Gastrointestinal Bleeding

(See also *Harrison's Principles of Internal Medicine*, 17th Edition, Chapter 42)

Definition

- Bleeding from the gastrointestinal (GI) tract may present in 5 ways.
  - Hematemesis: vomitus of red blood or "coffee grounds" material
  - Melena: black, tarry, foul-smelling stool
  - Hematochezia: passage of bright red or maroon blood from the rectum
  - Occult GI bleeding: blood in the stool in the absence of overt bleeding
  - Symptoms of blood loss or anemia (e.g., lightheadedness or shortness of breath)
- Upper GI bleeding
  - Source in the upper gastrointestinal tract (above the ligament of Treitz)
- Lower GI bleeding
  - Source in the small intestine or colon (below the ligament of Treitz)
- Obscure GI bleeding
  - Recurrent acute or chronic bleeding for which no known source is identified

Epidemiology

- Incidence
  - Hospital admissions for upper GI bleeding in the U.S. and Europe: approximately 100 per 100,000 persons annually
  - Hospitalization rate for lower GI bleeding is about one-fifth that for upper GI bleeding.

Mechanism

- Disruption of the gastrointestinal mucosa secondary to inflammation, infection, trauma, or cancer
- Vascular abnormalities, such as vascular ectasias or varices due to portal hypertension
- See Differential Diagnosis for discussion of specific sources of GI bleeding.

Symptoms & Signs

- Differentiation of upper GI bleeding from lower GI bleeding
  - Hematemesis
    - Vomiting of blood or altered blood ("coffee grounds" appearance) indicates an upper GI source of bleeding (above the ligament of Treitz).
  - Melena
    - Altered (black) blood passed by rectum (>100 mL blood for 1 melenic stool)
    - Indicates that blood has been present in the GI tract for at least 14 hours

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        - Usually indicates bleeding proximal to ligament of Treitz, but may be as distal as ascending colon
        - The more proximal the bleeding site, the more likely melena will occur.
    - Hematochezia
        - Bright red or maroon rectal bleeding
        - Usually represents a lower GI source of bleeding, although an upper GI lesion may bleed so briskly that blood does not remain in the bowel long enough for melena to develop
        - When hematochezia is the presenting symptom of upper GI bleeding, it is associated with hemodynamic instability and decreasing hemoglobin concentration.
        - Bleeding lesions of the small bowel may present as melena or hematochezia.
        - Hematochezia that presents with abdominal pain may represent ischemic colitis, particularly in elderly persons or those with cardiovascular risk factors.
        - Hematochezia associated with diverticulosis and colonic tumors is typically painless.
    - Other clues
        - Hyperactive bowel sounds and an elevated blood urea nitrogen level (due to volume depletion and blood proteins absorbed in the small intestine) suggest upper GI bleeding.
        - Nasogastric tube lavage usually, but not always, yields blood or "coffee grounds" material.
        - Nonbloody nasogastric aspirate may be seen in up to 16% of patients with upper GI bleeding
            - Usually from a duodenal source
            - Bile-stained appearance does not exclude a bleeding postpyloric lesion because reports of bile in the aspirate are incorrect in about 50% of cases.
        - Mallory–Weiss tears usually occur in patients who have a history of vomiting, retching, or coughing preceding hematemesis, especially in an alcoholic patient
        - Erosive gastropathy (subepithelial hemorrhages or erosions) is commonly related to NSAID use, alcohol intake, and stress.
        - Diverticular (colonic) bleeding is abrupt in onset, usually painless, sometimes massive, and often from the right colon; minor and occult bleeding is not characteristic.
- Physical examination
    - Clinically significant bleeding leads to postural changes in heart rate or blood pressure, tachypnea, tachycardia, and recumbent hypotension.
    - Hemodynamic changes
        - Orthostatic decrease in blood pressure >10 mmHg usually indicates >20% reduction in blood volume.
        - Systolic blood pressure <100 mmHg usually indicates <30% reduction in blood volume.

