (94%). Velayos et al. prospectively studied patients admitted with LGIB, and identified three predictors of severity and adverse outcome in the first hour of evaluation: initial hematocrit less than 35%, presence of abnormal vital signs 1 hour after initial medical evaluation,
and gross blood on initial rectal examination. These predictive tools may help guide the initial triage and approach to the patient with LGIB (259). The scoring system elements are summarized in Table 153.6; however, an ideal risk scoring system does not yet exist.

Which factors are most predictive of adverse patient outcome? The presence of comorbidity, evidence of acute hemodynamic instability, and presence of high-risk endoscopic stigmata of recent hemorrhage are considered most predictive of an adverse outcome. The initial hemoglobin level may be an inaccurate marker of the severity of GI bleeding. Which risk stratification system is better—endoscopic or nonendoscopic? It has been suggested that endoscopic triage in acute LGIB may not be as efficacious as with acute UGIB (255).

**TABLE 153.6**

<table>
<thead>
<tr>
<th>Scoring system</th>
<th>Risk factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>The BLEED classification system (performed at point of initial evaluation in the emergency department)</td>
<td>■ Ongoing bleeding &lt;br&gt; ■ Systolic blood pressure less than 100 mm Hg &lt;br&gt; ■ Prothrombin time greater than 1.2 times control &lt;br&gt; ■ Altered mental status &lt;br&gt; ■ Unstable comorbid disease</td>
</tr>
<tr>
<td>Scoring system of Strate et al. (performed within 4 h of initial evaluation)</td>
<td>■ Tachycardia &lt;br&gt; ■ Low systolic blood pressure &lt;br&gt; ■ Syncope &lt;br&gt; ■ Nontender abdominal examination &lt;br&gt; ■ Bleeding per rectum within the first 4 h of medical assessment &lt;br&gt; ■ Use of aspirin &lt;br&gt; ■ More than two active comorbid conditions</td>
</tr>
<tr>
<td>Scoring system of Velayos et al. (performed within the first hour of initial evaluation)</td>
<td>■ Initial hematocrit less than 35% &lt;br&gt; ■ Presence of abnormal vital signs 1 h after initial medical evaluation &lt;br&gt; ■ Gross blood on initial rectal examination</td>
</tr>
</tbody>
</table>

LGIB, lower gastrointestinal bleeding.

**OBSCURE LOWER GASTROINTESTINAL BLEEDING**

Upper GI endoscopy and colonoscopy are the usual initial evaluation tools for GI bleeding; they will be negative in the patient with LGIB. The scoring system elements are summarized in Table 153.6; however, an ideal risk scoring system does not yet exist. Which factors are most predictive of adverse patient outcome? The presence of comorbidity, evidence of acute hemodynamic instability, and presence of high-risk endoscopic stigmata of recent hemorrhage are considered most predictive of an adverse outcome. The initial hemoglobin level may be an inaccurate marker of the severity of GI bleeding. Which risk stratification system is better—endoscopic or nonendoscopic? It has been suggested that endoscopic triage in acute LGIB may not be as efficacious as with acute UGIB (255).

When repeated endoscopy of the upper or lower GI tract is negative, investigation should rapidly focus on the small intestine. However, before surveying the small bowel, one needs to ensure that a repeat upper GI endoscopy has been performed, as 25% to 64% of patients with a negative upper GI endoscopy and colonoscopy are found to have UGI tract lesions at the time of repeat UGI endoscopy (264–267). Interestingly, the source of obscure GI bleeding may be identified in up to 58% of cases within a month from the last bleeding episode, even if previous investigations did not allow identification (268).

Clinically, the age of the patient is very important in the differential diagnosis of GI bleeding. Patients younger than 40 are more likely to suffer from small bowel tumors, anatomic anomalies, genetic problems, or Crohn disease/ulcerative colitis. Patients older than 40 are more prone to bleeding from vascular lesions and neoplasm (12). A special group of patients may have the specific causes of LGIB. In populations with immunosuppression, such as patients with HIV infection, renal transplant, or pancreatic transplant, LGIB often is caused by CMV ulcers. Renal failure and aortic valvular stenosis are well-known risk factors for angiodysplasia (14,15). Radiation colitis should be considered in patients with a history of radiation therapy for cervical or prostate cancer. Aortoenteric fistulae may be considered in patients with obscure GI bleeding and prior aortic aneurysm repair. The approach to the evaluation of the obscure bleeder is listed in Table 153.7 and Figure 153.4.

