therapy and receive standard doses of oral PPI. Approximately one-third of patients with bleeding ulcers will rebleed within the next 1–2 years if no preventive strategies are employed. Prevention of recurrent bleeding focuses on the three main factors in ulcer pathogenesis, *Helicobacter pylori*, NSAIDs, and acid. Eradication of *H. pylori* in patients with bleeding ulcers decreases rates of rebleeding to <5%. If a bleeding ulcer develops in a patient taking NSAIDs, the NSAIDs should be discontinued. If NSAIDs must be given, a cyclooxygenase 2 (COX-2) selective inhibitor (coxib) plus a PPI should be used. PPI co-therapy alone or a coxib alone is associated with an annual rebleeding rate of ~10% in patients with a recent bleeding ulcer, whereas the combination of a coxib and PPI provides a further significant decrease in recurrent ulcer bleeding. Patients with established cardiovascular disease who develop bleeding ulcers while taking low-dose aspirin should restart aspirin as soon as possible after their bleeding episode (1–7 days). A randomized trial showed that failure to restart aspirin was associated with no significant difference in rebleeding (5% vs. 10% at 30 days) but a significant increase in mortality at 30 days (9% vs. 1%) and 8 weeks (13% vs. 1%) compared with immediate reinstitution of aspirin. Patients with bleeding ulcers unrelated to *H. pylori* or NSAIDs should remain on PPI therapy indefinitely. **Peptic ulcers are discussed in Chap. 348.**

MALLORY-WEISS TEARS The classic history is vomiting, retching, or coughing preceding hematemesis, especially in an alcoholic patient. Bleeding from these tears, which are usually on the gastric side of the gastroesophageal junction, stops spontaneously in 80–90% of patients and recurs in only 0–10%. Endoscopic therapy is indicated for actively bleeding Mallory-Weiss tears. Angiographic therapy with embolization and operative therapy with oversewing of the tear are rarely required. **Mallory-Weiss tears are discussed in Chap. 347.**

ESOPHAGEAL VARICES Patients with variceal hemorrhage have poorer outcomes than patients with other sources of UGIB. Urgent endoscopy within 12 h is recommended in cirrhotics with UGIB, and if esophageal varices are present, endoscopic ligation is performed and an IV vasoactive medication (e.g., octreotide 50 μg bolus and 50 μg/h infusion) is given for 2–5 days. Combination endoscopic and medical therapy appears to be superior to either therapy alone in decreasing rebleeding. In patients with advanced liver disease (e.g., Child-Pugh class C with score 10–13), a transjugular intrahepatic portosystemic shunt (TIPS) should be strongly considered within the first 1–2 days of hospitalization because randomized trials show significant decreases in rebleeding and mortality compared with standard endoscopic and medical therapy. Over the long term, treatment with nonselective beta blockers plus endoscopic ligation is recommended because the combination of endoscopic and medical therapy is more effective than either alone in reduction of recurrent esophageal variceal bleeding.

In patients who have persistent or recurrent bleeding despite endoscopic and medical therapy, TIPS is recommended. Decompressive surgery (e.g., distal splenorenal shunt) may be considered instead of TIPS in patients with well-compensated cirrhosis.

Portal hypertension is also responsible for bleeding from gastric varices, varices in the small and large intestine, and portal hypertensive gastropathy and enterocolopathy. Bleeding gastric varices due to cirrhosis are treated with endoscopic injection of tissue adhesive (e.g., *n*-butyl cyanoacrylate), if available; if not, TIPS is performed.

HEMORRHAGIC AND EROSIVE GASTROPATHY ("GASTRITIS") Hemorrhagic and erosive gastropathy, often labeled gastritis, refers to endoscopically visualized subepithelial hemorrhages and erosions. These are mucosal lesions and do not cause major bleeding due to the absence of arteries and veins in the mucosa. Erosions develop in various clinical settings, the most important of which are NSAID use, alcohol intake, and stress. Half of patients who chronically ingest NSAIDs have erosions, whereas up to 20% of actively drinking alcoholic patients with symptoms of UGIB have evidence of subepithelial hemorrhages or erosions.

