Approach to acute upper gastrointestinal bleeding in adults

Author
John R Saltzman, MD, FACP, FACG, FASGE, AGAF

Section Editor
Mark Feldman, MD, MACP, AGAF, FACG

Deputy Editor
Anne C Travis, MD, MSc, FACG, AGAF

All topics are updated as new evidence becomes available and our peer review process is complete.
Literature review current through: Aug 2014. | **This topic last updated:** May 16, 2014.

INTRODUCTION — Patients with acute upper gastrointestinal (GI) bleeding commonly present with hematemesis (vomiting of blood or coffee-ground like material) and/or melena (black, tarry stools). The initial evaluation of patients with acute upper GI bleeding involves an assessment of hemodynamic stability and resuscitation if necessary. Diagnostic studies (usually endoscopy) follow, with the goal of both diagnosis, and when possible, treatment of the specific disorder.

The diagnostic and initial therapeutic approach to patients with clinically significant (ie, the passage of more than a scant amount of blood) acute upper GI bleeding will be reviewed here. This approach is consistent with a multidisciplinary international consensus statement updated in 2010, a 2012 guideline issued by the American Society for Gastrointestinal Endoscopy, and a 2012 guideline issued by the American College of Gastroenterology [1-4]. The causes of upper GI bleeding, the endoscopic management of acute upper GI bleeding, and the management of active variceal hemorrhage are discussed separately. (See "Major causes of upper gastrointestinal bleeding in adults" and "Uncommon causes of upper gastrointestinal bleeding in adults" and "Overview of the treatment of bleeding peptic ulcers" and "General principles of the management of variceal hemorrhage" and "Methods to achieve hemostasis in patients with acute variceal hemorrhage".)

A table outlining the emergency management of acute severe upper gastrointestinal bleeding is provided (table 1).

INITIAL EVALUATION — The initial evaluation of a patient with a suspected clinically significant acute upper GI bleed includes a history, physical examination, laboratory tests, and in some cases, nasogastric lavage. The goal of the evaluation is to assess the severity of the bleed, identify potential sources of the bleed, and determine if there are conditions present that may affect subsequent management. The information gathered as part of the initial evaluation is used to guide decisions regarding triage, resuscitation, empiric medical therapy, and diagnostic testing.

Factors that are predictive of a bleed coming from an upper GI source identified in a meta-analysis included a patient-reported history of melena (likelihood ratio [LR] 5.1-5.9), melenic stool on examination (LR 25), blood or coffee grounds detected during nasogastric lavage (LR 9.6), and a ratio of blood urea nitrogen to serum creatinine greater than 30 (LR 7.5) [5]. On the other hand, the presence of blood clots in the stool made an upper GI source less likely (LR 0.05). Factors associated with severe bleeding included red blood detected during nasogastric lavage (LR 3.1), tachycardia (LR 4.9), or a hemoglobin level of less than 8 g/dL (LR 4.5-6.2).

Bleeding manifestations — Hematemesis (either red blood or coffee-ground emesis) suggests bleeding proximal to the ligament of Treitz. The presence of frankly bloody emesis suggests moderate to severe bleeding that may be ongoing, whereas coffee-ground emesis suggests more limited bleeding.

The majority of melena (black, tarry stool) originates proximal to the ligament of Treitz (90 percent), though it may also originate from the small bowel or right colon [6]. Melena may be seen with variable degrees of blood loss, being seen

with as little as 50 mL of blood [7].

Hematochezia (red or maroon blood in the stool) is usually due to lower GI bleeding. However, it can occur with massive upper GI bleeding [8], which is typically associated with orthostatic hypotension. (See 'Physical examination' below.)

Past medical history — Patients should be asked about prior episodes of upper GI bleeding, since up to 60 percent of patients with a history of an upper GI bleed are bleeding from the same lesion [9]. In addition, the patient's past medical history should be reviewed to identify important comorbid conditions that may lead to upper GI bleeding or may influence the patient's subsequent management.

Potential bleeding sources suggested by a patient's past medical history include:

- Varices or portal hypertensive gastropathy in a patient with a history of liver disease or alcohol abuse
- Aorto-enteric fistula in a patient with a history of an abdominal aortic aneurysm or an aortic graft
- Angiodysplasia in a patient with renal disease, aortic stenosis, or hereditary hemorrhagic telangiectasia
- Peptic ulcer disease in a patient with a history of *Helicobacter pylori*, nonsteroidal anti-inflammatory drug (NSAIDs) use, or smoking
- Malignancy in a patient with a history of smoking, alcohol abuse, or *H. pylori* infection
- Marginal ulcers(ulcers at an anastomotic site) in a patient with a gastroenteric anastomosis

Comorbid illnesses may influence patient management in the setting of an acute upper GI bleed. Comorbid illnesses may:

- Make patients more susceptible to hypoxemia (eg, coronary artery disease, pulmonary disease). Such patients may need to be maintained at higher hemoglobin levels than patients without these disorders. (See 'Blood transfusions' below.)
- Predispose patients to volume overload in the setting of fluid resuscitation or blood transfusions (eg, renal disease, heart failure). Such patients may need more invasive monitoring during resuscitation. (See 'General support' below.)
- Result in bleeding that is more difficult to control (eg, coagulopathies, thrombocytopenia, significant hepatic dysfunction). Such patients may need transfusions of fresh frozen plasma or platelets. (See 'Blood transfusions' below.)
- Predispose to aspiration (eg, dementia, hepatic encephalopathy). Endotracheal intubation should be considered in such patients. (See 'General support' below.)

Medication history — A thorough medication history should be obtained, with particular attention paid to drugs that:

- Predispose to peptic ulcer formation, such as aspirin and other nonsteroidal antiinflammatory drugs (NSAIDs) (see "NSAIDs (including aspirin): Pathogenesis of gastroduodenal toxicity")
- Are associated with pill esophagitis (see "Medication-induced esophagitis")
- Promote bleeding, such as antiplatelet agents (eg, clopidogrel) and anticoagulants
- May alter the clinical presentation, such as bismuth and iron, which can turn the stool black

Symptom assessment — Patients should be asked about symptoms as part of the assessment of the severity of the bleed and as a part of the evaluation for potential bleeding sources. Symptoms that suggest the bleeding is severe

include orthostatic dizziness, confusion, angina, severe palpitations, and cold/clammy extremities.

Specific causes of upper GI bleeding may be suggested by the patient's symptoms [6]:

- Peptic ulcer: Epigastric or right upper quadrant pain
- Esophageal ulcer: Odynophagia, gastroesophageal reflux, dysphagia
- Mallory-Weiss tear: Emesis, retching, or coughing prior to hematemesis
- Variceal hemorrhage or portal hypertensive gastropathy: Jaundice, weakness, fatigue, anorexia, abdominal distention
- Malignancy: Dysphagia, early satiety, involuntary weight loss, cachexia

Physical examination — The physical examination is a key component of the assessment of hemodynamic stability. Signs of hypovolemia include [6]:

- Mild to moderate hypovolemia: Resting tachycardia
- Blood volume loss of at least 15 percent: Orthostatic hypotension (a decrease in the systolic blood pressure of more than 20 mmHg and/or an increase in heart rate of 20 beats per minute when moving from recumbency to standing)
- Blood volume loss of at least 40 percent: Supine hypotension

Examination of the stool color may provide a clue to the location of the bleeding, but it is not a reliable indicator. In a series of 80 patients with severe hematochezia (red or maroon blood in the stool), 74 percent had a colonic lesion, 11 percent had an upper GI lesion, 9 percent had a presumed small bowel source, and no site was identified in 6 percent [8]. Nasogastric lavage may be carried out if there is doubt as to whether a bleed originates from the upper GI tract. (See 'Nasogastric lavage' below.)

The presence of abdominal pain, especially if severe and associated with rebound tenderness or involuntary guarding, raises concern for perforation. If any signs of an acute abdomen are present, further evaluation to exclude a perforation is required prior to endoscopy. (See "Diagnostic approach to abdominal pain in adults", section on 'Surgical abdomen'.)

Finally, as with the past medical history, the physical examination should include a search for evidence of significant comorbid illnesses. (See 'Past medical history' above.)

Laboratory data — Laboratory tests that should be obtained in patients with acute upper gastrointestinal bleeding include a complete blood count, serum chemistries, liver tests, and coagulation studies. In addition, serial electrocardiograms and cardiac enzymes may be indicated in patients who are at risk for a myocardial infarction, such as the elderly, patients with a history of coronary artery disease, or patients with symptoms such as chest pain or dyspnea. (See "Criteria for the diagnosis of acute myocardial infarction".)

The initial hemoglobin in patients with acute upper GI bleeding will often be at the patient's baseline because the patient is losing whole blood. With time (typically after 24 hours or more) the hemoglobin will decline as the blood is diluted by the influx of extravascular fluid into the vascular space and by fluid administered during resuscitation. It should be kept in mind that overhydration can lead to a falsely low hemoglobin value. The initial hemoglobin level is monitored every two to eight hours, depending upon the severity of the bleed.

Patients with acute bleeding should have normocytic red blood cells. Microcytic red blood cells or iron deficiency anemia suggest chronic bleeding. Because blood is absorbed as it passes through the small bowel and patients may have decreased renal perfusion, patients with acute upper GI bleeding typically have an elevated blood urea nitrogen (BUN)-

to-creatinine or urea-to-creatinine ratio (>20:1 or >100:1, respectively) [10,11]. The higher the ratio, the more likely the bleeding is from an upper GI source [10].

Nasogastric lavage — Whether all patients with suspected acute upper GI bleeding require nasogastric tube (NGT) placement is controversial, in part because studies have failed to demonstrate a benefit with regard to clinical outcomes [12]. As an example, a retrospective study looked at whether there were clinical benefits from NGT lavage in 632 patients admitted with gastrointestinal bleeding [13]. Patients who underwent NGT lavage were matched with patients with similar characteristics who did not undergo NGT lavage. NGT lavage was associated with a shorter time to endoscopy. However, there were no differences between those who underwent NGT lavage and those who did not with regard to mortality, length of hospital stay, surgery, or transfusion requirement.

More often, NGT lavage is used when it is unclear if a patient has ongoing bleeding and thus might benefit from an early endoscopy. In addition, nasogastric tube lavage can be used to remove particulate matter, fresh blood, and clots from the stomach to facilitate endoscopy. (See "Nasogastric and nasoenteric tubes", section on 'Tube placement'.)

The presence of red blood or coffee ground material in the aspirate also confirms an upper GI source of bleeding and predicts whether the bleeding is caused by a lesion at increased risk for ongoing or recurrent bleeding [13,14]. However, lavage may not be positive if bleeding has ceased or arises beyond a closed pylorus. The presence of nonbloody bilious fluid suggests that the pylorus is open and that there is no active upper GI bleeding distal to the pylorus [8].

