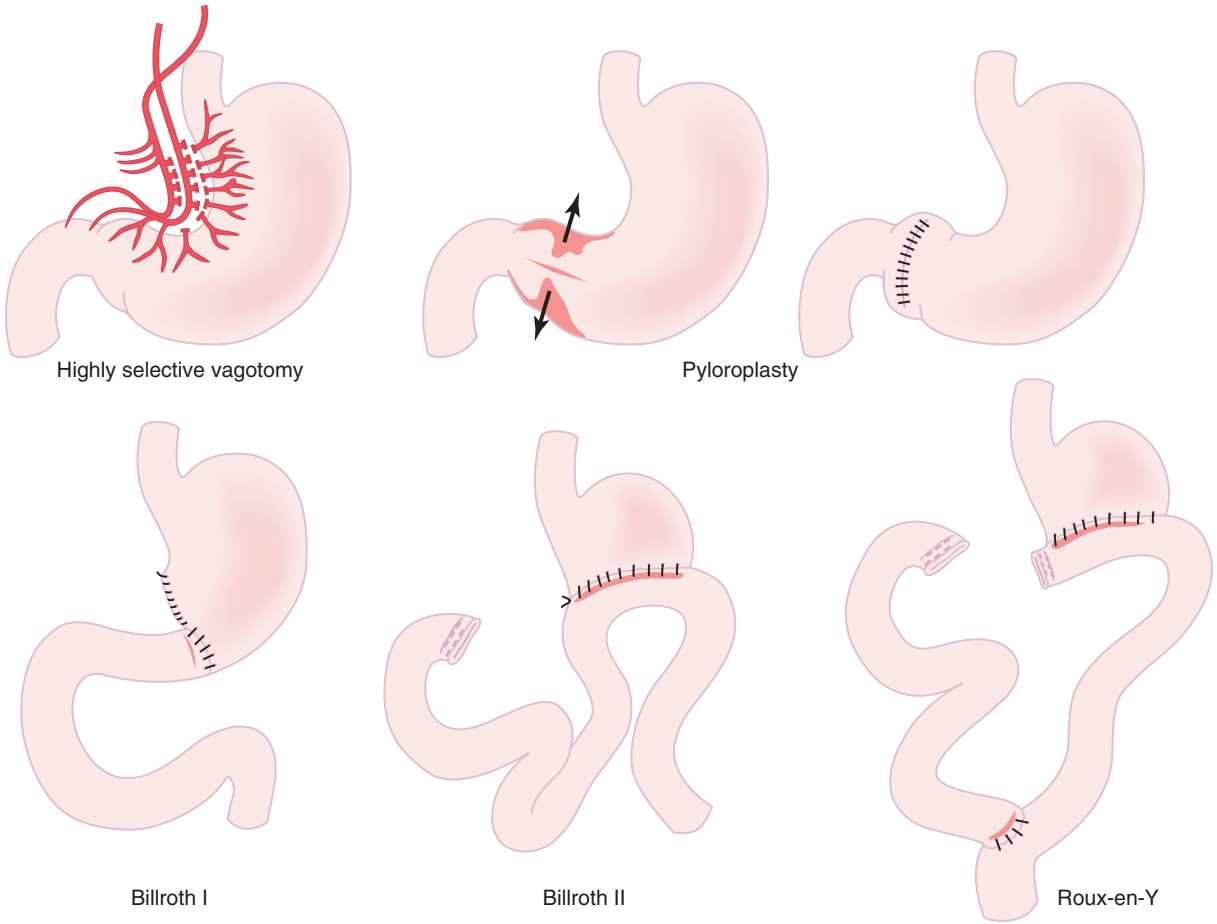

FIGURE 36-8 Endoscopic classification of peptic ulcers with prevalence and risk of rebleeding. (From Laine L, Peterson WL: Medical progress: Bleeding peptic ulcer, N Engl J Med 331:717-727, 1994.)

examination, no evidence of recent or active bleeding can be found, and after feeding has been initiated, oral administration can be substituted for the parenteral route.

Perforation

Perforation, which occurs when a peptic ulcer erodes through the full thickness of the stomach or duodenum, is far less common than bleeding. Ulcer perforation usually leads to peritonitis, which, if untreated, may result in sepsis and death. Patients exhibit a sudden onset of severe abdominal pain that typically begins in the epigastrium and radiates throughout the entire abdomen. When peritonitis is present, physical examination is remarkable for abdominal pain, guarding, rebound tenderness, and boardlike rigidity. The clinical suggestion of perforation may be confirmed in most cases by the presence of free intra-abdominal air (pneumoperitoneum) with either an upright chest radiograph or upright and supine abdominal radiographs. In less obvious instances, computed tomography (CT) or an upper GI water-soluble contrast study may be helpful. Perforation mandates surgical intervention. A perforated duodenal ulcer is typically repaired with an omental patch, whereas a perforated gastric ulcer necessitates either an omental patch or a partial resection.

Gastric Outlet Obstruction

In the era before acid suppression and *H. pylori* therapy, PUD accounted for 60% of the cases of gastric outlet obstruction. More recently, the incidence of both ulcers and obstruction requiring surgery has declined, and estimates indicate that fewer than 5% of patients with duodenal ulcer and less than 1% to 2% with gastric ulcer develop significant gastric outlet obstruction. Gastric outlet obstruction is typically caused by either pyloric channel or duodenal ulcers and may be seen in the setting of acute ulceration, in which edema, spasm, and inflammation lead to obstruction, or as a consequence of chronic ulceration with scarring and fibrosis. Patients usually exhibit symptoms of early satiety, bloating, nausea, vomiting, and weight loss. Endoscopy is the diagnostic test of choice but visualization of the lesion is frequently obscured by the presence of retained food residue. Patients in whom gastric outlet obstruction is suggested should undergo gastric decompression and lavage to remove retained gastric contents before endoscopic examination. Malignancy may now account for 50% of instances of gastric outlet obstruction and should be excluded with adequate biopsy and cytology samples. Occasionally, imaging techniques such as barium upper GI series and radionuclide gastric-emptying scans can also be used to determine the length of the obstructed area and to evaluate gastric emptying. In addition to the correction of fluid, electrolyte, and pH imbalances resulting from persistent vomiting, patients with gastric outlet obstruction should undergo nasogastric decompression for 3 to 5 days. During that time, acid suppression with an intravenous H_2 receptor antagonist or PPI should also be instituted. Adequacy of response may be assessed