Differential Diagnosis

- Upper GI bleeding
    - Ulcers (35–62%)
    - Varices (4–31%), depending on group
    - Mallory–Weiss tears (4–13%)
    - Gastroduodenal erosions (3–11%)
    - Erosive esophagitis (2–8%); major bleeding is rare

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    - Cancer (1–4%)
    - No source identified (7–25%)
        - Possible causes: erosive duodenitis, aortoenteric fistulae, vascular lesions, Dieulafoy's lesion, prolapse gastropathy, hemobilia, hemosuccus pancreaticus
    - Non-GI sources of bleeding
        - Generally involves swallowing blood from other sources
            - Epistaxis
            - Hemoptysis
    - Pseudomelena
        - Ingestion of iron, bismuth, licorice, beets, blueberries, charcoal, or red-tinted foods (e.g., red gelatin)
- Lower GI bleeding
    - Diverticulosis (33–50%)
    - Vascular ectasias (8–20% overall; more common in persons >65 years of age)
    - Tumors (19%)
        - Adenocarcinoma, leiomyoma, lymphoma, benign polyps, carcinoid, metastases, lipoma
    - Colitis (18%)
        - Inflammatory bowel disease (Crohn's disease, ulcerative colitis)
        - Infection
        - Ischemic
        - Radiation-induced
    - Hemorrhoids or anal fissures (5%)
    - Meckel's diverticulum
        - Most common cause of significant lower GI bleeding in children
        - With age, decreases in frequency as a cause of bleeding
    - Less common causes
        - Vasculitis, small-bowel varices, duplication cysts, intussusception, polypectomy, rectal ulcers, rectal varices, lymphoid nodular hyperplasia, aortocolic fistulae
- Chronic, obscure GI bleeding (recurrent iron-deficiency anemia or guaiac-positive stool of unknown source)
    - Small-intestinal erosions and ulcers related to NSAID use
    - In adults <40–50 years of age, small-bowel tumors often account for obscure GI bleeding.
    - In patients >50–60 years of age, vascular ectasias are usually responsible.

Diagnostic Approach

- History and physical examination
    - Focused history to identify risk factors (e.g., NSAID or alcohol use) and to evaluate likely source of bleeding
    - Physical examination
        - Evaluate hemodynamic stability by measurement of heart rate and blood pressure.
        - Search for signs of an underlying disorder (e.g., liver disease).
        - Rectal examination is usually indicated; consider anoscopy if appropriate.
- Laboratory evaluation
    - Degree of anemia
- Diagnostic procedures
    - Upper endoscopy in patients with suspected upper GI bleeding
    - Colonoscopy in patients with suspected lower GI bleeding
- Other imaging studies, when indicated

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Laboratory Tests

- Complete blood count
  - Hemoglobin concentration and hematocrit
    - May be normal initially in acute GI bleeding
    - Decrease as extravascular fluid enters the vascular space to restore volume
    - Patients with slow, chronic GI bleeding may have very low hemoglobin and hematocrit values despite normal blood pressure and heart rate.
  - Mild leukocytosis and thrombocytosis are typical.
  - Mean corpuscular volume and red-cell distribution width may be helpful in suggesting chronicity.
    - Expect microcytic anemia with elevated red-cell distribution width in iron-deficiency anemia related to chronic GI blood loss.
- Serum chemistry
  - Elevated blood urea nitrogen level is common in upper GI bleeding.
- Occult blood testing
  - Fecal occult blood test should be performed on stool that has passed naturally rather than that obtained from rectal examination to decrease the likelihood of false-positive results related to local trauma.
  - Testing of nasogastric aspirates that are not grossly bloody is not useful.
- Stool studies
  - Fecal leukocytes may be present in conditions characterized by inflammation or infection.
  - If infection is suspected, send stool cultures for evaluation of likely causes of infectious colitis.