We suggest that patients only undergo NGT lavage if particulate matter, fresh blood, or clots need to be removed from the stomach to facilitate endoscopy.

GENERAL MANAGEMENT

Triage — All patients with hemodynamic instability (shock, orthostatic hypotension) or active bleeding (manifested by hematemesis, bright red blood per nasogastric tube, or hematochezia) should be admitted to an intensive care unit for resuscitation and close observation with automated blood pressure monitoring, electrocardiogram monitoring, and pulse oximetry.

A table outlining the emergency management of acute severe upper gastrointestinal bleeding is provided (table 1).

Other patients can be admitted to a regular medical ward, though we suggest that all admitted patients with the exception of low-risk patients receive electrocardiogram monitoring. Outpatient management may be appropriate for some low-risk patients. (See 'Risk stratification' below.)

General support — Patients should receive supplemental oxygen by nasal cannula and should receive nothing per mouth. Two large caliber (16 gauge or larger) peripheral intravenous catheters or a central venous line should be inserted and placement of a pulmonary artery catheter should be considered in patients with hemodynamic instability or who need close monitoring during resuscitation. (See "Pulmonary artery catheterization: Indications and complications".)

Elective endotracheal intubation in patients with ongoing hematemesis or altered respiratory or mental status may facilitate endoscopy and decrease the risk of aspiration.

Fluid resuscitation — Adequate resuscitation and stabilization is essential prior to endoscopy to minimize treatment-associated complications [15]. Patients with active bleeding should receive intravenous fluids (eg, 500 mL of normal saline or lactated Ringer's solution over 30 minutes) while being typed and cross-matched for blood transfusion. Patients at risk of fluid overload may require intensive monitoring with a pulmonary artery catheter.

If the blood pressure fails to respond to initial resuscitation efforts, the rate of fluid administration should be increased.

Blood transfusions — The decision to initiate blood transfusions must be individualized. Our approach is to initiate blood transfusions if the hemoglobin is <7 g/dL (70 g/L) for most patients (including those with stable coronary artery

disease), with a goal of maintaining the hemoglobin at a level ≥7 g/dL (70 g/L) [4,16-18]. However, our goal is to maintain the hemoglobin at a level of ≥9 g/dL (90 g/L) for patients at increased risk of suffering adverse events in the setting of significant anemia, such as those with unstable coronary artery disease. We do not have an age cutoff for determining which patients should have a goal hemoglobin of ≥9 g/dL (90 g/L), and instead base the decision on the patient's comorbid conditions. However, patients with active bleeding and hypovolemia may require blood transfusion despite an apparently normal hemoglobin. (See 'Laboratory data' above.)

It is particularly important to avoid overtransfusion in patients with suspected variceal bleeding, as it can precipitate worsening of the bleeding [17,19-22]. Transfusing patients with suspected variceal bleeding to a hemoglobin >10 g/dL (100 g/L) should be avoided. (See "General principles of the management of variceal hemorrhage", section on 'Hemodynamic resuscitation'.)

A randomized trial suggests that using a lower hemoglobin threshold for initiating transfusion improves outcomes. In the trial, 921 adults with acute upper GI bleeding were assigned to either a restrictive transfusion strategy (transfusion only when the hemoglobin fell to <7 g/dL [70 g/L]) or a liberal transfusion strategy (transfusion when the hemoglobin fell to <9 g/dL [90 g/L]) [17].

Patients were excluded if they had massive exsanguinating bleeding, an acute coronary syndrome, symptomatic peripheral vasculopathy, stroke, transient ischemic attack, transfusion within the prior 90 days, recent trauma or surgery, a Rockall score of 0 with a hemoglobin level higher than 12 g/dL (120 g/L, patients at low risk of further bleeding), or if the attending clinician previously decided that a patient should avoid a specific medical therapy. Older age was not an exclusion criterion, and the mean age of patients was 64 years in the restrictive group and 66 years in the liberal group. In addition, cirrhosis was not an exclusion criterion (31 percent of the patients in the study had cirrhosis). The majority of patients in the restrictive and liberal transfusion groups had bleeding due to a peptic ulcer (51 and 47 percent, respectively), followed by variceal bleeding (23 and 24 percent, respectively).

Patients in the restrictive group were more likely than those in the liberal group to avoid transfusion (51 versus 14 percent) and received fewer units of blood (mean 1.5 versus 3.8 units). Mortality was lower in the restrictive strategy group (5 versus 9 percent, adjusted hazard ratio 0.55, 95% confidence interval 0.33-0.92). Patients in the restrictive group were also less likely to have further bleeding or to suffer complications. Among patients with cirrhosis, the risks of death and further bleeding were lower with the restrictive strategy for patients with Child A or B cirrhosis, but were similar for patient with Child C cirrhosis.

It is important to note, however, that all the patients in this study underwent emergent upper endoscopy with a mean duration from admission to upper endoscopy of 5 hours. Endoscopic therapy was given to those with active bleeding, a nonbleeding visible vessel, an adherent clot, or bleeding esophageal varices. It is not clear whether similar results would be seen in patients who do not receive an upper endoscopy and endoscopic therapy as quickly as the patients included in this study. Theoretically, patients treated with a restrictive transfusion strategy may have worse outcomes in the setting of delayed endoscopy with ongoing bleeding.

A retrospective study also suggested that outcomes may be worse in patients who receive blood transfusions. The study included 1677 patients with nonvariceal upper GI bleeding [23]. While not associated with mortality, blood transfusion within the first 24 hours of presentation was independently associated with an increased risk of rebleeding after adjusting for factors such as hemodynamic instability, endoscopic therapy, high-risk stigmata of recurrent hemorrhage, initial hemoglobin value, and the presence of blood on rectal examination or in the nasogastric tube aspirate (OR 1.8, 95% CI 1.2-2.8).

Patients with active bleeding and a coagulopathy (prolonged prothrombin time with INR >1.5) or low platelet count (<50,000/microL) should be transfused with fresh frozen plasma (FFP) or platelets, respectively. Provided the patient is hemodynamically stable, urgent endoscopy can usually proceed simultaneously with transfusion and should not be

postponed until the coagulopathy is corrected. However, in patients with an INR ≥3, we attempt to correct the INR to <3 prior to starting an endoscopy, with additional FFP being given after the endoscopy if high-risk stigmata for recurrent bleeding were found or if endoscopic therapy was performed and the INR is still >1.5. This approach is based on data that suggest endoscopy is safe in patients who are mildly to moderately anticoagulated [24]. In addition, because packed red blood cells do not contain coagulation factors, transfusion of a unit of FFP should be considered after every four units of packed red blood cells [25].

Platelet transfusions should also be considered in patients with life-threatening bleeding who have received antiplatelet agents such as aspirin or clopidogrel [26]. If the patient is taking the medications because of a recent (<1 year) vascular stent placement or acute coronary syndrome, when possible, a cardiologist should be consulted prior to stopping the agent or giving a platelet transfusion.

Medications

Acid suppression — Patients admitted to the hospital with acute upper GI bleeding are typically treated with a proton pump inhibitor (PPI). We suggest that patients with acute upper GI bleeding be started empirically on an intravenous PPI. It can be started at presentation and continued until confirmation of the cause of bleeding. Once the source of the bleeding has been identified and treated (if possible), the need for ongoing acid suppression can be determined.

Several studies have examined the role of acid suppression given before or after endoscopy (with or without therapeutic intervention) [27]. In the setting of active upper GI bleeding from an ulcer, acid suppressive therapy with H2 receptor antagonists has **not** been shown to significantly lower the rate of ulcer rebleeding [28-30]. By contrast, high dose antisecretory therapy with an intravenous infusion of a PPI significantly reduces the rate of rebleeding compared with standard treatment in patients with bleeding ulcers [31]. Oral and intravenous PPI therapy also decrease the length of hospital stay, rebleeding rate, and need for blood transfusion in patients with high-risk ulcers treated with endoscopic therapy. (See "Overview of the treatment of bleeding peptic ulcers", section on 'Acid suppression'.)

PPIs may also promote hemostasis in patients with lesions other than ulcers. This likely occurs because neutralization of gastric acid leads to the stabilization of blood clots [32].

An intravenous PPI given before endoscopic therapy in patients with acute upper GI bleeding can reduce signs of bleeding and the need for endoscopic therapy. One of the most methodologically rigorous controlled trials evaluating this approach included 638 patients with acute upper GI bleeding who were randomly assigned to intravenous omeprazole or placebo before endoscopy [33]. Patients randomized to omeprazole had significantly shorter lengths of stay, fewer actively bleeding ulcers (6 versus 15 percent), and more ulcers with a clean base. There was no significant difference in the proportion needing surgery or with recurrent bleeding. A possible limitation of the study is that multiple outcomes were examined, without a statistical correction for the multiple comparisons. In a cost-effectiveness analysis, administering an intravenous PPI before endoscopy was slightly more costly but more effective than administering it after endoscopy [34].

Although omeprazole has been the most extensively studied intravenous PPI, other intravenous formulations given in doses that are known to inhibit gastric acid secretion are probably acceptable alternatives. Pantoprazole and esomeprazole are the only intravenous formulations available in the United States, and lansoprazole, which was previously available, has been removed from the world market. The suggested dose of intravenous pantoprazole is 80 mg bolus followed by 8 mg/hr infusion. Omeprazole and esomeprazole have also been used as 80 mg boluses followed by 8 mg/hr infusions. The infusion is usually continued for 72 hours. If there is no rebleeding the patient may be switched to oral pantoprazole 40 mg/day or omeprazole 20 mg/day. Twice daily dosing of an oral proton pump inhibitor may be a reasonable alternative if intravenous formulations are not available. (See "Overview of the treatment of bleeding peptic ulcers", section on 'High-dose versus non-high-dose PPIs'.)

Prokinetics — Both erythromycin and metoclopramide have been studied in patients with acute upper GI bleeding. The goal of using a prokinetic agent is to improve gastric visualization at the time of endoscopy by clearing the stomach of blood, clots, and food residue. We suggest that erythromycin be considered in patients who are likely to have a large amount of blood in their stomach, such as those with severe bleeding. A reasonable dose is 3 mg/kg intravenously over 20 to 30 minutes, 30 to 90 minutes prior to endoscopy.

Erythromycin promotes gastric emptying based upon its ability to be an agonist of motilin receptors. Using erythromycin to improve gastric visualization has been studied in at least four randomized controlled trials [35-38]. The studies suggested that a single dose of intravenous erythromycin given 20 to 120 minutes before endoscopy can significantly improve visibility, shorten endoscopy time, and reduce the need for second-look endoscopy. Treatment appears to be safe.

