A

cute lower gastrointestinal bleeding (LGIB) is distinct clinically from upper gastrointestinal hemorrhage in epidemiology, prognosis, management, and outcome. LGIB encompasses a wide clinical spectrum ranging from trivial hematochezia to massive hemorrhage with shock, requiring emergency hospitalization. The spectrum of LGIB is broad. It may be acute or chronic, obvious or occult. This article focuses on managing patients with acute LGIB. Occult bleeding and obscure bleeding are discussed in articles by Lin and Rockey elsewhere in this issue.

Evaluation of hemodynamic status and resuscitation are the cornerstones in the initial treatment of LGIB. They should take place concomitantly with the history and examination. Postural changes, chest pain, palpitations, syncope, pallor, dyspnea, and tachycardia suggest hemodynamic compromise [1]. An orthostatic decrease in systolic blood pressure of greater than 10 mmHg or an increase in heart rate of more than 10 beats/min indicates an acute loss of at least 15% of blood volume [1,2]. Two large caliber peripheral catheters or a central venous line should be placed immediately in patients with hemodynamic compromise. Initial laboratory studies should include a complete blood count, coagulation profile, type and cross-match, and electrolytes. The use of anticoagulants or nonsteroidal anti-inflammatory drugs (NSAIDs), the presence of liver disease, and serious comorbid medical conditions should be identified rapidly. Coagulopathy or thrombocytopenia should be corrected with fresh frozen plasma or platelet transfusions. In elderly patients or those with a history of cardiac disease, an electrocardiogram or cardiac enzymes should be obtained. The character and frequency of stool output should be noted, as it allows critical assessment of the severity of bleeding. Patients with brown or infrequent stools are unlikely to have brisk bleeding; those with frequent passage of red or maroon stool, however, may have aggressive ongoing bleeding [3].
GENERAL APPROACH
Assessment of Severity in Lower Gastrointestinal Bleeding

In contrast to upper gastrointestinal bleeding, predictors of poor outcome in LGIB are not defined as well [4–7]. Because most episodes of LGIB will stop spontaneously, the early identification of high-risk patients would allow the more selective delivery of urgent therapeutic interventions to the patients most likely to benefit. Two recent studies have examined this. Strate and colleagues [8] retrospectively collected data on 24 clinical variables available in the first 4 hours of evaluation in 252 consecutive patients. They defined severe bleeding as either: continued bleeding within the first 24 hours of hospitalization, or recurrent bleeding after 24 hours of stability. Independent correlates of severe bleeding were: heart rate of at least 100 beats per minute, systolic blood pressure no more than 115 mmHg, syncope, nontender abdominal examination, bleeding per rectum during the first 4 hours of evaluation, aspirin use, and more than two active comorbid medical conditions.

In another study [9] clinical predictors in the first hour of evaluation in patients with severe LGIB included initial hematocrit of no more than 35%, presence of abnormal vital signs 1 hour after initial medical evaluation, and gross blood on initial rectal examination. In this study of 448 prospective patients, severe LGIB was defined as gross red blood per rectum after leaving the emergency department associated with either abnormal vital signs—defined as systolic blood pressure less than 100 mmHg or heart rate above 100 beats per minute—or more than a two-unit blood transfusion during hospitalization. Thus, patients with unstable vital signs, particularly when they persist after initial medical evaluation, are at risk for severe bleeding. Additional studies are needed to refine other predictors.

Evaluation of the Upper Gastrointestinal Tract

An upper gastrointestinal source of bleeding is detected in 10% to 15% of patients presenting with severe hematochezia [10]. Patients with hemodynamic compromise and hematochezia should have a nasogastric tube placed. If bile is present, an upper source is unlikely [11,12]. If the aspirate is nondiagnostic (no blood or bile), or if there is a strong suspicion of an upper bleeding source (i.e., history of previous peptic ulcer disease or frequent NSAID use), then an upper endoscopy should be performed before examining the colon [13–15]. An upper endoscopy should be performed if no source of bleeding is identified during colonoscopy.