Imaging

- ^{99m}Technetium-labeled red-cell scintigraphy
  - Can detect bleeding rate of 0.1–0.5 mL/min
  - Used primarily as a screening test
    - When bleeding is intermittent and of unclear origin
    - To identify the general area of lower GI bleeding
    - To confirm that bleeding is rapid enough for arteriography to be of value
  - Allows repeated imaging for up to 24 hours after initial injection of the patient's radionuclide-labeled cells
  - Perform in:
    - Patients with continued obscure GI bleeding who require transfusions or repeated hospitalizations
    - In diagnosis of Meckel's diverticulum, especially in the evaluation of young patients
  - Radionuclide scans should be interpreted with caution because results are highly variable, especially from later images.
- Angiography
  - Requires bleeding rate >0.5 mL/min
  - Can detect the site of bleeding in active lower GI bleeding (extravasation of contrast into the gut) and permits treatment with intra-arterial infusion of vasopressin or embolization
  - Even after bleeding has stopped, angiography may identify lesions with abnormal vasculature, such as vascular ectasias or tumors.

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- Radiographic examination of small bowel (e.g., enteroclysis)
  - If enteroscopy and video-capsule endoscopy are negative or unavailable
  - May be considered in patients with iron-deficiency anemia
- Upper GI barium radiography
  - Accuracy ~80% in identifying an upper GI lesion
  - Does not confirm source of bleeding
  - Acceptable alternative to endoscopy in resolved or chronic low-grade bleeding
- Selective mesenteric arteriography
  - When brisk bleeding precludes identification of source at endoscopy

Diagnostic Procedures

- Upper endoscopy
  - Test of choice in patients with upper GI bleeding
  - Should be performed urgently to identify source of bleeding and allow intervention in patients with hemodynamic instability
    - Hypotension
    - Tachycardia
    - Postural changes in heart rate or blood pressure
  - Patients with hematochezia and hemodynamic instability should have upper endoscopy before evaluation of the lower GI tract.
  - Interventions can be performed to decrease bleeding, e.g., injection or cauterization of ulcers and ligation or sclerotherapy of bleeding varices.
  - In patients with occult GI bleeding who have normal colonoscopy, upper endoscopy is usually recommended if iron-deficiency anemia or upper GI symptoms are present.
- Colonoscopy
  - Oral lavage solution followed by colonoscopy is the procedure of choice in patients admitted with lower GI bleeding unless:
    - Bleeding is too massive
    - Sigmoidoscopy has disclosed an obvious actively bleeding lesion
  - Evaluation of a positive test for fecal occult blood generally should begin with colonoscopy, particularly in patients >40 years of age.
- Sigmoidoscopy
  - Patients with presumed lower GI bleeding may undergo early sigmoidoscopy for the detection of obvious, low-lying lesions.
    - Difficult with brisk bleeding
    - Usually not possible to identify the area of bleeding
    - Useful primarily in patients <40 years of age with minor bleeding
- Push enteroscopy
  - May identify probable bleeding sites in 20–40% of patients with obscure GI bleeding
- Video-capsule endoscopy
  - Allows endoscopic examination of the entire small intestine and increases diagnostic yield in obscure GI bleeding
    - Bleeding sites were identified in approximately 30–65% of cases in the initial published reports.
    - Lack of control of the capsule prevents its manipulation and full visualization of the intestine.
    - Tissue cannot be sampled and therapy cannot be applied.
- Intraoperative endoscopy
  - Indicated when all tests are unrevealing in patients with severe recurrent or persistent bleeding requiring repeated transfusions

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Treatment Approach

- Hemodynamic resuscitation and stabilization
- Procedural intervention
  - Endoscopic hemostatic therapy
  - Colonoscopic removal of bleeding polyp or mass
  - Surgical resection if necessary
- Targeted medical therapy