Stress-related gastric mucosal injury occurs only in extremely sick patients, such as those who have experienced serious trauma, major surgery, burns covering more than one-third of the body surface area, major intracranial disease, or severe medical illness (i.e., ventilator dependence, coagulopathy). Severe bleeding should not develop unless ulceration occurs. The mortality rate in these patients is quite high because of their serious underlying illnesses.

The incidence of bleeding from stress-related gastric mucosal injury has decreased dramatically in recent years, most likely due to better care of critically ill patients. Pharmacologic prophylaxis for bleeding may be considered in the high-risk patients mentioned above. Meta-analyses of randomized trials indicate that PPIs are more effective than H_2 receptor antagonists in reduction of overt and clinically important UGIB without differences in mortality or nosocomial pneumonia.

OTHER CAUSES Other less frequent causes of UGIB include erosive duodenitis, neoplasms, aortoenteric fistulas, vascular lesions (including hereditary hemorrhagic telangiectasias [Osler-Weber-Rendu] and gastric antral vascular ectasia ["watermelon stomach"]), Dieulafoy's lesion (in which an aberrant vessel in the mucosa bleeds from a pinpoint mucosal defect), prolapse gastropathy (prolapse of proximal stomach into esophagus with retching, especially in alcoholics), and hemobilia or hemosuccus pancreaticus (bleeding from the bile duct or pancreatic duct).

Small-Intestinal Sources of Bleeding Small-intestinal sources of bleeding (bleeding from sites beyond the reach of the standard upper endoscope) are often difficult to diagnose and are responsible for the majority of cases of obscure GIB. Fortunately, small-intestinal bleeding is uncommon. The most common causes in adults are vascular ectasias, tumors (e.g., GI stromal tumor, carcinoid, adenocarcinoma, lymphoma, metastases), and NSAID-induced erosions and ulcers. Other less common causes in adults include Crohn's disease, infection, ischemia, vasculitis, small-bowel varices, diverticula, Meckel's diverticulum, duplication cysts, and intussusception.

Meckel's diverticulum is the most common cause of significant LGIB in children, decreasing in frequency as a cause of bleeding with age. In adults <40–50 years, small-bowel tumors often account for obscure GIB; in patients >50–60 years, vascular ectasias and NSAID-induced lesions are more commonly responsible.

Vascular ectasias should be treated with endoscopic therapy if possible. Although estrogen/progesterone compounds have been used for vascular ectasias, a large double-blind trial found no benefit in prevention of recurrent bleeding. Octreotide is also used, based on case series but no randomized trials. A randomized trial reported significant benefit of thalidomide and awaits further confirmation. Other isolated lesions, such as tumors, are generally treated with surgical resection.

Colonic Sources of Bleeding Hemorrhoids are probably the most common cause of LGIB; anal fissures also cause minor bleeding and pain. If these local anal processes, which rarely require hospitalization, are excluded, the most common causes of LGIB in adults are diverticula, vascular ectasias (especially in the proximal colon of patients >70 years), neoplasms (primarily adenocarcinoma), colitis (ischemic, infectious, idiopathic inflammatory bowel disease), and postpolypectomy bleeding. Less common causes include NSAID-induced ulcers or colitis, radiation proctopathy, solitary rectal ulcer syndrome, trauma, varices (most commonly rectal), lymphoid nodular hyperplasia, vasculitis, and aortocolic fistulas. In children and adolescents, the most common colonic causes of significant GIB are inflammatory bowel disease and juvenile polyps.

Diverticular bleeding is abrupt in onset, usually painless, sometimes massive, and often from the right colon; chronic or occult bleeding is not characteristic. Clinical reports suggest that bleeding colonic diverticula stop bleeding spontaneously in ~80% of patients and, on long-term follow-up, rebleed in ~15–25% of patients. Case series suggest endoscopic therapy may decrease recurrent bleeding in the uncommon case when colonoscopy identifies the specific bleeding diverticulum. When diverticular bleeding is found at angiography, transcatheter arterial embolization by superselective technique stops bleeding in a majority of patients. If bleeding persists or recurs, segmental surgical resection is indicated.