SPECIFIC INTERVENTIONS/TREATMENTS

Once the patient has been resuscitated, the severity and acuity of bleeding assessed, and an upper gastrointestinal source of bleeding excluded, management rapidly shifts to localization and therapy. The three major modalities available for the treatment of LGIB include: surgery, radiology techniques, and colonoscopy. Pharmacologic measures also are becoming available for a limited number of very specific bleeding sources. The merits of each modality vary by the source of bleeding and the clinical situation.
Colonoscopy

In an effort to improve outcomes by identifying more lesions amendable to endoscopic therapy (either actively bleeding or with stigmata of recent bleeding), interest has increased in performing colonoscopy early in the course of diverticular bleeding. The definition of urgent has varied widely in the literature, from within 8 hours to within 24 hours of presentation [16–21]. Numerous authors have reported urgent colonoscopy to be safe and have a high diagnostic yield in patients who have acute lower gastrointestinal bleeding (Table 1) [17–21]. Bowel preparation generally is recommended before urgent colonoscopy, as complications such as perforation are more common in the uncleansed colon because of decreased visibility. This is usually a polyethylene glycol lavage solution by mouth or nasogastric tube.

In published series, 10% to 15% of patients undergoing urgent colonoscopy received endoscopic therapy (see Table 1). Endoscopic hemostasis methods include injection therapy (epinephrine or saline), heater probe therapy, monopolar and multi-polar electrocoagulation, argon plasma coagulation, hemoclips, and band ligation. In contrast to upper gastrointestinal bleeding, there are no data to compare the effectiveness of each modality in the treatment of diverticular hemorrhage. As a corollary from peptic ulcer bleeding, many experts favor combining epinephrine injection and multi-polar electrocoagulation. Diverticula with active bleeding are injected with 1 mL of epinephrine (dilution, 1:20,000 or 1:10,000) in three or four quadrants around the mouth of the diverticula to stop bleeding. Visible vessels then are treated with multi-polar electrocoagulation with 10 to 15 watts of power with moderate pressure on the vessel [22,23]. Management of adherent clots is more controversial. Some experts recommend cold guillotining of the clot [24]. This involves injecting epinephrine in four quadrants around the pedicle of the clot and shaving it down to 3 to 4 mm above the attachment with a cold polypectomy snare. The underlying stigmata (often a nonbleeding visible vessel) then are treated with multi-polar electrocoagulation. Other experts treat adherent clots by four-quadrant epinephrine injection and then multi-polar electrocoagulation to the base of the clot without intentionally removing it.

Table 1

<table>
<thead>
<tr>
<th>Study (year)</th>
<th>N</th>
<th>Bowel preparation %</th>
<th>Specific diagnosis N (%)</th>
<th>Endoscopic therapy N (%)</th>
</tr>
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<tr>
<td>Chaudhry (1998) [17]</td>
<td>85</td>
<td>0</td>
<td>82</td>
<td>17</td>
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<tr>
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<td>190</td>
<td>85</td>
<td>148</td>
<td>10</td>
</tr>
<tr>
<td>Jensen (2000) [19]</td>
<td>121</td>
<td>100</td>
<td>121</td>
<td>10a</td>
</tr>
<tr>
<td>Angtuaco (2001) [20]</td>
<td>39</td>
<td>100</td>
<td>29</td>
<td>4</td>
</tr>
<tr>
<td>Green (2005) [21]</td>
<td>50</td>
<td>100</td>
<td>48</td>
<td>17</td>
</tr>
<tr>
<td>Total</td>
<td>485</td>
<td>–</td>
<td>438 (88%)</td>
<td>58 (12%)</td>
</tr>
</tbody>
</table>

*Reported only patients who received therapy specifically for diverticular lesions.
Jenson and colleagues [19] employed these hemostasis techniques in the treatment of 10 patients who had either active or stigmata of diverticular hemorrhage. There was a 100% success rate and no early or late rebleeding. Bloomfield and colleagues [25] employed similar hemostasis techniques to 12 patients with diverticular hemorrhage, but one patient experienced early rebleeding, and four had late rebleeding. In a prospective study comparing urgent colonoscopy with angiography-based therapy by Green and colleagues [21], 13 patients who had diverticular hemorrhage were treated with the same endoscopic techniques of epinephrine injection or multi-polar electrocoagulation. Although treatment initially was successful in all patients, two had early rebleeding, and two had late rebleeding. Although the efficacy of epinephrine or multi-polar electrocautery for diverticular hemorrhage has varied, it appears to be safe, as no patients have suffered complications. Numerous case reports and small series also have reported the successful treatment of diverticular hemorrhage with various combinations of epinephrine or multi-polar electrocautery (Fig. 1, Table 2) [17–21,25–32]. Unfortunately, the follow-up has been limited in many of the smaller series.