A meta-analysis examined five trials with 316 patients who were assigned to erythromycin, metoclopramide, or placebo [39]. The analysis found that the use of a prokinetic agent decreased the need for second-look endoscopy, but did not affect the number of units of blood transfused, length of hospital stay, or need for surgery. In subgroup analyses, erythromycin continued to show a benefit with regard to the need for second-look endoscopy, but metoclopramide did not.

A second meta-analysis examined four trials with 335 patients who were assigned to either erythromycin or a control group [40]. The meta-analysis found that patients who received erythromycin were significantly more likely to have an empty stomach at the time of endoscopy compared with patients in the control group (69 versus 37 percent). Patients treated with erythromycin also had significant reductions in the need for second endoscopy, volume of blood transfused, and length of hospital stay. Finally, there was a trend toward shorter endoscopic procedure times and decreased mortality for patients treated with erythromycin.

Erythromycin has also been compared with nasogastric lavage. A randomized trial with 253 patients that compared erythromycin alone with nasogastric lavage alone and nasogastric lavage plus erythromycin found that the quality of visualization did not differ significantly among the three groups [41]. In addition, there were no differences among the groups with regard to procedure duration, rebleeding rates, need for second endoscopy, number of transfused units of blood, and mortality.

Somatostatin and its analogs — Somatostatin, or its analog octreotide, is used in the treatment of variceal bleeding and may also reduce the risk of bleeding due to nonvariceal causes [42]. In patients with suspected variceal bleeding, octreotide is given as an intravenous bolus of 20 to 50 mcg, followed by a continuous infusion at a rate of 25 to 50 mcg per hour. (See "Methods to achieve hemostasis in patients with acute variceal hemorrhage", section on 'Somatostatin and its analogs'.)

Octreotide is not recommended for routine use in patients with acute nonvariceal upper GI bleeding, but it can be used as adjunctive therapy in some cases. Its role is generally limited to settings in which endoscopy is unavailable or as a means to help stabilize patients before definitive therapy can be performed. (See "Overview of the treatment of bleeding peptic ulcers", section on 'Somatostatin and octreotide'.)

Antibiotics for patients with cirrhosis — Bacterial infections are present in up to 20 percent of patients with cirrhosis who are hospitalized with gastrointestinal bleeding; up to an additional 50 percent develop an infection while hospitalized. Such patients have increased mortality.

Multiple trials evaluating the effectiveness of prophylactic antibiotics in cirrhotic patients hospitalized for bleeding suggest an overall reduction in infectious complications and possibly decreased mortality. Antibiotics may also reduce the risk of recurrent bleeding in hospitalized patients who bled from esophageal varices. A reasonable conclusion from these data is that patients with cirrhosis who present with acute upper GI bleeding (from varices or other causes) should be given prophylactic antibiotics, preferably before endoscopy (although effectiveness has also been demonstrated

when given after endoscopy). (See "General principles of the management of variceal hemorrhage", section on 'Infection and use of prophylactic antibiotics'.)

Tranexamic acid — Tranexamic acid is an antifibrinolytic agent that has been studied in patients with upper GI bleeding. A meta-analysis that included seven randomized trials of tranexamic acid in patients with upper GI bleeding found a benefit with regard to mortality but not with regard to bleeding, surgery, or transfusion requirements [43]. When only studies that used antiulcer drugs and/or endoscopic therapy were included, there was no beneficial effect. This suggests that there is no role for tranexamic acid in the treatment of upper GI bleeding, since the current standard of care is to treat patients with proton pump inhibitors and endoscopic therapy (if indicated).

Anticoagulants and antiplatelet agents — When possible, anticoagulants and antiplatelet agents should be held in patients with upper GI bleeding. However, the thrombotic risk of reversing anticoagulation should be weighed against the risk of continued bleeding without reversal, and thus the decision to discontinue medications or administer reversal agents needs to be individualized. In some cases (eg, stopping a nonsteroidal anti-inflammatory drug in a patient who is taking it for mild joint pain), the decision to stop these agents may be straightforward. However, in more complicated cases, consultation with the provider who prescribed the anticoagulant/antiplatelet medication should be considered. (See "Therapeutic use of warfarin and other vitamin K antagonists", section on 'Resumption of warfarin after bleeding' and "Management of anticoagulants in patients undergoing endoscopic procedures", section on 'Urgent procedures in anticoagulated patients' and "Endoscopic procedures in patients with disorders of hemostasis" and "Management of antiplatelet agents in patients undergoing endoscopic procedures", section on 'Urgent procedures in patients on antiplatelet agents'.)

When to resume these medications once hemostasis has been achieved will also depend on the patient's risks for thrombosis and recurrent bleeding. (See "Management of anticoagulants in patients undergoing endoscopic procedures", section on 'Resumption of anticoagulants' and "Overview of the treatment of bleeding peptic ulcers", section on 'Risk factors for persistent or recurrent bleeding'.)

Consultations — Gastroenterological consultation should be obtained in all patients with suspected clinically significant acute upper GI bleeding. The decision to obtain surgical and interventional radiology consultations prior to endoscopy should be based upon the likelihood of persistent or recurrent bleeding, or risks/complications stemming from endoscopic therapy (perforation, precipitation of massive bleeding).

As a general rule, we obtain surgical and interventional radiology consultation if endoscopic therapy is unlikely to be successful, if the patient is deemed to be at high risk for rebleeding or complications associated with endoscopy, or if there is concern that the patient may have an aorto-enteric fistula. In addition, a surgeon and an interventional radiologist should be promptly notified of all patients with severe acute upper GI bleeding [44].

DIAGNOSTIC STUDIES — Algorithms providing an overview of the diagnostic approach to patients with suspected upper gastrointestinal bleeding are provided (algorithm 1 and algorithm 2).

Upper endoscopy — Upper endoscopy is the diagnostic modality of choice for acute upper GI bleeding [45,46]. Endoscopy has a high sensitivity and specificity for locating and identifying bleeding lesions in the upper GI tract. In addition, once a bleeding lesion has been identified, therapeutic endoscopy can achieve acute hemostasis and prevent recurrent bleeding in most patients. Early endoscopy (within 24 hours) is recommended for most patients with acute UGI bleeding, though whether early endoscopy affects outcomes and resource utilization is unsettled. (See 'Early endoscopy' below and "Methods to achieve hemostasis in patients with acute variceal hemorrhage", section on 'Initial management' and "Overview of the treatment of bleeding peptic ulcers", section on 'Endoscopic therapy'.)

Endoscopic findings in patients with peptic ulcers may be described using the Forrest classification [47]. Findings include spurting hemorrhage (class Ia) (picture 1), oozing hemorrhage (class Ib), a nonbleeding visible vessel (class IIa) (picture 2), an adherent clot (class IIb) (picture 3), a flat pigmented spot (class IIc), and a clean ulcer base (class III). The

endoscopic appearance helps determine which lesions require endoscopic therapy. (See "Overview of the treatment of bleeding peptic ulcers", section on 'Endoscopic therapy'.)

It may be helpful to irrigate the stomach prior to endoscopy to help remove residual blood and other gastric contents. However, despite irrigation, the stomach can be obscured with blood, potentially making it difficult to establish a clear diagnosis and/or perform therapeutic maneuvers. In patients in whom blood obscures the source of bleeding, a second endoscopy may be required to establish a diagnosis and to potentially apply therapy, but routine second-look endoscopy is not recommended. (See 'Nasogastric lavage' above and "Overview of the treatment of bleeding peptic ulcers", section on 'Second-look endoscopy'.)

Risks of endoscopy — Risks of upper endoscopy include aspiration, adverse reactions to conscious sedation, perforation, and increasing bleeding while attempting therapeutic intervention. Patients need to be hemodynamically stable prior to undergoing endoscopy.

However, while patients need to be hemodynamically stable, data suggest that most patients do not need to have a normal hematocrit in order to safely undergo endoscopy [48]. In addition, endoscopy appears to be safe in patients who are mildly to moderately anticoagulated [24]. In a retrospective study of 920 patients with upper GI bleeding undergoing upper endoscopy, patients with low hematocrits (<30 percent) were similar to those with high hematocrits (>30 percent) with regard to cardiovascular complications and mortality [48]. In another retrospective study with 233 patients with upper GI bleeding who received endoscopic therapy, an elevated INR was not associated with an increased risk of rebleeding, transfusion requirement, surgery, length of stay, or mortality [24]. The INR was between 1.3 and 2.7 in 95 percent of the patients, so the authors caution that the results of the study may only apply to patients who are mildly to moderately anticoagulated.

The risks versus benefits of upper endoscopy should be considered in high-risk patients, such as those who have had a recent myocardial infarction. In one study, for example, 200 patients who underwent endoscopy within 30 days after myocardial infarction (MI) were compared with 200 controls matched for age, sex, and endoscopic indication [49]. Complications (including fatal ventricular tachycardia, near respiratory arrest, and mild hypotension) occurred more often in patients who had a recent MI (8 versus 2 percent). Complications occurred more often (21 versus 2 percent) in patients who were very ill (Apache II score >16 or hypotension prior to endoscopy). However, such patients are at increased risk for complications even without endoscopy and may be particularly vulnerable to complications from continued bleeding without endoscopy. (See "Predictive scoring systems in the intensive care unit".)

Other diagnostic tests — Other diagnostic tests for acute upper GI bleeding include angiography and a tagged red blood cell scan, which can detect active bleeding [50,51]. Upper GI barium studies are **contraindicated** in the setting of acute upper GI bleeding because they will interfere with subsequent endoscopy, angiography, or surgery [45]. There is also interest in using wireless capsule endoscopy for patients who have presented to the emergency department with suspected upper GI bleeding. An esophageal capsule (which has a recording time of 20 minutes) can be given in the emergency department and reviewed immediately for evidence of bleeding. Confirming the presence of blood in the stomach or duodenum may aid with patient triage and identify patients more likely to benefit from early endoscopy [52-55]. (See "Angiographic control of nonvariceal gastrointestinal bleeding in adults" and "Evaluation of obscure gastrointestinal bleeding" and "Wireless video capsule endoscopy", section on 'Esophageal capsule endoscopy'.)