Bleeding from the small intestine that occurs between the ligament of Treitz and the ileocecal valve represents a challenging problem because of the relative inaccessibility of traditional endoscopy to the long, looping small intestine. Small
TABLE 153.7
APPROACH TO THE EVALUATION OF OBSCURE BLEEDING

<table>
<thead>
<tr>
<th>Elderly patients</th>
<th>Higher risk for vascular lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Renal disease</td>
<td></td>
</tr>
<tr>
<td>Aortic valvular stenosis</td>
<td></td>
</tr>
<tr>
<td>Connective tissue disease</td>
<td></td>
</tr>
<tr>
<td>von Willebrand disease</td>
<td></td>
</tr>
<tr>
<td>Surgical patients</td>
<td>Higher risk for anastomotic bleeding or aortoenteric fistulae</td>
</tr>
<tr>
<td>NSAID drug use</td>
<td>Increased risk of small bowel ulcerations</td>
</tr>
<tr>
<td>Immune-compromised patients</td>
<td>Cytomegalo virous ulcers</td>
</tr>
<tr>
<td>History of radiation to pelvis</td>
<td>Radiation colitis</td>
</tr>
<tr>
<td>NSAID, nonsteroidal anti-inflammatory drug.</td>
<td></td>
</tr>
</tbody>
</table>

GI bleeding may occur after myocardial infarction (MI) due to both medications and interventions, or it can induce an MI secondary to hemodynamic instability and anemia. For the critical care practitioner, management of coexisting GI bleeding and coronary arterial events includes how to predict and detect an MI that occurs after GI bleeding, how to prevent and manage GI bleeding associated with interventions related to the acute MI, and the necessity/safety/timing of endoscopic procedures.

The prevalence of acute MI in patients with GI bleeding ranges from 1% to 14% (271–274). Conversely, acute MI seen with significant upper or lower GI bleeding occurs in 30% to 49% of patients admitted to the ICU, with an overall mortality rate of 3% to 10% (271,272). Significant GI bleeding deleteriously affects myocardial function, as massive blood loss may cause hypovolemia, hypoperfusion, and decreased oxygenation delivery to the myocardium, eventually leading to an acute MI. Elderly and patients with a history of coronary artery disease (CAD) are candidates for acute MI after a significant GI bleed. On occasion, the overt symptoms of GI bleeding may mask the typical symptoms of an evolving MI. Therefore, cardiac enzymes, including troponin-I, and an electrocardiogram are routinely suggested in high-risk patients with GI bleeding, even when the patient has no chest pain, to avoid a delay in diagnosis.

![Suspected occult lower GI bleeding with stable hemodynamics](image1)

- Correct anemia
- Capsule endoscopy/RBC scan
- Tx of individual lesions
  - PE for proximal jejunal lesions
  - DBE for inconclusive studies or deep small bowel lesions
- US Helical CT SBFT/enteroclysis
- Observation and conservative Tx
- Repeat studies according to the findings
- Intervention with IA vasoressin or embolization

FIGURE 153.4. Approach for occult lower gastrointestinal bleeding. GI, gastrointestinal; RBC, red blood cell; Tx, treatment; PE, push-type enteroscopy; DBE, double-balloon enteroscopy; CT, computed tomography; SBFT, small bowel follow-through; US, ultrasound.
TABLE 153.8

<table>
<thead>
<tr>
<th>Studies</th>
<th>N</th>
<th>Y</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Barium studies</td>
<td>Small bowel follow-through</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>Nuclear studies</td>
<td>Angiography</td>
<td>Y</td>
<td></td>
</tr>
<tr>
<td>Enteroscopy</td>
<td>Double-balloon enteroscopy</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>Capsule endoscopy</td>
<td>Exploratory laparotomy with and without endoscopic assistance</td>
<td>Y</td>
<td></td>
</tr>
</tbody>
</table>