![Fig. 1. Treatment of diverticular bleeding— injection and thermal therapy. Shown is a visible vessel associated with a diverticula (A). (B) The lesion after injection with epinephrine (5 cc 1:10,000), and (C) after thermal therapy. (From Bloomfeld RS, Rockey DC, Shetzline MA. Endoscopic therapy of acute diverticular hemorrhage. Am J Gastroenterol 2001;96:2369; with permission.)](image-url)
Innovative therapeutic endoscopists have employed various other techniques to control diverticular hemorrhage [33–35]. Hokama and colleagues [34] described three patients successfully treated with an endoscopic hemoclip, and the clinical experience with hemoclips suggests that this approach is likely to be effective (Fig. 2). Farrell and colleagues [35] described the successful use of endoscopic band ligation to control diverticular hemorrhage in four patients. Although these techniques are exciting, they remain experimental and not ready for widespread application. In contrast, the safety and efficacy of endoscopic hemostasis with epinephrine injection or multi-polar electrocautery are

<table>
<thead>
<tr>
<th>Study (year)</th>
<th>Study type</th>
<th>N</th>
<th>Rebleeding</th>
<th>Treatment</th>
</tr>
</thead>
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<tr>
<td>Green (2005) [21]</td>
<td>Prospective</td>
<td>17</td>
<td>4</td>
<td>Epinephrine/coagulation</td>
</tr>
<tr>
<td>Jensen (2000) [17]</td>
<td>Prospective</td>
<td>10</td>
<td>0</td>
<td>Epinephrine/coagulation</td>
</tr>
<tr>
<td>Prakash (1999) [25]</td>
<td>Case series</td>
<td>3</td>
<td>0</td>
<td>Coagulation</td>
</tr>
<tr>
<td>Ramirez (1996) [27]</td>
<td>Case series</td>
<td>4</td>
<td>0</td>
<td>Epinephrine</td>
</tr>
<tr>
<td>Savides (1994) [31]</td>
<td>Case series</td>
<td>3</td>
<td>0</td>
<td>Coagulation</td>
</tr>
<tr>
<td>Foutch (1996) [33]</td>
<td>Case series</td>
<td>4</td>
<td>1</td>
<td>Coagulation</td>
</tr>
<tr>
<td>Johnston (1986) [26]</td>
<td>Case series</td>
<td>4</td>
<td>0</td>
<td>Coagulation</td>
</tr>
</tbody>
</table>

Fig. 2. Treatment of diverticular bleeding—injection and hemoclip therapy. (A) Visible vessel inside of a diverticulum. (B) After epinephrine injection therapy, the lesion is approached with a hemoclip. (C) Lesion is clipped. (D) Hemoclip firmly placed on the visible vessel. (Courtesy of Louis Wang and the Mayo Clinic Bleeding Team.)
Angiography

Angiography can identify and localize bleeding accurately, but this requires a bleeding rate of at least 0.5 to 1.0 mL/min to be positive. This sometimes may be a problem because of the intermittent nature of bleeding. It, however, has the advantage of not requiring time to prepare the colon. When the bleeding is identified, it may be treated with intra-arterial infusion of vasopressin or super selective embolization with various agents (gelatin sponge, microcoils, and polyvinyl alcohol particles). Vasopressin was the first modality employed, and it controlled bleeding in up to 91% of cases [37,38]. Unfortunately, the major complication rate was 10% to 20% and included arrhythmias, pulmonary edema, hypertension, and ischemia [39,40]. Rebleeding also occurred in up to 50% of patients after cessation of the infusion [37]. Early attempts at embolization occasionally caused bowel infarction, but current super-selective techniques have made this unusual [39,40]. Selective embolization initially controls hemorrhage in up to 100% of patients, but rebleeding rates are 15% to 40% [39,40]. A recent study by Debaros and colleagues, involving 27 patients undergoing super-selective arterial embolization, found an initial success rate of 100% and a rebleeding rate of only 7% [41]. Another recent study by Burgess and Evans [42] in 15 patients found an initial success rate of 93%, but 53% had rebleeding within 24 hours. Kuo and colleagues [42] achieved immediate hemostasis in 100% of 22 patients, and only 14% experienced rebleeding. A literature review of 144 cases of super-selective arterial embolization found a minor complication rate of 9% and a 0% major complication rate [42]. Although most patients in these studies were bleeding from diverticula, several patients with vascular malformations of the colon and small bowel were included. Because angiographic therapy is felt to be equally effective for both lesions, there is no reason to suspect that this would alter the findings appreciably if diverticular hemorrhage was considered alone.