Specific Treatments

Hemodynamic resuscitation and stabilization

- Venous access
  - Large-bore needle (14–18 gauge)
  - Central venous line for major bleeding and patients with cardiac disease
- Monitor vital signs, urine output, and hemoglobin and hematocrit (decrease may lag).
- Gastric lavage is of unproven benefit but clears the stomach before endoscopy.
  - Iced saline may lyse clots and reactivate bleeding; room-temperature tap water may be preferable.
- Intubation may be required to protect the airway.
- Support blood pressure with isotonic fluids (normal saline) and blood products as necessary.
- Type and cross-match blood (6 units for major bleeding)
  - Packed red blood cells
  - Whole blood if massive bleeding
  - Transfuse as necessary on the basis of clinical signs, with a target hematocrit >25–30%.
- Reverse coagulopathy (if present) with fresh-frozen plasma and vitamin K.
  - Consider in cirrhotic patients or those taking anticoagulants.
- Intravenous calcium (up to 10–20 mL 10% calcium gluconate IV over 10–15 min) if serum calcium level decreases due to transfusion of citrated blood
- Surgical standby when bleeding is massive
- Indications for emergency surgery
  - Uncontrolled or prolonged bleeding, severe rebleeding, aortoenteric fistula
  - For intractable variceal bleeding, consider transjugular intrahepatic portosystemic shunt (TIPS).
  - One-third of patients with active bleeding or a nonbleeding visible vessel have further bleeding that requires urgent surgery if they are treated conservatively.

Upper GI bleeding

- Peptic ulcers
  - Endoscopic therapy with bipolar electrocoagulation, heater probe, or injection therapy (e.g., absolute alcohol, 1:10,000 epinephrine) if indicated
  - High-dose, constant IV infusion of proton-pump inhibitor, to increase intragastric pH to 6–7 and enhance clot stability
    - Decreases further bleeding (but does not change mortality), even after use of appropriate endoscopic therapy in patients with high-risk ulcers (active bleeding, nonbleeding visible vessel, and perhaps adherent clot)
    - Many clinical trials have used intravenous omeprazole.

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        - In the U.S., pantoprazole and lansoprazole are the proton-pump inhibitors that are available in an intravenous formulation.
            - IV pantoprazole: initial dose, 80-mg bolus, followed by 8 mg/h
            - IV lansoprazole: initial dose, 60-mg bolus, followed by 6 mg/h
    - Eradication of *Helicobacter pylori* in patients with bleeding ulcers decreases rates of rebleeding to <5%.
    - If a bleeding ulcer develops in a patient taking NSAIDs, the NSAID therapy should be discontinued if possible.
        - If NSAID therapy must be continued, treatment with a proton-pump inhibitor should be continued and the risk–benefit profile of switching to a cyclooxygenase 2 (COX-2)–specific inhibitor should be carefully reviewed.
        - Among nonselective NSAIDs, risk of bleeding seems to be lowest for ibuprofen.
        - Use of misoprostol (100 μg tid or qid, increasing to 200 μg tid or qid, if necessary) can prevent NSAID GI toxicity.
        - If a COX-2 inhibitor is essential, data on celecoxib suggest lowest cardiovascular risk; additional studies are ongoing.
    - Patients with bleeding ulcers unrelated to *H. pylori* or NSAIDs should continue to receive full-dose antisecretory therapy indefinitely.
    - Patients with clean-based ulcers may be discharged on the first hospital day after stabilization if there is no other reason for hospitalization.
    - Patients without clean-based ulcers should usually remain in the hospital for 3 days, since most episodes of recurrent bleeding occur within this time frame.
- Mallory–Weiss tears
    - Bleeding usually occurs on the gastric side of the gastroesophageal junction.
    - Stops spontaneously in 80–90% of patients
    - Endoscopic therapy is indicated for active bleeding.
    - Angiographic therapy with intra-arterial infusion of vasopressin or embolization and operative therapy with oversewing of the tear are rarely required.
- Esophageal varices
    - Endoscopic therapy for acute bleeding and repeated sessions of endoscopic therapy to eradicate esophageal varices significantly reduces rebleeding and mortality.
    - Ligation is the endoscopic therapy of choice for esophageal varices compared with sclerotherapy.
        - Less rebleeding
        - Lower mortality rate
        - Fewer local complication
        - Fewer treatment sessions to achieve variceal eradication
    - Short-term treatment with octreotide (50-μg bolus and 50-μg/h IV infusion for 2–5 days) may help control acute bleeding.
        - Has replaced vasopressin as the medical therapy of choice for acute variceal bleeding
        - Somatostatin and terlipressin, available outside the U.S., are also effective.
    - Long-term treatment with nonselective beta blockers decreases recurrent bleeding from esophageal varices.
        - Commonly given along with long-term endoscopic therapy.
    - In patients who have persistent or recurrent bleeding despite endoscopic and medical therapy, more invasive therapy is warranted.
        - TIPS decreases rebleeding more effectively than endoscopic therapy, although hepatic encephalopathy is more common and mortality rates are similar.