A colonoscopy is generally required for patients with hematochezia and a negative upper endoscopy unless an alternative source for the bleeding has been identified. In addition, patients with melena and a negative upper endoscopy frequently undergo colonoscopy to rule out a right-sided colonic source for the bleeding, as such lesions may present with melena. In a study that included 1743 colonoscopies performed for the evaluation of melena following a non-diagnostic upper endoscopy, a suspected bleeding source was identified in 5 percent of patients, a rate that was higher than that seen in 194,979 average-risk screening controls (1 percent). Despite the relatively low yield in patients with melena, we routinely perform a colonoscopy in patients with melena and a negative upper endoscopy, as well as in

patients with hematochezia. (See "Approach to acute lower gastrointestinal bleeding in adults", section on 'Colonoscopy'.)

RISK STRATIFICATION — Endoscopic, clinical, and laboratory features may be useful for risk stratification of patients who present with acute upper GI bleeding (table 2 and picture 4) [56-65], and the use of risk stratification tools is recommended by the International Consensus Upper Gastrointestinal Bleeding Conference Group [2]. Factors associated with rebleeding identified in a meta-analysis included [66]:

- Hemodynamic instability (systolic blood pressure less than 100 mmHg, heart rate greater than 100 beats per minute)
- Hemoglobin less than 10 g/L
- Active bleeding at the time of endoscopy
- Large ulcer size (greater than 1 to 3 cm in various studies)
- Ulcer location (posterior duodenal bulb or high lesser gastric curvature)

Several investigators have developed decision rules and predictive models that permit identification of patients who are at low risk for recurrent or life-threatening hemorrhage [67]. Such patients may be suitable for early hospital discharge or even outpatient care. The effectiveness of such rules has been evaluated in a variety of clinical settings, with most studies suggesting that patients deemed to be low-risk can safely be discharged early or treated as outpatients [56-62,67-75]. In addition, this approach is associated with reduced resource utilization compared with universal hospitalization of patients with acute upper GI bleeding.

Risk scores — Two commonly cited scoring systems are the Rockall score and the Blatchford score:

- The Rockall score is based upon age, the presence of shock, comorbidity, diagnosis, and endoscopic stigmata of recent hemorrhage (calculator 1) [56]. In one validation study, only 32 of 744 patients (4 percent) who scored 2 or less (out of a maximum of 11) rebled and only one died.

  On the other hand, in a later study of 247 patients who underwent endoscopic therapy for bleeding peptic ulcers, the model performed poorly when predicting recurrent bleeding, underscoring the need for validation of these models [76].

- The Blatchford score (also known as the Glasgow Blatchford score), unlike the Rockall score, does not take endoscopic data into account and thus can be used when the patient first presents (calculator 2) [61]. The score is based upon the blood urea nitrogen, hemoglobin, systolic blood pressure, pulse, and the presence of melena, syncope, hepatic disease, and/or cardiac failure. The score ranges from zero to 23 and the risk of requiring endoscopic intervention increases with increasing score. One meta-analysis found that a Blatchford score of zero was associated with a low likelihood of the need for urgent endoscopic intervention (likelihood ratio 0.02, 95% confidence interval [CI] 0-0.05) [5].

  A simpler version of the score, known as the modified Glasgow Blatchford score, is calculated using only the blood urea nitrogen, hemoglobin, systolic blood pressure, and pulse. The score ranges from 0 to 16. A prospective study of the modified score found that it performed as well as the full Blatchford score and that it outperformed the Rockall score with regard to predicting the need for clinical intervention, rebleeding, and mortality [77].

AIMS65 is another scoring system that uses data available prior to endoscopy. Studies suggest it has high accuracy for predicting inpatient mortality among patients with upper GI bleeding [65,78]. The score was derived using data from a database that contained information from 187 United States hospitals. The derivation cohort used data from 29,222

hospital admissions. The score was then validated using a separate data set containing information from 32,504 admissions. The study found that five factors were associated with increased inpatient mortality:

- **A**lbumin less than 3.0 g/dL (30 g/L)
- **I**NR greater than 1.5
- Altered **M**ental status (Glasgow coma score less than 14, disorientation, lethargy, stupor, or coma)
- **S**ystolic blood pressure of 90 mmHg or less
- Age older than **65** years

In the validation cohort, the mortality rate increased significantly as the number of risk factors present increased:

- Zero risk factors: 0.3 percent
- One risk factor: 1 percent
- Two risk factors: 3 percent
- Three risk factors: 9 percent
- Four risk factors: 15 percent
- Five risk factors: 25 percent

In addition to predicting mortality, an increasing score was also associated with increased length of stay (from 3.4 days for zero risk factors to 8.1 days for five risk factors) and increased cost (average cost of $5647 USD with zero risk factors to $15,776 USD with five risk factors). Prospective studies are needed to confirm the ability of the score to predict mortality, length of stay, and cost. In addition, it is not yet known if the score predicts rebleeding following endoscopic therapy.

Early endoscopy — Studies have reached variable conclusions when determining whether the application of early endoscopy for risk stratification and treatment reduces resource utilization or affects patient outcomes [70,79-82]. Whereas some studies have demonstrated reduced resource utilization and improved outcomes from early endoscopy [81,82], other studies, including a randomized trial, did not [70,79]:

- In the randomized trial, 93 outpatients with acute upper GI bleeding were assigned to urgent endoscopy (before hospitalization) or elective endoscopy after admission [70]. Results of the urgent endoscopy and a recommendation regarding patient disposition were provided to the attending clinician who made the final decision regarding patient disposition.

  The timing of endoscopy did not affect resource utilization or patient outcomes. Length of stay was similar (4 versus 5 days in the urgent and delayed groups, respectively), as was the mean number of days in the intensive care unit (1.2). Outpatient care was recommended for 19 patients (40 percent) in the urgent endoscopy group. However, the attending clinicians who were responsible for making the discharge decisions only followed the recommendation for outpatient care in four patients.

  This trial suggests that in order for early endoscopy to reduce resource utilization, stratification needs to translate into changes in patient management. Studies showing reduced utilization have incorporated processes by which patient disposition was linked directly to the risk stratification system.

- A benefit for early endoscopy (defined as endoscopy within one day of admission) was suggested by a large retrospective study using a database of hospital inpatient admissions (Nationwide Inpatient Sample) [82]. The study looked at 35,747 adults with acute variceal bleeding and 435,765 adults with nonvariceal upper GI bleeding. Among patients with acute variceal hemorrhage, inpatient mortality was 8.3 percent for those who underwent upper endoscopy within one day of admission and was 15.3 percent for those who did not (adjusted odds ratio

[OR] 1.18; 95% CI 1.08-1.31). For patients with nonvariceal upper GI bleeding, the corresponding mortality rates were 2.5 and 6.6 percent, respectively (adjusted OR 1.32; 95% CI 1.26-1.38).

However, a limitation of the study is that it did not differentiate patients who were admitted with upper GI bleeding from those who developed upper GI bleeding while hospitalized for other reasons (most of whom would presumably undergo endoscopy more than one day following hospital admission). This could skew the results toward increased mortality in the patients who did not undergo early endoscopy since patients who develop bleeding as inpatients are known to have higher mortality rates [83,84].

- Another study that suggested a benefit with regard to mortality included 8222 patients with upper GI bleeding [81]. Patients who died had a significantly longer waiting time to endoscopy than those who survived (1.65 versus 0.95 days; adjusted OR 1.10, 95% CI 1.06-1.14).

Implementation — The data presented above suggest that risk stratification is feasible and permits identification of patients who can be managed safely without hospitalization. However, for these systems to be successful, the risk stratification system must be tied directly to decisions regarding patient discharge. None of the published risk scores has yet been adopted widely.

As a general rule, we discharge patients who meet the following criteria:

- Have no comorbidities
- Have stable vital signs
- Have a normal hemoglobin
- Have a likely bleeding source identified on upper endoscopy
- Have a source of bleeding that is not associated with a high risk of rebleeding (eg, variceal bleeding, active bleeding, bleeding from a Dieulafoy's lesion, or ulcer bleeding with high-risk stigmata) (table 2)

However, the decision to discharge a patient also depends upon individual-patient factors, such as reliability for follow-up and confidence in the diagnosis; in some cases, we admit patients who appear to be low-risk for observation.

If patients do not meet these criteria we admit them to a monitored setting or intensive care unit (depending upon the severity of bleeding, comorbidities, and stability of vital signs). Most patients who have received endoscopic treatment for high-risk stigmata should be hospitalized for 72 hours to monitor for rebleeding, since most rebleeding occurs during this time [2].

TREATMENT — The treatment of patients with upper GI bleeding due to various causes is discussed separately. (See "Overview of the treatment of bleeding peptic ulcers" and "Contact thermal devices for the treatment of bleeding peptic ulcers" and "Methods to achieve hemostasis in patients with acute variceal hemorrhage".)

INFORMATION FOR PATIENTS — UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient information: Upper endoscopy (The Basics)" and "Patient information: GI bleed (The Basics)")
- Beyond the Basics topics (see "Patient information: Upper endoscopy (Beyond the Basics)" and "Patient information: Peptic ulcer disease (Beyond the Basics)")

SUMMARY AND RECOMMENDATIONS

- A table outlining the emergency management of acute severe upper gastrointestinal bleeding is provided (table 1). (See 'Introduction' above.)
- A careful history should be obtained to identify potential sources of the upper GI bleed, assess the severity of the bleed, and to identify comorbid conditions that may influence the patient's subsequent management. (See 'Initial evaluation' above.)
- The physical examination should focus on signs that indicate the severity of blood loss, help localize the source of the bleeding, and suggest complications. (See 'Physical examination' above.)
- The presence of abdominal pain, especially if severe and associated with rebound tenderness or involuntary guarding raises concern for perforation. If any signs of an acute abdomen are present, further evaluation to exclude a perforation is required prior to endoscopy. (See 'Physical examination' above.)
- Laboratory tests obtained in patients with acute upper gastrointestinal bleeding include a complete blood count, serum chemistries, liver tests, and coagulation studies. In addition, we suggest ruling out a myocardial infarction in elderly patients and those with known cardiovascular disease who have severe bleeding, especially if there has been hemodynamic instability. (See 'Laboratory data' above.)
- We suggest that patients only undergo NGT lavage if particulate matter, fresh blood, and clots need to be removed from the stomach to facilitate endoscopy. (See 'Nasogastric lavage' above.)
- Patients who require hospitalization should be admitted to a monitored bed or intensive care unit depending upon the severity of bleeding. (See 'Triage' above.)
- We suggest incorporation of a validated risk score for upper gastrointestinal bleeding into routine clinical practice to facilitate optimal triage decisions. (See 'Risk scores' above.)
- General supportive measures include:
    - Provision of supplemental oxygen by nasal cannula
    - Nothing per mouth
    - Two large caliber (16 gauge or larger) peripheral catheters or a central venous line
    - Placement of a pulmonary artery catheter should be considered in patients with hemodynamic instability or who need close monitoring during resuscitation
- For the majority of patients with acute upper gastrointestinal bleeding who do not have significant comorbid illnesses, we recommend giving blood transfusions to maintain the hemoglobin at ≥7 g/dL (70 g/L) rather than ≥9 g/dL (90 g/L) (**Grade 1B**). However, patients with active bleeding and hypovolemia may require blood transfusion despite an apparently normal hemoglobin. For patients at increased risk of suffering adverse events in the setting of significant anemia, such as those with unstable coronary artery disease, we suggest transfusing to maintain the hemoglobin at ≥9 g/dL (90 g/L) rather than ≥7 g/dL (70 g/L) (**Grade 2C**). (See 'Blood transfusions' above and "Overview of the non-acute management of unstable angina and non-ST elevation myocardial infarction", section

on 'Red cell transfusion'.).