Surgery

Surgery usually is employed for hemorrhage in two settings: massive or recurrent bleeding. It is required in 15% to 25% of patients who have diverticular bleeding and is recommended for patients with a high transfusion requirement (generally more than four units within a 24-hour period or greater than 10 units total) [43]. Recurrent bleeding from diverticula occurs in 20% to 40% of patients and generally is considered an indication for surgery [44]. In patients with serious comorbid medical conditions and without exsanguinating hemorrhage, this decision should be made carefully. Great effort should be made to accurately localize the site of bleeding preoperatively so that segmental rather than subtotal colectomy can be performed. If the specific bleeding diverticulum can be identified during colonoscopy, the adjacent mucosa should be labeled
with India ink so that it can be localized if surgery becomes necessary. Surgical therapy generally is not recommended on the basis of tagged RBC scintigraphy alone, because of variable accuracy of red blood cell (RBC) bleeding scans [45–47]. Tagged RBC scintigraphy is used most often as a screening test before visceral angiography. Although one study [48] showed this policy increased the yield of angiography from 22% to 53%, other studies [49] have had confounding results. The role of RBC scintigraphy in acute LGIB is uncertain and requires further investigation. Whenever possible, it is preferable to perform surgery on an elective basis rather than emergently. Operative mortality is 10% even with accurate localization and up to 57% with blind subtotal colectomy [50–52].

**MANAGEMENT OF SPECIFIC CAUSES OF LOWER GASTROINTESTINAL BLEEDING**

**Diverticula**

Diverticula are the most common source of acute LGIB (see the article by Strate in this issue). Despite this, there is a paucity of prospective clinical data on specific treatment strategies, and moreover, the ones that are published are primarily case series and small, often nonrandomized studies (see Table 2). The traditional management of diverticular bleeding largely has been supportive, with colonoscopy performed after bleeding had ceased and the colon adequately prepared. Unfortunately, the detection rate of actively or recently bleeding lesions with expectant colonoscopy was low, thus limiting the ability of interventional endoscopic hemostasis to prove benefit. Nonetheless, the available data suggest that endoscopic therapy for diverticular hemorrhage is safe and likely to be beneficial.

It is notable that while in at least 75% of patients, diverticular bleeding stops spontaneously, those in whom it persists or recurs are difficult to manage (and they typically require surgery), and thus, often suffer substantial morbidity and mortality.

**Vascular Ectasia**

Vascular ectasia most often are located in the right colon. They are found frequently in elderly patients, particularly those with chronic underlying medical conditions, most notably renal failure. Their multifocal tendency and frequent location in the right colon can make management difficult. Lesions in the colon often present with intermittent hematochezia, while small bowel lesions more often cause occult blood loss and iron deficiency anemia.

Colonoscopy is relatively accurate for identifying vascular ectasias, but unless stigmata of bleeding are found, it is difficult to clearly identify the offending lesion [53]. A poor bowel preparation or the transient decrease in mucosal blood flow associated with narcotic medications used for sedation can make identification difficult [54,55]. Naloxone may enhance their colonoscopic appearance [55]. The best treatment for vascular ectasias is likely therapeutic endoscopy with any of a variety of techniques (ie, electrocoagulation, injection
therapy, laser, or argon plasma coagulation) [56]. When multiple lesions are present (and the offending one cannot be ascertained), all should be treated. When contact coagulation is performed on a large angioectasia, the outer margin should be treated first to obliterate feeding vessels and prevent brisk bleeding. Care is needed, because the risk of perforation, especially in the right colon, is significant [57]. Because of this risk, argon plasma coagulation is becoming popular as a noncontact and presumably safer method of treatment [57,58]. Sclerosing agents such as ethanolamine also can be injected, but they are not used widely [59].

Angiography also can identify and treat bleeding from vascular ectasias by intra-arterial vasopressin or embolization techniques [60–62]. Angiography is the treatment of choice for angioectasia in the ileum and jejunum. The intermittent and often obscure nature of bleeding from distal small bowel angioectasia has prompted the use of provocative angiography at some specialty centers. This involves the selective and careful infusion of heparin or thrombolytic agents into the mesenteric vessels supplying the suspected area of bowel in an effort to provoke bleeding from the index lesion, thus allowing therapy [63]. This technique can be employed for bleeding from various sources but requires great expertise and immediate surgical back-up. Hormonal therapy (estrogen) to reduce the frequency of bleeding from multi-focal angioectasia has been studied but was ineffective in recent studies [64,65]. Surgery is rarely necessary, but it is required for uncontrollable or recurrent bleeding. As with diverticular bleeding, surgical outcomes are best when the index lesion has been localized previously to a specific portion of the colon.