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        - Most patients with TIPS have shunt stenosis within 1–2 years and require reinstrumentation.
        - TIPS is most appropriate for patients with more severe liver disease and those in whom transplant is anticipated.
    - Patients with milder, well-compensated cirrhosis should probably undergo decompressive surgery (e.g., distal splenorenal shunt).
    - Portal hypertension is also responsible for bleeding from gastric varices, varices in the small and large intestine, and portal hypertensive gastropathy and enterocolopathy.
- Hemorrhagic and erosive gastropathy ("gastritis")
    - Removal of the offending agent if possible (e.g., NSAIDs, alcohol).
    - Medical therapy with proton-pump inhibitor; H2 blockers and sucralfate are alternatives.

Small-intestinal bleeding

- Vascular ectasias
    - Treated with endoscopic therapy if possible.
    - Surgical therapy can be used for vascular ectasias isolated to a segment of the small intestine when endoscopic therapy is unsuccessful.
    - Estrogen/progesterone compounds have been used for vascular ectasias, but a double-blind trial found no benefit in prevention of recurrent bleeding.
    - Isolated lesions, such as tumors, diverticula, or duplications, are generally treated with surgical resection.

Colonic bleeding

- Bleeding colonic diverticula stop bleeding spontaneously in approximately 80% of patients.
    - If bleeding persists or recurs, segmental surgical resection is indicated.
- Endoscopic polypectomy, if possible, is used to treat bleeding colonic polyps.
- Colonic tumors typically require surgical resection.
- Bleeding from right colonic vascular ectasias in elderly persons tends to be chronic and only occasionally is hemodynamically significant.
- Surgical therapy is generally required for major, persistent, or recurrent bleeding from the wide variety of colonic sources of GI bleeding that cannot be treated medically or endoscopically.

Monitoring

- Patients with acute GI bleeding typically require hospitalization.
- Patients with subacute or chronic GI bleeding may undergo outpatient evaluation if they do not have significant comorbid conditions.
- Patients who present with active GI bleeding require close monitoring.
- Intensive care unit may be indicated for patients with hemodynamic instability, those requiring blood transfusions, and those with continued active bleeding.
- After endoscopy
    - Patients with lower-risk endoscopic findings (clean-based ulcer) may be discharged on medical therapy.
    - Patients with higher-risk endoscopic findings (active bleeding or visible vessel) require continued inpatient monitoring for several days.
- After colonoscopy, the level of monitoring may be determined by whether definitive intervention has eliminated source of bleeding.