In patients with suspected variceal bleeding, we suggest transfusing to a hemoglobin of no more than 10 g/dL (100 g/L) (**Grade 2C**). It is particularly important to avoid overtransfusion in patients with suspected variceal bleeding, as it can precipitate worsening of the bleeding. (See 'Blood transfusions' above.)

- We suggest that patients with acute upper GI bleeding be treated with an intravenous PPI at presentation until confirmation of the cause of bleeding, after which the need for specific therapy and the duration of PPI use can be determined (**Grade 2B**). (See 'Acid suppression' above and "Overview of the treatment of bleeding peptic ulcers", section on 'Acid suppression'.)
- We suggest that erythromycin be given prior to endoscopy in patients who are likely to have a large amount of blood in their stomach, such as those with severe bleeding. A reasonable dose is 3 mg/kg intravenously over 20 to 30 minutes, 30 to 90 minutes prior to endoscopy. (See 'Prokinetics' above.)
- We recommend that patients known to have cirrhosis who present with acute upper GI bleeding receive antibiotics, preferably before endoscopy (**Grade 1A**). (See "General principles of the management of variceal hemorrhage", section on 'Infection and use of prophylactic antibiotics'.)
- We recommend upper endoscopy for the evaluation and management of clinically significant (ie, more than a scant amount of blood) acute upper GI bleeding (**Grade 1A**). Additional diagnostic tests may be required in specific circumstances. Algorithms providing an overview of the diagnostic approach to patients with suspected upper gastrointestinal bleeding are provided (algorithm 1 and algorithm 2). (See 'Diagnostic studies' above.)
- The treatment of patients with upper GI bleeding due to various causes is discussed separately. (See "Overview of the treatment of bleeding peptic ulcers" and "Contact thermal devices for the treatment of bleeding peptic ulcers" and "Methods to achieve hemostasis in patients with acute variceal hemorrhage".)

REFERENCES

1. Barkun A, Bardou M, Marshall JK, Nonvariceal Upper GI Bleeding Consensus Conference Group. Consensus recommendations for managing patients with nonvariceal upper gastrointestinal bleeding. Ann Intern Med 2003; 139:843.
2. Barkun AN, Bardou M, Kuipers EJ, et al. International consensus recommendations on the management of patients with nonvariceal upper gastrointestinal bleeding. Ann Intern Med 2010; 152:101.
3. Hwang JH, Fisher DA, Ben-Menachem T, et al. The role of endoscopy in the management of acute non-variceal upper GI bleeding. Gastrointest Endosc 2012; 75:1132.
4. Laine L, Jensen DM. Management of patients with ulcer bleeding. Am J Gastroenterol 2012; 107:345.
5. Srygley FD, Gerardo CJ, Tran T, Fisher DA. Does this patient have a severe upper gastrointestinal bleed? JAMA 2012; 307:1072.
6. Cappell MS, Friedel D. Initial management of acute upper gastrointestinal bleeding: from initial evaluation up to gastrointestinal endoscopy. Med Clin North Am 2008; 92:491.
7. Current diagnosis and treatment: Surgery, 13, Doherty G (Ed), McGraw-Hill Companies, 2010. p.493.
8. Jensen DM, Machicado GA. Diagnosis and treatment of severe hematochezia. The role of urgent colonoscopy after purge. Gastroenterology 1988; 95:1569.
9. Palmer ED. The vigorous diagnostic approach to upper-gastrointestinal tract hemorrhage. A 23-year prospective study of 1,4000 patients. JAMA 1969; 207:1477.

10. Richards RJ, Donica MB, Grayer D. Can the blood urea nitrogen/creatinine ratio distinguish upper from lower gastrointestinal bleeding? J Clin Gastroenterol 1990; 12:500.
11. Mortensen PB, Nøhr M, Møller-Petersen JF, Balslev I. The diagnostic value of serum urea/creatinine ratio in distinguishing between upper and lower gastrointestinal bleeding. A prospective study. Dan Med Bull 1994; 41:237.
12. Pallin DJ, Saltzman JR. Is nasogastric tube lavage in patients with acute upper GI bleeding indicated or antiquated? Gastrointest Endosc 2011; 74:981.
13. Huang ES, Karsan S, Kanwal F, et al. Impact of nasogastric lavage on outcomes in acute GI bleeding. Gastrointest Endosc 2011; 74:971.
14. Aljebreen AM, Fallone CA, Barkun AN. Nasogastric aspirate predicts high-risk endoscopic lesions in patients with acute upper-GI bleeding. Gastrointest Endosc 2004; 59:172.
15. Baradarian R, Ramdhaney S, Chapalamadugu R, et al. Early intensive resuscitation of patients with upper gastrointestinal bleeding decreases mortality. Am J Gastroenterol 2004; 99:619.
16. Duggan JM. Gastrointestinal hemorrhage: should we transfuse less? Dig Dis Sci 2009; 54:1662.
17. Villanueva C, Colomo A, Bosch A, et al. Transfusion strategies for acute upper gastrointestinal bleeding. N Engl J Med 2013; 368:11.
18. Qaseem A, Humphrey LL, Fitterman N, et al. Treatment of anemia in patients with heart disease: a clinical practice guideline from the American College of Physicians. Ann Intern Med 2013; 159:770.
19. Kravetz D, Bosch J, Arderiu M, et al. Hemodynamic effects of blood volume restitution following a hemorrhage in rats with portal hypertension due to cirrhosis of the liver: influence of the extent of portal-systemic shunting. Hepatology 1989; 9:808.
20. Cerqueira RM, Andrade L, Correia MR, et al. Risk factors for in-hospital mortality in cirrhotic patients with oesophageal variceal bleeding. Eur J Gastroenterol Hepatol 2012; 24:551.
21. Krige JE, Kotze UK, Distiller G, et al. Predictive factors for rebleeding and death in alcoholic cirrhotic patients with acute variceal bleeding: a multivariate analysis. World J Surg 2009; 33:2127.
22. McCormick PA, Jenkins SA, McIntyre N, Burroughs AK. Why portal hypertensive varices bleed and bleed: a hypothesis. Gut 1995; 36:100.
23. Restellini S, Kherad O, Jairath V, et al. Red blood cell transfusion is associated with increased rebleeding in patients with nonvariceal upper gastrointestinal bleeding. Aliment Pharmacol Ther 2013; 37:316.
24. Wolf AT, Wasan SK, Saltzman JR. Impact of anticoagulation on rebleeding following endoscopic therapy for nonvariceal upper gastrointestinal hemorrhage. Am J Gastroenterol 2007; 102:290.
25. Maltz GS, Siegel JE, Carson JL. Hematologic management of gastrointestinal bleeding. Gastroenterol Clin North Am 2000; 29:169.
26. ASGE Standards of Practice Committee, Anderson MA, Ben-Menachem T, et al. Management of antithrombotic agents for endoscopic procedures. Gastrointest Endosc 2009; 70:1060.
27. Dorward S, Sreedharan A, Leontiadis GI, et al. Proton pump inhibitor treatment initiated prior to endoscopic diagnosis in upper gastrointestinal bleeding. Cochrane Database Syst Rev 2006; :CD005415.
28. Gisbert JP, González L, Calvet X, et al. Proton pump inhibitors versus H2-antagonists: a meta-analysis of their efficacy in treating bleeding peptic ulcer. Aliment Pharmacol Ther 2001; 15:917.
29. Kaviani MJ, Hashemi MR, Kazemifar AR, et al. Effect of oral omeprazole in reducing re-bleeding in bleeding peptic ulcers: a prospective, double-blind, randomized, clinical trial. Aliment Pharmacol Ther 2003; 17:211.
30. Lau JY, Sung JJ, Lee KK, et al. Effect of intravenous omeprazole on recurrent bleeding after endoscopic treatment of bleeding peptic ulcers. N Engl J Med 2000; 343:310.
31. Chan WH, Khin LW, Chung YF, et al. Randomized controlled trial of standard versus high-dose intravenous omeprazole after endoscopic therapy in high-risk patients with acute peptic ulcer bleeding. Br J Surg 2011; 98:640.
32. Green FW Jr, Kaplan MM, Curtis LE, Levine PH. Effect of acid and pepsin on blood coagulation and platelet aggregation. A possible contributor prolonged gastroduodenal mucosal hemorrhage. Gastroenterology 1978; 74:38.