**Hemorrhoids**

Hemorrhoids are common and account for 5% to 10% of acute LGIB, but they rarely cause massive bleeding. Hematochezia should not be ascribed to hemorrhoids without examining the colon. Conservative management with sitz baths, avoidance of straining, and dietary modification are usually effective, although surgical hemorrhoidectomy and rubber band ligation are options for refractory cases [66].

**Ischemic Colitis**

Ischemic colitis is an increasingly recognized cause of acute lower gastrointestinal hemorrhage. Bleeding is usually mild, diffuse, and infrequently of hemodynamic significance. Treatment is supportive with antibiotics, bowel rest, intravenous fluids, optimization of hemodynamic status, and correction of the precipitating condition [67]. The role of endoscopy is principally to aid in the diagnosis of lesions rather than to treat them. When surgery is necessary, it is most often because of transmural infarction with necrosis rather than bleeding.

**Inflammatory and Infectious Colitis**

Colitis can be caused by numerous different diseases; each of these is a potential cause of LGIB. Inflammatory bowel disease is the most frequent. Both
ulcerative colitis and Crohn’s disease can cause severe LGIB [68]. Bleeding is usually self-limited and responds to medical therapy. Infliximab has been used successfully to avert emergency surgery in Crohn’s patients who have severe bleeding [69,70]. An endoscopically treatable lesion is uncommon, and surgical intervention may be necessary, especially in patients with recurrent hemorrhage. Patients who have ulcerative colitis are treated best with total colectomy, while patients who have Crohn’s disease should have only the diseased segment removed.

Numerous infectious agents can penetrate and injure the colonic mucosa and cause acute LGIB. The principle use of endoscopy is to visualize the mucosa and obtain biopsies to guide the use of antimicrobial agents. Endoscopic therapy with epinephrine injection or multi-polar electrocoagulation is helpful when a precise focus of bleeding is present. In populations with immunosuppression, such as patients who have HIV [71], renal transplant patients [72], or pancreatic transplant patients [73], LGIB often is caused by cytomegalovirus (CMV) ulcers, which often are treated with endoscopic hemostasis followed by medical therapy for CMV.

Radiation Colitis
Radiation therapy to the colon (most commonly the rectum) induces inflammatory changes and can produce radiation colitis. Many different medical

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**Fig. 3.** Management of acute lower gastrointestinal bleeding. One potential algorithm is suggested. In this approach, urgent colonoscopy is advocated. Urgent is taken to mean within 8 hours of presentation. The evidence to support this approach is based on cumulative literature and the author’s experience.
therapies, including steroids, hyperbaric oxygen, 5-aminosalicylic acid compounds, and sucralfate have been used to treat radiation proctitis, but little data support their effectiveness [74–76]. Endoscopic therapy with either laser or argon beam coagulation is the most effective treatment [77–81]. A recent study [82] found an 87% success rate with a mean of 2.3 sessions of therapy. Severe complications (severe bleeding, extensive necrosis of the rectum, or perforation) occurred in 10% of patients. Complications can be reduced by using a power setting of less than 45 watts and by treating individual telangiectases rather than diffusely painting the mucosa.

Postpolypectomy Bleeding
Brisk bleeding immediately after polypectomy is usually arterial and can be controlled by resnaring the polyp stalk and holding pressure [83,84]. Delayed bleeding is caused by sloughing of the eschar, and this usually occurs 1 to 2 weeks later. The use of NSAIDS may increase the frequency of this. Delayed bleeding is self-limited in most cases, but persistent bleeding can be treated with various endoscopic techniques. These include injection of epinephrine followed by thermal therapy, loop or band ligation of the remaining polyp stalk, and hemoclips [85–91]. All of these techniques are safe and effective.

SUMMARY
Although acute LGIB is only about one fifth as common and is usually less hemodynamically significant than upper gastrointestinal bleeding, it presents numerous unique clinical challenges. The best diagnostic approach for patients with active bleeding is unknown, but urgent prepared colonoscopy is safe and likely to be beneficial (Fig. 3, Table 2). In patients who have aggressive bleeding or recurrent bleeding, it is critical for the practitioner to judge when angiography and surgery are necessary.

References