**IMAGING STUDIES FOR SMALL BOWEL BLEEDING**

**Angiodysplasia**

Angiodysplasia, composed of ectatic, dilated submucosal veins, is the most common vascular anomaly of the GI tract. It includes vascular ectasias, AVFs, and angiomata. Angiodysplasia is thought to be due to degeneration of the submucosal venules, and thus is seen predominantly in the elderly (284). It has been reported as a common cause of acute major LGIB and slow

...
intermittent blood loss (285,286). The percentage of acute LGIB that has been attributed to angiodysplasia varies from 3% to 40%, depending on the study (287,288). Angiodysplasia is also the most common cause of small bowel bleeding, accounting for 70% to 80% of episodes (289). Angiodysplasia may be a clinically challenging problem, as it frequently has multiple lesions that may be difficult to identify, and bleeding associated with angiodysplasia is more likely to be intermittent than diverticular bleeding. Furthermore, angiodysplasia is the most common cause of recurrent LGIB of the elderly, with recurrent bleeding rates reported between 10% and 30% (285,286,289). The lesions of angiodysplasia are predominantly located in the right colon (cecum and ascending colon, 54%), followed by the sigmoid colon (18%) and rectum (14%) (291), whereas angiodysplasia can be found throughout the small intestine. Overt bleeding from angiodysplasia is typically brisk, painless, and intermittent. Modalities for diagnosing angiodysplasia as a cause of LGIB include colonoscopy, mesentry angiography, enteroscopy, capsule endoscopy, and, sometimes, helical CT angiography. The sensitivity of colonoscopy for detecting angiodysplasia exceeds 80% (202). At colonoscopy, angiodysplastic lesions have a characteristic appearance: red, flat, ectatic blood vessels radiating from a central feeding vessel. A pale halo may typically be seen around the lesion. Use of narcotic medications for sedation and analgesia have been reported to decrease the sensitivity of colonoscopy for detecting angiodysplasia because of a transient decrease in mucosal blood flow. Additionally, colonoscopy can provide a therapeutic function; this mode of therapy is safe and effective for angiodysplasia. Argon plasma coagulation is increasingly popular for the treatment of bleeding colon angiodysplastic lesions and angiodysplasia located in the small bowel (292,293). Other methods include heater probe, bipolar coagulation, and injection therapy. If no other cause of bleeding is identified in a patient with recurrent or persistent GI bleeding requiring transfusions, the presence of angiodysplasia is an indication for treatment. Angiography is also diagnostic for angiodysplasia. After injection of contrast, angiodysplasia is seen as ectatic slowly emptying veins, vascular tufts, or small veins with early filling. When angiography identifies a bleeding angiodysplasia, treatment with embolization therapy or infusion of vasopressin may be performed. Helical CT angiography has shown that ischemic colitis, not angiodysplasia or diverticulosis, is one of the most frequent causes of LGIB (5). Ischemic colitis is usually caused by “low-flow states” and occlusion of small, rather than large, vessels. Mesenteric hemodynamics may be compromised by changes in the systemic circulation or by anatomic or functional changes in the mesenteric vasculature. In patients who have undergone aortoiliac reconstructive surgery, the frequency of postoperative colonic ischemia is 1% to 7% (304–306). The typical segments affected by nonocclusive colonic ischemia are the “watershed” areas of the colon: the splenic flexure and the rectosigmoid junction. Clinically, ischemic colitis most frequently involves the splenic flexure, the descending colon, and the sigmoid colon. Ischemic colitis with segmental distribution has an abrupt transition between damaged and normal mucosa at colonoscopy. Clinically, ischemic colitis presents with the sudden onset of mild, left lower quadrant, crampy abdominal pain with infrequent hemodynamic alterations. The pain may be accompanied or followed by bright red blood per rectum or bloody diarrhea. Conditions that compromise colonic blood flow can lead to ischemia, and include cardiovascular insults; aortic bypass surgery; aneurysmal rupture; vasculitis; inherited or acquired hypercoagulable states.