33. Lau JY, Leung WK, Wu JC, et al. Omeprazole before endoscopy in patients with gastrointestinal bleeding. N Engl J Med 2007; 356:1631.
34. Al-Sabah S, Barkun AN, Herba K, et al. Cost-effectiveness of proton-pump inhibition before endoscopy in upper gastrointestinal bleeding. Clin Gastroenterol Hepatol 2008; 6:418.
35. Frossard JL, Spahr L, Queneau PE, et al. Erythromycin intravenous bolus infusion in acute upper gastrointestinal bleeding: a randomized, controlled, double-blind trial. Gastroenterology 2002; 123:17.
36. Coffin B, Pocard M, Panis Y, et al. Erythromycin improves the quality of EGD in patients with acute upper GI bleeding: a randomized controlled study. Gastrointest Endosc 2002; 56:174.
37. Altraif I, Handoo FA, Aljumah A, et al. Effect of erythromycin before endoscopy in patients presenting with variceal bleeding: a prospective, randomized, double-blind, placebo-controlled trial. Gastrointest Endosc 2011; 73:245.
38. Carbonell N, Pauwels A, Serfaty L, et al. Erythromycin infusion prior to endoscopy for acute upper gastrointestinal bleeding: a randomized, controlled, double-blind trial. Am J Gastroenterol 2006; 101:1211.
39. Barkun AN, Bardou M, Martel M, et al. Prokinetics in acute upper GI bleeding: a meta-analysis. Gastrointest Endosc 2010; 72:1138.
40. Bai Y, Guo JF, Li ZS. Meta-analysis: erythromycin before endoscopy for acute upper gastrointestinal bleeding. Aliment Pharmacol Ther 2011; 34:166.
41. Pateron D, Vicaut E, Debuc E, et al. Erythromycin infusion or gastric lavage for upper gastrointestinal bleeding: a multicenter randomized controlled trial. Ann Emerg Med 2011; 57:582.
42. Imperiale TF, Birgisson S. Somatostatin or octreotide compared with H2 antagonists and placebo in the management of acute nonvariceal upper gastrointestinal hemorrhage: a meta-analysis. Ann Intern Med 1997; 127:1062.
43. Gluud LL, Klingenberg SL, Langholz E. Tranexamic acid for upper gastrointestinal bleeding. Cochrane Database Syst Rev 2012; 1:CD006640.
44. Kolkman JJ, Meuwissen SG. A review on treatment of bleeding peptic ulcer: a collaborative task of gastroenterologist and surgeon. Scand J Gastroenterol Suppl 1996; 218:16.
45. Jutabha R, Jensen DM. Management of upper gastrointestinal bleeding in the patient with chronic liver disease. Med Clin North Am 1996; 80:1035.
46. Adang RP, Vismans JF, Talmon JL, et al. Appropriateness of indications for diagnostic upper gastrointestinal endoscopy: association with relevant endoscopic disease. Gastrointest Endosc 1995; 42:390.
47. Forrest JA, Finlayson ND, Shearman DJ. Endoscopy in gastrointestinal bleeding. Lancet 1974; 2:394.
48. Balderas V, Bhore R, Lara LF, et al. The hematocrit level in upper gastrointestinal hemorrhage: safety of endoscopy and outcomes. Am J Med 2011; 124:970.
49. Cappell MS, Iacovone FM Jr. Safety and efficacy of esophagogastroduodenoscopy after myocardial infarction. Am J Med 1999; 106:29.
50. Barth KH. Radiological intervention in upper and lower gastrointestinal bleeding. Baillieres Clin Gastroenterol 1995; 9:53.
51. Emslie JT, Zarnegar K, Siegel ME, Beart RW Jr. Technetium-99m-labeled red blood cell scans in the investigation of gastrointestinal bleeding. Dis Colon Rectum 1996; 39:750.
52. Gralnek IM, Ching JY, Maza I, et al. Capsule endoscopy in acute upper gastrointestinal hemorrhage: a prospective cohort study. Endoscopy 2013; 45:12.
53. Meltzer AC, Ali MA, Kresiberg RB, et al. Video capsule endoscopy in the emergency department: a prospective study of acute upper gastrointestinal hemorrhage. Ann Emerg Med 2013; 61:438.
54. Chandran S, Testro A, Urquhart P, et al. Risk stratification of upper GI bleeding with an esophageal capsule. Gastrointest Endosc 2013; 77:891.
55. Meltzer AC, Pinchbeck C, Burnett S, et al. Emergency physicians accurately interpret video capsule endoscopy findings in suspected upper gastrointestinal hemorrhage: a video survey. Acad Emerg Med 2013; 20:711.
56. Rockall TA, Logan RF, Devlin HB, Northfield TC. Selection of patients for early discharge or outpatient care after

acute upper gastrointestinal haemorrhage. National Audit of Acute Upper Gastrointestinal Haemorrhage. Lancet 1996; 347:1138.

57. Corley DA, Stefan AM, Wolf M, et al. Early indicators of prognosis in upper gastrointestinal hemorrhage. Am J Gastroenterol 1998; 93:336.
58. Stanley AJ, Robinson I, Forrest EH, et al. Haemodynamic parameters predicting variceal haemorrhage and survival in alcoholic cirrhosis. QJM 1998; 91:19.
59. Hay JA, Maldonado L, Weingarten SR, Ellrodt AG. Prospective evaluation of a clinical guideline recommending hospital length of stay in upper gastrointestinal tract hemorrhage. JAMA 1997; 278:2151.
60. Hay JA, Lyubashevsky E, Elashoff J, et al. Upper gastrointestinal hemorrhage clinical--guideline determining the optimal hospital length of stay. Am J Med 1996; 100:313.
61. Blatchford O, Murray WR, Blatchford M. A risk score to predict need for treatment for upper-gastrointestinal haemorrhage. Lancet 2000; 356:1318.
62. Cipolletta L, Bianco MA, Rotondano G, et al. Outpatient management for low-risk nonvariceal upper GI bleeding: a randomized controlled trial. Gastrointest Endosc 2002; 55:1.
63. Marmo R, Koch M, Cipolletta L, et al. Predicting mortality in non-variceal upper gastrointestinal bleeders: validation of the Italian PNED Score and Prospective Comparison with the Rockall Score. Am J Gastroenterol 2010; 105:1284.
64. Pang SH, Ching JY, Lau JY, et al. Comparing the Blatchford and pre-endoscopic Rockall score in predicting the need for endoscopic therapy in patients with upper GI hemorrhage. Gastrointest Endosc 2010; 71:1134.
65. Saltzman JR, Tabak YP, Hyett BH, et al. A simple risk score accurately predicts in-hospital mortality, length of stay, and cost in acute upper GI bleeding. Gastrointest Endosc 2011; 74:1215.
66. García-Iglesias P, Villoria A, Suarez D, et al. Meta-analysis: predictors of rebleeding after endoscopic treatment for bleeding peptic ulcer. Aliment Pharmacol Ther 2011; 34:888.
67. Das A, Wong RC. Prediction of outcome of acute GI hemorrhage: a review of risk scores and predictive models. Gastrointest Endosc 2004; 60:85.
68. Brullet E, Campo R, Calvet X, et al. A randomized study of the safety of outpatient care for patients with bleeding peptic ulcer treated by endoscopic injection. Gastrointest Endosc 2004; 60:15.
69. Gralnek IM, Dulai GS. Incremental value of upper endoscopy for triage of patients with acute non-variceal upper-GI hemorrhage. Gastrointest Endosc 2004; 60:9.
70. Bjorkman DJ, Zaman A, Fennerty MB, et al. Urgent vs. elective endoscopy for acute non-variceal upper-GI bleeding: an effectiveness study. Gastrointest Endosc 2004; 60:1.
71. Longstreth GF, Feitelberg SP. Successful outpatient management of acute upper gastrointestinal hemorrhage: use of practice guidelines in a large patient series. Gastrointest Endosc 1998; 47:219.
72. Lee JG, Turnipseed S, Romano PS, et al. Endoscopy-based triage significantly reduces hospitalization rates and costs of treating upper GI bleeding: a randomized controlled trial. Gastrointest Endosc 1999; 50:755.
73. Imperiale TF, Dominitz JA, Provenzale DT, et al. Predicting poor outcome from acute upper gastrointestinal hemorrhage. Arch Intern Med 2007; 167:1291.
74. Das A, Ben-Menachem T, Farooq FT, et al. Artificial neural network as a predictive instrument in patients with acute nonvariceal upper gastrointestinal hemorrhage. Gastroenterology 2008; 134:65.
75. Stanley AJ, Ashley D, Dalton HR, et al. Outpatient management of patients with low-risk upper-gastrointestinal haemorrhage: multicentre validation and prospective evaluation. Lancet 2009; 373:42.
76. Church NI, Dallal HJ, Masson J, et al. Validity of the Rockall scoring system after endoscopic therapy for bleeding peptic ulcer: a prospective cohort study. Gastrointest Endosc 2006; 63:606.
77. Cheng DW, Lu YW, Teller T, et al. A modified Glasgow Blatchford Score improves risk stratification in upper gastrointestinal bleed: a prospective comparison of scoring systems. Aliment Pharmacol Ther 2012; 36:782.
78. Hyett BH, Abougergi MS, Charpentier JP, et al. The AIMS65 score compared with the Glasgow-Blatchford score in predicting outcomes in upper GI bleeding. Gastrointest Endosc 2013; 77:551.

79. Sarin N, Monga N, Adams PC. Time to endoscopy and outcomes in upper gastrointestinal bleeding. Can J Gastroenterol 2009; 23:489.
80. Tsoi KK, Ma TK, Sung JJ. Endoscopy for upper gastrointestinal bleeding: how urgent is it? Nat Rev Gastroenterol Hepatol 2009; 6:463.
81. Tsoi KK, Chiu PW, Chan FK, et al. The risk of peptic ulcer bleeding mortality in relation to hospital admission on holidays: a cohort study on 8,222 cases of peptic ulcer bleeding. Am J Gastroenterol 2012; 107:405.
82. Wysocki JD, Srivastav S, Winstead NS. A nationwide analysis of risk factors for mortality and time to endoscopy in upper gastrointestinal haemorrhage. Aliment Pharmacol Ther 2012; 36:30.
83. Hearnshaw SA, Logan RF, Lowe D, et al. Acute upper gastrointestinal bleeding in the UK: patient characteristics, diagnoses and outcomes in the 2007 UK audit. Gut 2011; 60:1327.
84. Marmo R, Koch M, Cipolletta L, et al. Predicting mortality in patients with in-hospital nonvariceal upper GI bleeding: a prospective, multicenter database study. Gastrointest Endosc 2014; 79:741.