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Complications

- Hypovolemic shock and subsequent end-organ damage
- Complications related to blood transfusions, such as acquired infections or transfusion reaction
- Complications related to procedural interventions, such as perforation and infection

Prognosis

- Upper GI bleeding
  - Mortality rate of ~5%–10%
    - <1% in patients <60 years of age in the absence of cancer or organ failure
  - Patients rarely die of exsanguination; rather, they die of decompensation from other underlying illnesses.
  - Adverse prognostic signs
    - 3 independent clinical predictors of death in patients hospitalized with upper GI bleeding
      - Increasing age
      - Comorbid conditions
      - Hemodynamic compromise (tachycardia or hypotension)
    - Other poor prognostic signs: coagulopathy, immunosuppression, presentation with shock, rebleeding, onset of bleeding in hospital, variceal bleeding, endoscopic stigmata of recent bleeding
  - Peptic ulcer
    - Patients with clean-based ulcers have a rate of recurrent bleeding of approximately 5% and a mortality rate of approximately 2%.
    - Patients with actively bleeding ulcers at endoscopy have a rebleeding rate of approximately 50% and a mortality rate of approximately 10%.
  - Mallory–Weiss tears
    - Bleeding recurs in 0–5% of patients.
  - Esophageal varices
    - Patients with variceal hemorrhage have poorer outcomes than patients with other sources of upper GI bleeding.
  - Stress-related gastric mucosal injury
    - Mortality rate is high because of serious underlying illness.
- Lower GI bleeding
  - Bleeding colonic diverticula
    - Approximately 20–25% of patients have episodes of rebleeding.

Prevention

- Prevention of recurrent bleeding is focused on treatment of the underlying cause.
- Examples
  - Recurrent bleeding ulcer
    - Address 3 main factors in ulcer pathogenesis.
      - Treat *Helicobacter pylori* infection.
      - Discontinue NSAID therapy.
      - Provide acid suppression with a proton-pump inhibitor.
  - Esophageal varices
    - Nonselective beta blocker to decrease portal hypertension
    - TIPS for severe disease

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- GI cancer
  - Treatment of underlying condition, and surgery if appropriate
- Angiodysplasia
  - Rebleeding risk may be as high as 30% after local therapy (endoscopic coagulation).
  - Usual source is another lesion rather than the same lesion.

ICD-9-CM

- 578.9 Hemorrhage of gastrointestinal tract, unspecified

See Also

- Anal fissures
- Cirrhosis and its Complications
- Colorectal Cancer
- Diverticulosis
- Gastrointestinal Endoscopy
- Hemorrhoids
- Inflammatory Bowel Disease
- Peptic Ulcer Disease
- Shock

Internet Sites

- Professionals
  - Clinical trials, GI hemorrhage
    ClinicalTrials.gov, National Institutes of Health
  - Diagnosis of Gastrointestinal Bleeding in Adults
    American Family Physician
- Patients
  - Gastrointestinal Bleeding
    MedlinePlus

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PEARLS

- The stool guaiac test is based on the conversion of a phenol, α-guaiaconic acid, to a blue-colored quinone, guaiacum blue, through the liberation of hydrogen peroxide.
    - In this reaction, free heme or hemoglobin acts as a pseudoperoxidase.
- In "A Study in Scarlet," Dr. Watson is first introduced to Sherlock Holmes in a laboratory at St. Bart's Hospital in which Holmes has just discovered a new test for occult blood aimed at improving on the guaiac test. We are still trying to find Holmes's discovery.
- Dietary influences on the guaiac test include items that produce false-positive results: red meat (or any food containing blood), turnips, horseradish, radishes, grapefruit, cantaloupe, fig, cauliflower, artichokes, mushroom, bananas, or uncooked broccoli.
    - These foods have enough peroxidase activity to create a false-positive test result.
- Ingestion of high doses of vitamin C may cause a false-negative test result, as vitamin C will scavenge free-radical peroxide produced by a positive reaction.
- Iron tablets can make stool black as a false-positive sign of melena and produce a false-positive guaiac test.
- The presence of bile in a gastric aspirate implies an open pylorus.
    - In this setting, a negative result on gastric lavage can be interpreted as making post-pyloric bleeding less likely.
- Angiodysplasia increases with increasing age, but other medical conditions may also increase its frequency; these conditions include chronic renal failure and, possibly, aortic stenosis.

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