### Ischemic Colitis

Ischemic colitis, resulting from a sudden, often temporary, reduction in mesenteric blood flow, is increasingly recognized as a cause of acute LGIB. Ischemic colitis accounts for approximately 1% to 19% of LGIB (303), and may be transient and reversible. Data from a study limited to medical ICU patients have shown that ischemic colitis, not angiodysplasia or diverticulosis, is one of the most frequent causes of LGIB (5). Ischemic colitis is usually caused by “low-flow states” and occlusion of small, rather than large, vessels. Mesenteric hemodynamics may be compromised by changes in the systemic circulation or by anatomic or functional changes in the mesenteric vasculature. In patients who have undergone aortoiliac reconstructive surgery, the frequency of postoperative colonic ischemia is 1% to 7% (304–306). The typical segments affected by nonocclusive colonic ischemia are the “watershed” areas of the colon: the splenic flexure and the rectosigmoid junction. Clinically, ischemic colitis most frequently involves the splenic flexure, the descending colon, and the sigmoid colon. Ischemic colitis with segmental distribution has an abrupt transition between damaged and normal mucosa at colonoscopy. Clinically, ischemic colitis presents with the sudden onset of mild, left lower quadrant, crampy abdominal pain with infrequent hemodynamic alterations. The pain may be accompanied or followed by bright red blood per rectum or bloody diarrhea. Conditions that compromise colonic blood flow can lead to ischemia, and include cardiovascular insults; aortic bypass surgery; aneurysmal rupture; vasculitis; inherited or acquired hypercoagulable states.

### Colonic Diverticular Bleeding

Colonic diverticular bleeding results from rupture of the intramural branches (vasa recta) of the marginal artery at the dome of a diverticulum or at the antimesenteric margin (295,296). Diverticula are the second most common source, if not the most common, of acute LGIB in some studies, and have been reported to comprise 20% to 55% of all cases of LGIB. Although greater than 75% of diverticula are found in the left colon, the right colon is the source of diverticular bleeding in 50% to 90% of patients. Most of the diverticula are not symptomatic, whereas approximately 20% develop diverticulitis and 3% to 5% develop acute severe bloody stool (11). Clinical presentation in LGIB generally is acute, painless hematochezia (295–297). Diverticulosis is rare in patients under 40 years of age. Age and NSAIDs have been shown to be associated with diverticular bleeding (298,299). At least 75% of diverticular bleeding will stop spontaneously, but up to 25% will require emergent intervention (300). Recurrent bleeding from diverticulum occurs in 14% to 38% of patients (4,11,169,237). Colonoscopy and angiography are used to diagnose diverticular bleeding. Endoscopic therapy utilized includes epinephrine injection, bipolar coagulation, band ligation, and placement of hemoclips; the latter has been more popular in recent years. Diverticular hemorrhage is most likely to produce extravasation on angiography (230). Vasopressin infusion and embolization have also been used to stop bleeding. The traditional management of diverticular bleeding has largely been supportive. Nonsurgical therapy may be performed with angiography or colonoscopy. Surgical intervention is required when hemodynamic instability persists despite aggressive resuscitation. Surgical intervention may be necessary in 18% to 25% of cases. In the elderly patient with comorbid conditions, diverticular bleeding results in morbidity and mortality rates of 10% to 20% (301,302).

Chapter 153: Approach to Lower Gastrointestinal Bleeding
such as pregnancy and oral contraceptives; intense exercise (304); and medications or drugs that reduce colonic motility or blood flow, such as catecholamines. Among them, intense exercise results in blood being shunted from the viscera to the working muscles, resulting in decreased splanchnic blood flow by as much as 80% (303). Colonoscopy or flexible sigmoidoscopy have replaced barium enema as the choice for colonic ischemia. Edema, hemorrhage, and ulceration with a sharp demarcation between normal and abnormal mucosa can be shown at endoscopy. Histologically, submucosal hemorrhages, intravascular thrombus, and hyalinization of the lamina propria are seen, in addition to inflammatory cell infiltrates. In contrast to acute mesenteric ischemia, angiography is not necessary for ischemic colitis. Most cases of colonic ischemia resolve with conservative treatment. The 15% to 20% of patients who develop gangrene will require surgical intervention (306). A minority of patients will develop chronic ischemic colitis or stricture. Treatment is supportive with bowel rest, intravenous fluids, optimization of hematocrit status, and correction of the precipitating conditions (307). When surgery is necessary, it is often because of transmural infarction with necrosis rather than bleeding.