Topic 2548 Version 40.0

GRAPHICS

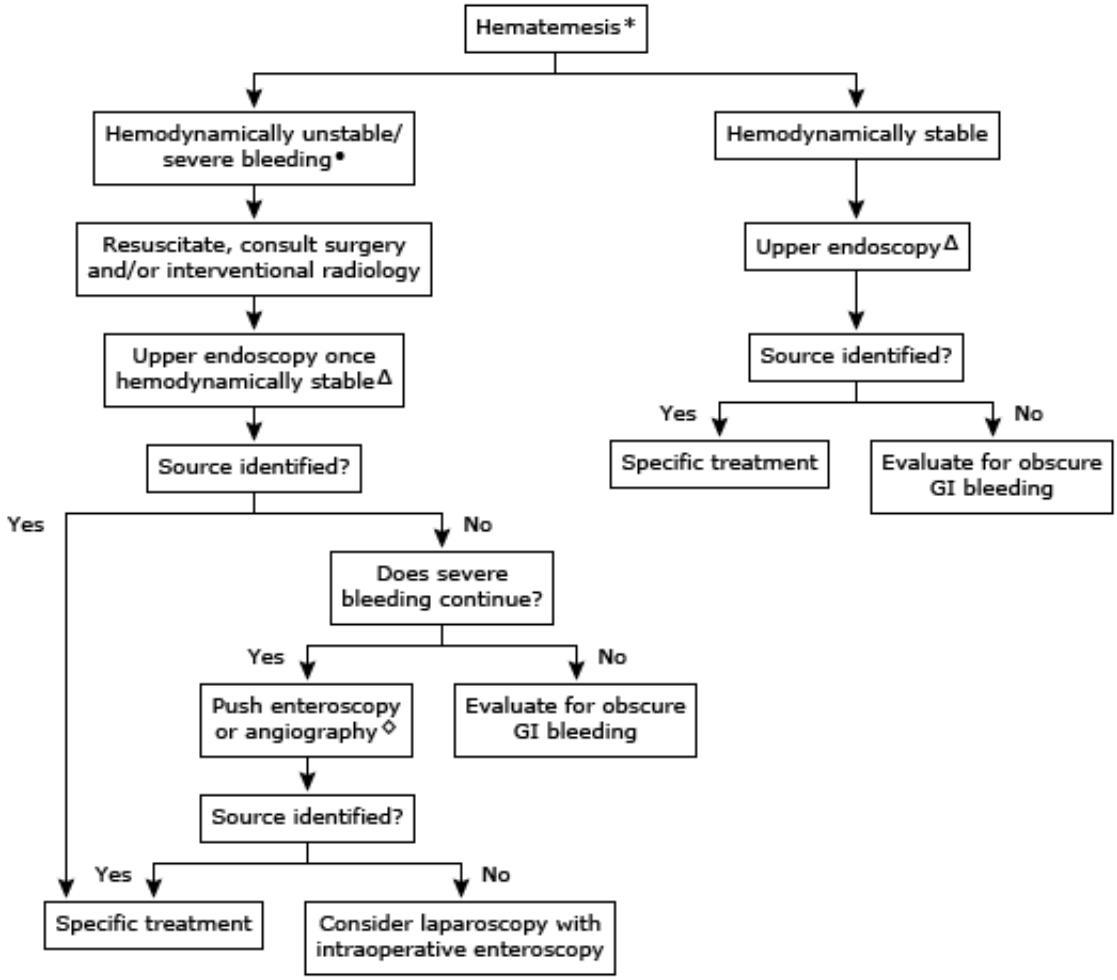
Rapid overview: Emergency management of severe upper GI bleeding

Major causes
Peptic ulcer, esophagogastric varices, arteriovenous malformation, tumor, esophageal (Mallory-Weiss) tear
Clinical features
History
Use of: NSAIDs, aspirin, anticoagulants, antiplatelet agents
Alcohol abuse; previous GI bleed; liver disease; coagulopathy
Symptoms and signs: Abdominal pain; hematemesis or "coffee ground" emesis; passing melena/tarry stool (stool may be frankly bloody or maroon with massive or brisk upper GI bleeding)
Examination
Tachycardia, orthostatic blood pressure changes suggest moderate to severe blood loss; hypotension suggests life-threatening blood loss (hypotension may be late finding in healthy younger adult)
Rectal examination is performed to assess stool color (melena vs hematochezia vs brown) and perform guaiac testing
Significant abdominal tenderness accompanied by signs of peritoneal irritation (eg, involuntary guarding) suggests perforation
Diagnostic testing
Obtain type and screen (or type and crossmatch for hemodynamic instability, severe bleeding, or high-risk patient)
Obtain hemoglobin concentration (normal measurement may be inaccurate with acute severe hemorrhage), platelet count, coagulation studies (prothrombin time with INR), liver enzymes (AST, ALT), albumin, BUN and creatinine
Nasogastric lavage may be helpful if the source of bleeding is unclear (upper or lower GI tract) or to clean the stomach prior to endoscopy
Treatment
Closely monitor airway, clinical status, vital signs, cardiac rhythm, urine output, nasogastric output (if nasogastric tube in place)
Do NOT give patient anything by mouth
Establish two large bore IV lines (16 gauge or larger)
Provide supplemental oxygen
Treat hypotension initially with rapid, bolus infusions of isotonic crystalloid
Transfuse for:
Hemodynamic instability despite crystalloid resuscitation
Hemoglobin <9 g/dL (90 g/L) in high-risk patients (eg, elderly, coronary artery disease)

Hemoglobin <7 g/dL (70 g/L) in low-risk patients
Avoid over-transfusion with possible variceal bleeding
Give fresh frozen plasma for coagulopathy; give platelets for thrombocytopenia (platelets <50,000) or platelet dysfunction (eg, chronic aspirin therapy)
Obtain immediate consultation with gastroenterologist; obtain surgical and interventional radiology consultation for any large-scale bleeding
Pharmacotherapy for all patients with suspected or known severe bleeding:
Give a proton pump inhibitor (eg, Esomeprazole 80 mg IV bolus, followed by 8 mg/hour OR Pantoprazole 80 mg IV bolus, followed by 8 mg/hour infusion)
Pharmacotherapy for known or suspected esophagogastric variceal bleeding and/or cirrhosis:
Give somatostatin or an analogue (eg, Octreotide 50 mcg bolus, followed by 50 mcg/hour infusion)
Give an antibiotic (eg, Ceftriaxone, Amoxicillin-clavulanate, or Quinolone)
Balloon tamponade may be performed as a temporizing measure for patients with uncontrollable hemorrhage likely due to varices using any of several devices (eg, Sengstaken-Blakemore tube, Minnesota tube); tracheal intubation is necessary if such a device is to be placed; ensure proper device placement prior to inflation to avoid esophageal rupture

Graphic 72195 Version 5.0

Evaluation of patients presenting with hematemesis



GI: gastrointestinal; CT: computed tomography.

* With or without melena or hematochezia.

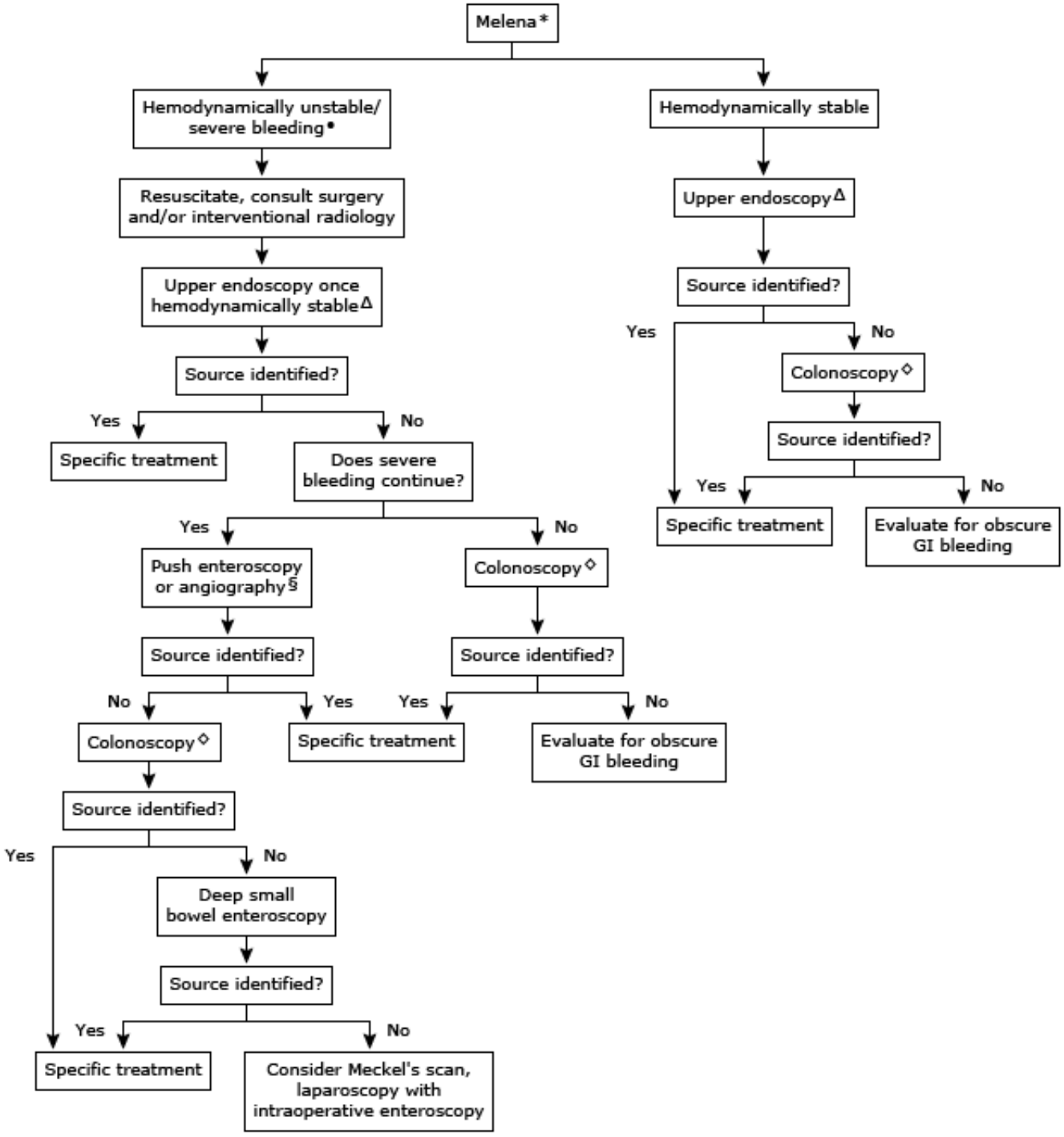
• Bleeding associated with signs such as hypotension, tachycardia, or orthostatic hypotension.

Δ Consider evaluation with a side-viewing duodenoscope if there are risk factors for hemobilia or hemosuccus pancreaticus, or CT angiography (followed by push enteroscopy if the CT angiography is negative) in patients at risk for an aortoenteric fistula.

◇ Push enteroscopy if the patient is hemodyanmically stable, conventional transvenous angiography if the patient is hemodynamically unstable.

Graphic 95233 Version 1.0

Evaluation of patients presenting with melena



GI: gastrointestinal; CT: computed tomography.

* If hematemesis is also present, use the evaluation of hematemesis algorithm.

• Bleeding associated with signs such as hypotension, tachycardia, or orthostatic hypotension.