Nonsteroidal Anti-inflammatory Drug-induced Enteropathy and Colonopathy

NSAID enteropathy and colonopathy are lesions related to the use of NSAIDs. NSAIDs have been demonstrated to exacerbate inflammatory bowel disease, cause colitis that resembles inflammatory bowel disease, and complicate diverticular diseases by increasing the risk of perforation and severe hematochezia (308,309). The terminal ileum and cecum are particularly susceptible to NSAID-induced injury. This is because the pills may be static for a longer period of time in the terminal ileum and cecum than in other segments of the bowel. History of use of NSAIDs and endoscopy (colonoscopy and enteroscopy) are essential for diagnosing NSAID enteropathy and colonopathy. The diaphragm-like structure is pathognomonic of NSAID injury as a result of a scarring reaction secondary to ulceration. They are most frequently found in the midsection of the small intestine, but have also been reported to occur in the terminal ileum and colon (310–313). Treatment of NSAID-induced mucosal injury is discontinuation of the NSAIDs. Performance of a repeat colonoscopy has been suggested 6 to 8 weeks after cessation of the NSAID in order to check for resolution of the ulcers or colitis. Surgical intervention is rarely required for NSAID-induced bleeding or perforation (310).

Radiation Colitis

Radiation therapy to the colon may induce inflammatory changes and can produce radiation colitis. A history of prior radiation therapy for prostate or pelvic cancer may indicate radiation proctitis, no matter how distant from radiation exposure. Argon plasma coagulation is the most effective treatment (314–316). Complications of argon plasma coagulation were reported, such as severe bleeding, extensive necrosis of the rectum, or perforation, which occurred in 10% of patients. Treatment of hemorrhagic, radiation-induced proctitis by topical application of formalin can be simple, effective, and inexpensive. No major systemic side effects have been described. Other treatments of radiation colitis include steroids, hyperbaric oxygen, 5-aminosalicylic acid compounds, and sucralfate, but little data support their effectiveness.

Dieulafoy Lesions

Dieulafoy lesions are unpredictable and life threatening, because bleeding is often massive and recurrent (317,318). Dieulafoy lesions should always be included in the differential diagnosis of GI bleeding, especially when a definitive source is not found on routine investigation (i.e., in the presence of obscure GI bleeding). In the colon, solid bowel content can contribute to mucosal stercoral ulceration over an abnormally dilated submucosal arteriole and subsequent rupture and bleeding. Endoscopic diagnosis and treatment of enteric Dieulafoy lesions beyond the duodenal bulb are difficult. In colonic Dieulafoy lesions, massive bleeding makes endoscopic diagnosis and treatment more problematic; angiography can be quite useful in localizing the source of bleeding in these situations. Because the Dieulafoy lesions are found by endoscopy, they can be treated with heater probe, electrocoagulation, sclerotherapy, band ligation, or hemoclips; failure of endoscopic treatment is not uncommon. Angiography can also be therapeutic, providing access for such treatments as embolization or tissue glue. Surgery is now reserved for lesions that cannot be controlled by endoscopic or angiographic techniques, estimated at up to 5% of patients.

Postpolypectomy Bleeding

Postpolypectomy bleeding is the cause of 2% to 5% of acute LGIB. A history of recent colonoscopy with polypectomy leads to the diagnosis of postpolypectomy bleeding as the most likely source. Most of this bleeding stops spontaneously. Persistent bleeding can be treated with various endoscopic techniques, including injection of epinephrine followed by thermal therapy, band ligation of the remaining polyp stalk, and hemoclips (39). Endoscopic therapy was successful in treating over 95% of patients in a retrospective case review study (319).

Ulcerative Colitis and Crohn Disease

Bleeding due to ulcerative colitis and Crohn disease is usually self-limited and responds to medical therapy, but can sometimes cause severe LGB (245). An endoscopically treatable lesion is uncommon, and surgical intervention may be necessary, especially in patients with recurrent bleeding. Bleeding is much more common in Crohn disease than in ulcerative colitis (245). Infliximab has been used successfully to avoid emergency surgery in Crohn patients with severe bleeding (246,247).

Less Common Causes of Lower Gastrointestinal Bleeding

There are less common causes of LGIB. Neoplastic lesions are the cause of acute LGB in 2% to 26% of cases (2); if limited
to ICU patients, the incidence may be even lower. Severe con-
sta tion should prompt an investigation for a stercoral ulcer.
Hemorrhoids are common and account for 2% to 9% of cases
of acute severe hematochezia (3,4). Conservative management
with sitz baths, avoidance of straining, and dietary modifica-
tion are usually effective. However, surgical hemorrhoidectomy
and rubber band ligation are options for refractory cases. Coli-
tis, including pseudomembranous colitis, can be caused by dif-
different diseases, each a potential cause of LGIB. Numerous in-
fec tious agents can penetrate and injure the colonic mucosa and
cause acute LGIB. The major role of endoscopy is to visualize
the mucosa and obtain biopsies to guide the use of antimicro-
bial agents. There are no reports of endoscopic therapy for
bleeding due to colitis, although sometimes an actively bleed-
ing ulcer or a visible vessel may warrant an attempt endoscopic
therapy.

Acute Hemorrhagic Rectal Ulcer or Acute
Hemorrhagic Rectal Ulcer Syndrome

Acute hemorrhagic rectal ulcer (AHRU) or acute hemorrhagic
rectal ulcer syndrome (AHRUS) has attracted the attention of
ICU practitioners. AHRUS has been reported as one of the
most frequent causes of LGIB in the ICU (5). AHRUS was first
introduced in 1981, and was recognized as a syndrome later;
so far, there are reports only from Japan and Taiwan (320–
323). AHRUS accounts for 2.8% of the patients with massive
LGIB (321). AHRUS characteristically occurs suddenly, with
painless, massive, fresh rectal bleeding in elderly, bedridden
patients with severe comorbid illness (321,322). It is prone to
occur in patients with diabetes mellitus who are using anticoag-
ulant or antiplatelet agents (323). Lesions of AHRUS locate at
the lower rectum. Endoscopically, they were characteristically
solitary or multiple rectal ulcers with round, circumferential,
geographic, or Dieulafoy-like lesions located within a mean
distance of 4.7 ± 1.5 cm from the dentate line. Histopatho-
logically, the lesions appear as necrosis, with denudation of the
covering epithelium, hemorrhage, and multiple thrombi in the
vessels of the mucosa and underlying stroma (320). Lesions of
AHRUS are considered to be similar to stress-related mucosal
injury. Therapies for AHRUS include injection therapy, heater
probe, hemoclips, and per anal suturing (322,323). As a hemo-
static strategy, hemoclipping alone showed a favorable result,
with a hemostatic success rate as high as 76.9% (323). There is
no established pharmacologic treatment. Risk factors associ-
ated with recurrent bleeding were severity of comorbid disease
and abnormal coagulation status. The prognosis of AHRUS de-
pends on the state of the underlying diseases and achievement of
hemostasis (322).

PEARLS

■ The consequences of LGIB in the ICU, anemia and hypo-
volemia, may prevent weaning and extubation, thus pro-
longing ICU length of stay.
■ Data indicating the true incidence of LGIB during ICU hos-
pitalization are lacking.
■ Patients who develop LGIB while hospitalized for another
disease process have a higher risk of death than those ad-
mitted with LGIB.