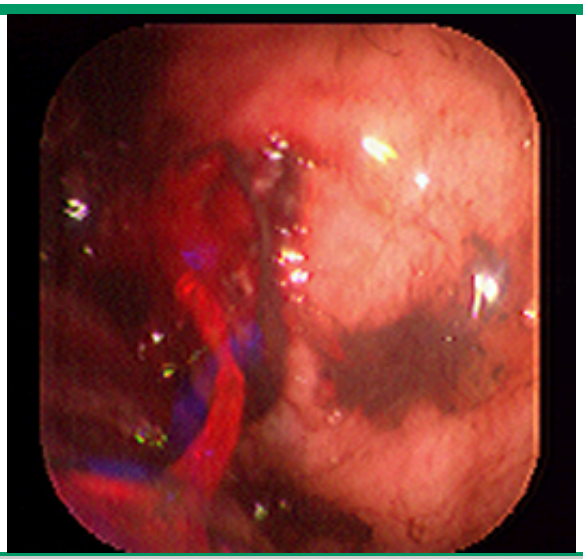
Δ Consider evaluation with a side-viewing duodenoscope if there are risk factors for hemobilia or hemosuccus pancreaticus, or CT angiography (followed by push enteroscopy if the CT angiography is negative) in patients at risk for an aortoenteric fistula.

◇ If the initial colonoscopy was inadequate (eg, fair or poor preparation, failure to reach the cecum), repeat colonoscopy should be considered.

§ Push enteroscopy if the patient is hemodyanmically stable, conventional transvenous angiography if the patient is hemodynamically unstable.

Graphic 95231 Version 1.0

Bleeding gastric ulcer

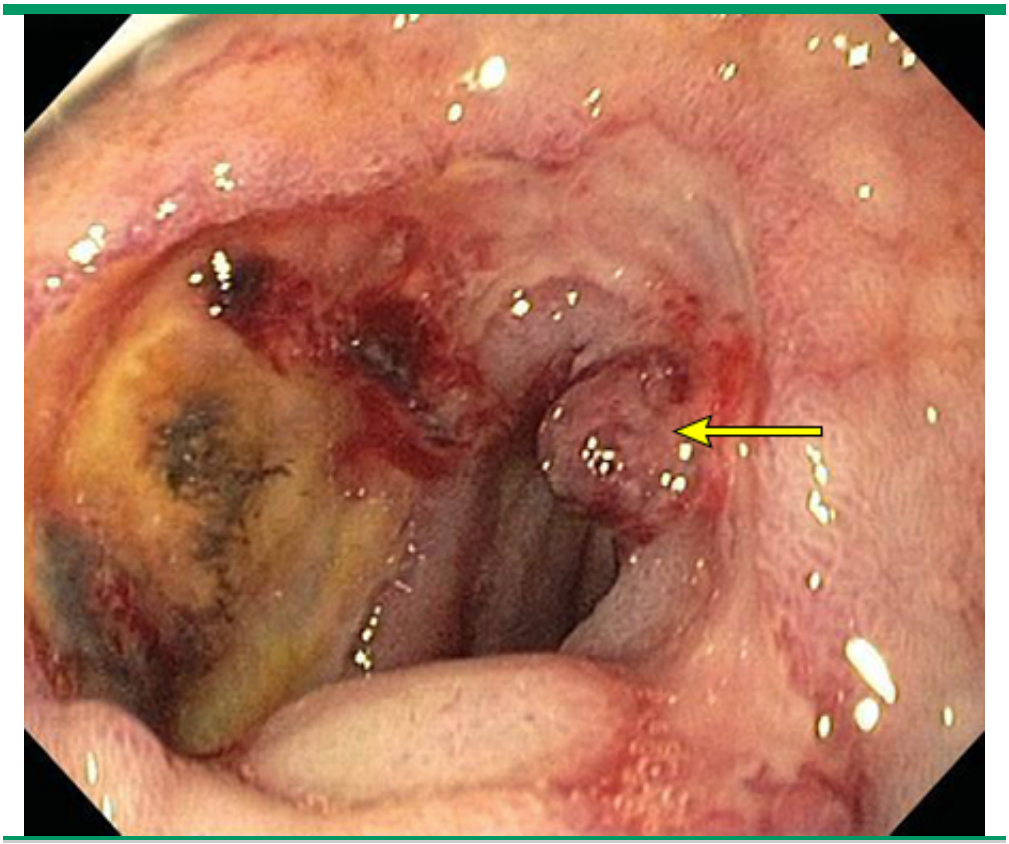


Endoscopy shows an actively bleeding gastric ulcer in the lesser curvature.

Courtesy of Rome Jutabha, MD and Dennis M Jensen, MD.

Graphic 61646 Version 1.0

Duodenal ulcer with visible vessel

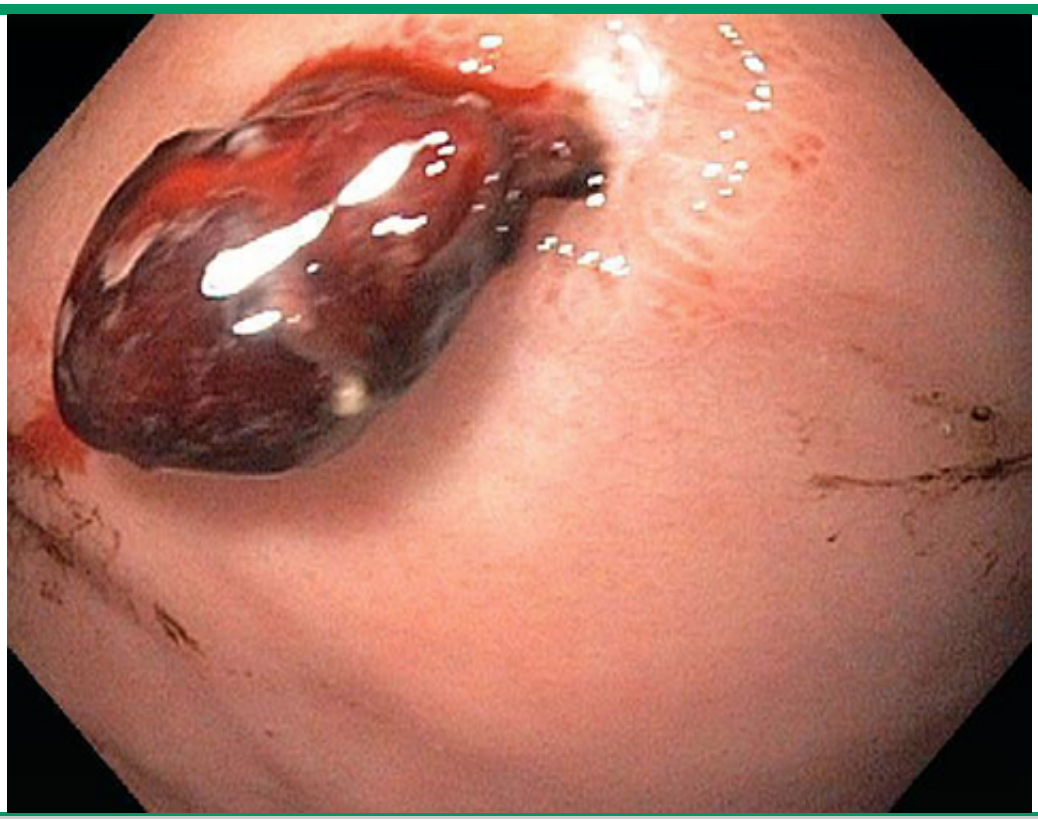


Upper endoscopy showing a duodenal ulcer with a nonbleeding visible vessel in a large circumferential ulcer (Forrest classification IIa).

Courtesy of Rome Jutabha.

Graphic 54960 Version 3.0

Gastric ulcer with adherent clot



Upper endoscopy showing a gastric ulcer with an adherent clot (Forrest classification IIb).

Courtesy of Rome Jutabha, MD.

Graphic 76246 Version 1.0

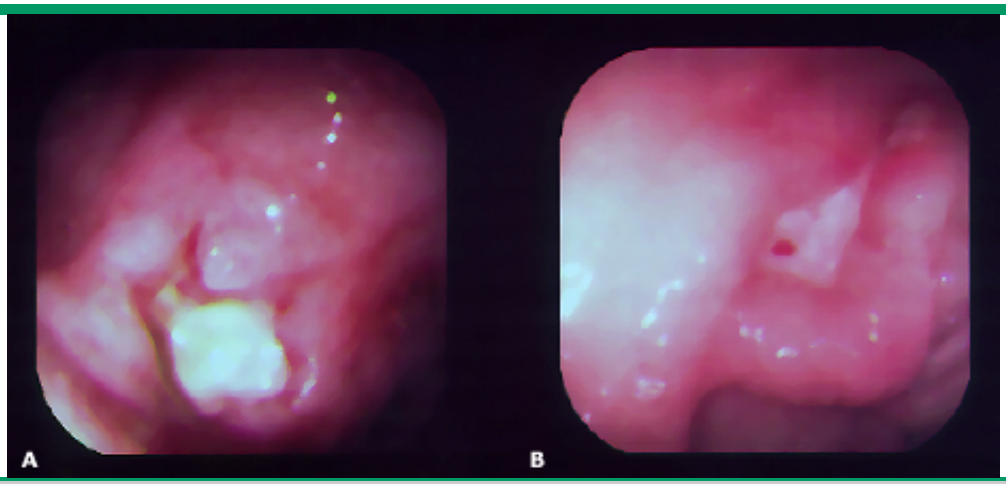
Endoscopic predictors of recurrent peptic ulcer hemorrhage

Endoscopic stigmata of recent hemorrhage	Prevalence, percent	Risk of rebleeding on medical management, percent
Active arterial bleeding (Forrest Ia)	10	90
Oozing without visible vessel (Forrest Ib)	10	10 to 20
Non-bleeding visible vessel (Forrest IIa)	25	50
Adherent clot (Forrest IIb)	10	25 to 30
Flat spot (Forrest IIc)	10	7 to 10
Clean ulcer base (Forrest (III)	35	3 to 5

Adapted from: Katschinski B, Logan R, Davies J, et al. Dig Dis Sci 1994; 39:706.

Graphic 78607 Version 5.0

Peptic ulcers at low risk for rebleeding



Ulcers with a clean base (panel A) or those with a flat pigmented red spot (panel B) are at low risk for rebleeding and do not need to be treated endoscopically.

Courtesy of Rome Jutabha, MD and Dennis M Jensen, MD.

Graphic 52497 Version 1.0

Disclosures

Disclosures: **John R Saltzman, MD, FACP, FACG, FASGE, AGAF** Nothing to disclose. **Mark Feldman, MD, MACP, AGAF, FACG** Nothing to disclose. **Anne C Travis, MD, MSc, FACG, AGAF** Employee of UpToDate, Inc. Equity Ownership/Stock Options: Proctor & Gamble. Contributor disclosures are reviewed for conflicts of interest by the editorial group. When found, these are addressed by vetting through a multi-level review process, and through requirements for references to be provided to support the content. Appropriately referenced content is required of all authors and must conform to UpToDate standards of evidence.

Conflict of interest policy