■ LGB patients with comorbid illness have higher mortality
than those without.
■ Two to 15% of patients with presumed LGIB will have
UGIB.
■ Studies limited to ICU patients show that ischemic colitis
and acute hemorrhagic rectal ulcers are the most frequent
causes of LGIB, followed by colitis and diverticula.
■ Pallor, fatigue, chest pain, palpitations, dyspnea, tachycardia,
pertussis-related dizziness, and syncope are sug-
gestive of hemodynamic compromise, and demand aggres-
sive care.
■ For detecting occult blood loss from the lower GI tract,
guaiac-based tests and immunochromical fecal occult blood
tests are optimal choices. Heme-porphyrin tests cannot dis-
criminate between UGIB and LGIB.
■ NG tube placement to rule out a UGI source of bleeding
should be considered in patients with hemodynamic com-
promise and hematochezia, or if a source is not identified on
colonoscopy.
■ The absence of blood in NG aspirate is not sufficient to
refute UGIB bleeding, but the presence of bile without blood
indicates the absence of an active UGB.
■ The past medical history may help to elucidate a specific
bleeding source. Key points include antecedent constipation
or diarrhea, the presence of diverticulosis, radiation therapy,
recent polyps, and vascular disease/systemic hypoten-
sion/atrial fibrillation/aortoiliac reconstructive surgery.
■ A rectal exam is essential in LGIB, serving to identify ano-rec-
tal lesions and confirm the stool color described by the pa-
tient.
■ Evaluation of the small bowel is indicated for those patients
in whom UGI endoscopy and colonoscopy are negative.
■ Current endoscopic tools for small bowel diseases in-
clude PE, DBE, intraoperative enteroscopy, and capsule en-
doscopy.
■ The diagnostic yield of PE is between 38% to 65% in pa-
patients with negative upper and lower endoscopy.
■ The diagnostic yield of DBE is between 52% and 76%.
■ Intraoperative endoscopy provides the highest diagnostic
and therapeutic yield (70%–100%) in patients with chronic
or acute recurrent LGIB.
■ The use of capsule endoscopy for small bowel or obscure
bleeding must be made on a case-by-case basis.
■ The timing of capsule endoscopy appears related to the di-
agnostic yield. A high yield may be possible in ongoing overt
bleeding.
■ Evidence suggests that capsule endoscopy may be safely used
in patients with pacemakers.
■ Radionuclide scanning or angiography may be appro-
 priate in patients with massive bleeding that precludes
colonoscopy or in whom a bleeding source is not identified
on colonoscopy.
■ Radionuclide scanning detects active bleeding at rates of 0.1
to 0.5 mL/minute, and is more sensitive than angiography,
but less specific than a positive endoscopic or angiographic
study.
■ Radionuclide scanning is normally not used as a defini-
tive study before surgical therapy, but rather as a tool to
guide further diagnostic studies or therapeutic interven-
tions.
■ Angiography may be a useful diagnostic and therapeutic tool
in patients with active bleeding.
During the vasopressin infusion, patients need to be in an ICU setting.

For critically ill patients, maintenance of adequate intravascular volume is very important to prevent contrast nephrotoxicity.

In general, the presence of comorbidity, acute hemodynamic instability, and high-risk endoscopic stigmata of recent bleeding are considered most predictive of an adverse outcome.

References


2300

Section XV: Gastrointestinal Disease and Dysfunction

115. Harwood GC, Gosling GJ, Farnell MA, et al. Prospective controlled assess-


121. Huyer M, Avox AT, Molinari S. Diagnostic yield and effect on clini-


128. May A, Nachbar L, Wardak A, et al. Double-balloon enteroscopy: pre-


132. May A, Neschke L, Wiedim A, et al. Double-balloon enteroscopy (push-


Section XV: Gastrointestinal Disease and Dysfunction


CHAPTER 154  LIVER FAILURE: ACUTE AND CHRONIC

ACUTE LIVER FAILURE

Definitions and Immediate Concerns

Acute liver failure (ALF) may be defined as the development of hepatic encephalopathy (HE) and coagulopathy in a patient with no history of previous liver disease, with the onset of the disease within 26 weeks of jaundice (1). It should be stressed that ALF is not a disease, but rather a clinical syndrome triggered by numerous etiologic agents; consequently, ALF is extremely heterogeneous, and its management has not been well defined.

Overview

There are three possible outcomes after ALF: spontaneous survival without orthotopic liver transplantation (OLT), OLT, or death. In the U.S. Acute Liver Failure Study cohort consisting of more than 1,000 enrollees with ALF between 1998 and 2006, one third of patients died, one quarter underwent OLT, and the remainder (42%) recovered spontaneously (W. M. Lee, personal communication) (2). The dismal overall survival of patients with ALF: 25% in the 1970s, has improved to approximately 65% with the advent of OLT as a rescue treatment and improvements in intensive care management (3).

Etiology, Prevalence, and Initial Testing

In the U.S. ALF Study Group (SG) Cohort, acetaminophen (APAP) accounts for approximately 45% of cases (3), half of those due to ingestion of a single large dose with suicidal intent, and the other half as “therapeutic misadventures” (4,5). Patients in the latter group frequently ingest large doses of APAP in combination with narcotic preparations, and ingestions tend to be multiple over time (5). The second most common cause of ALF remains indeterminate even after extensive investigation (4,5). Patients in the latter group frequently ingest large doses of APAP in combination with narcotic preparations, and ingestions tend to be multiple over time (5). The second most common cause of ALF remains indeterminate even after extensive investigation (4,5).