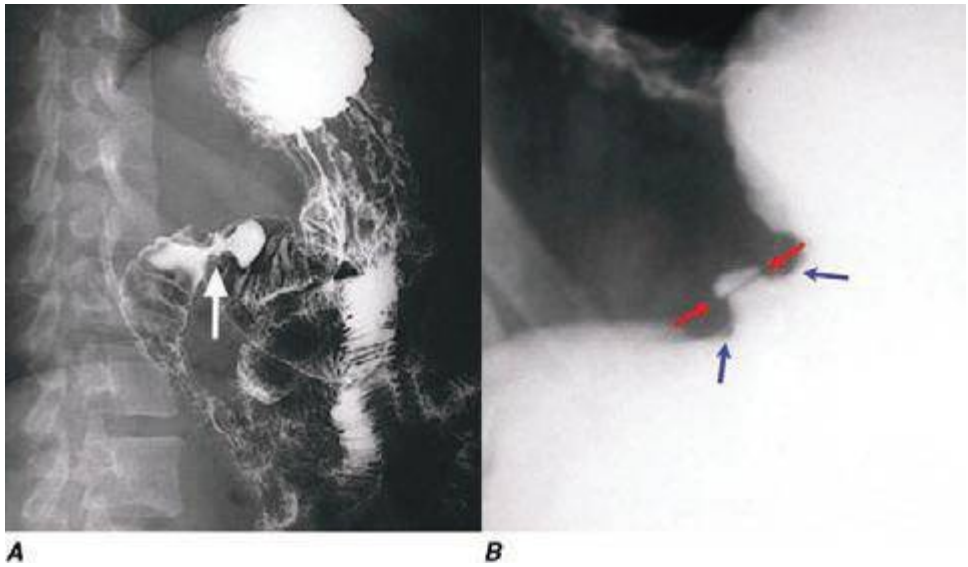

FIGURE 348-10 **Barium study demonstrating** (***A***) a benign duodenal ulcer and (***B***) a benign gastric ulcer.

processes that can mimic this disease, the clinician is often confronted with having to establish the presence of an ulcer. Documentation of an ulcer requires either a radiographic (barium study) or an endoscopic procedure. However, a large percentage of patients with symptoms suggestive of an ulcer have NUD; testing for *H. pylori* and antibiotic therapy (see below) is appropriate for individuals who are otherwise healthy and <45 years of age, before embarking on a diagnostic evaluation (Chap. 54).

Barium studies of the proximal GI tract are still occasionally used as a first test for documenting an ulcer. The sensitivity of older single-contrast barium meals for detecting a DU is as high as 80%, with a double-contrast study providing detection rates as high as 90%. Sensitivity for detection is decreased in small ulcers (<0.5 cm), with presence of previous scarring, or in postoperative patients. A DU appears as a well-demarcated crater, most often seen in the bulb (Fig. 348-10*A*). A GU may represent benign or malignant disease. Typically, a benign GU also appears as a discrete crater with radiating mucosal folds originating from the ulcer margin (Fig. 348-10*B*). Ulcers >3 cm in size or those associated with a mass are more often malignant. Unfortunately, up to 8% of GUs that appear to be benign by radiographic appearance are malignant by endoscopy or surgery. Radiographic studies that show a GU must be followed by endoscopy and biopsy.

Endoscopy provides the most sensitive and specific approach for examining the upper GI tract (Fig. 348-11). In addition to permitting direct visualization of the mucosa, endoscopy facilitates photographic documentation of a mucosal defect and tissue biopsy to rule out malignancy (GU) or *H. pylori*. Endoscopic examination is particularly helpful in identifying lesions too small to detect by radiographic examination, for evaluation of atypical radiographic abnormalities, or to determine if an ulcer is a source of blood loss.

Although the methods for diagnosing *H. pylori* are outlined in Chap. 181, a brief summary will be included here (Table 348-2). Several biopsy urease tests have been developed (PyloriTek, CLOtest, Hpfast, Pronto Dry) that have a sensitivity and specificity of >90–95%. Several noninvasive methods for detecting this organism have been developed. Three types of studies routinely used include serologic testing, the ^{13}C- or ^{14}C-urea breath test, and the fecal *H. pylori* (Hp) antigen test. A urinary Hp antigen test, as well as a refined monoclonal antibody stool antigen test, appears promising.

Occasionally, specialized testing such as serum gastrin and gastric acid analysis or sham feeding may be needed in individuals with complicated or refractory PUD (see "Zollinger-Ellison Syndrome [ZES]," below). Screening for aspirin or NSAIDs (blood or urine) may also be necessary in refractory *H. pylori*–negative PUD patients.

TREATMENT PEPTIC ULCER DISEASE

Before the discovery of *H. pylori,* the therapy of PUD was centered on the old dictum by Schwartz of "no acid, no ulcer." Although acid secretion is still important in the pathogenesis of PUD, eradication of *H. pylori* and therapy/prevention of NSAID-induced disease is the mainstay of treatment. A summary of commonly used drugs for treatment of acid peptic disorders is shown in Table 348-3.

ACID-NEUTRALIZING/INHIBITORY DRUGS

Antacids Before we understood the important role of histamine in stimulating parietal cell activity, neutralization of secreted acid with antacids constituted the main form of therapy for peptic

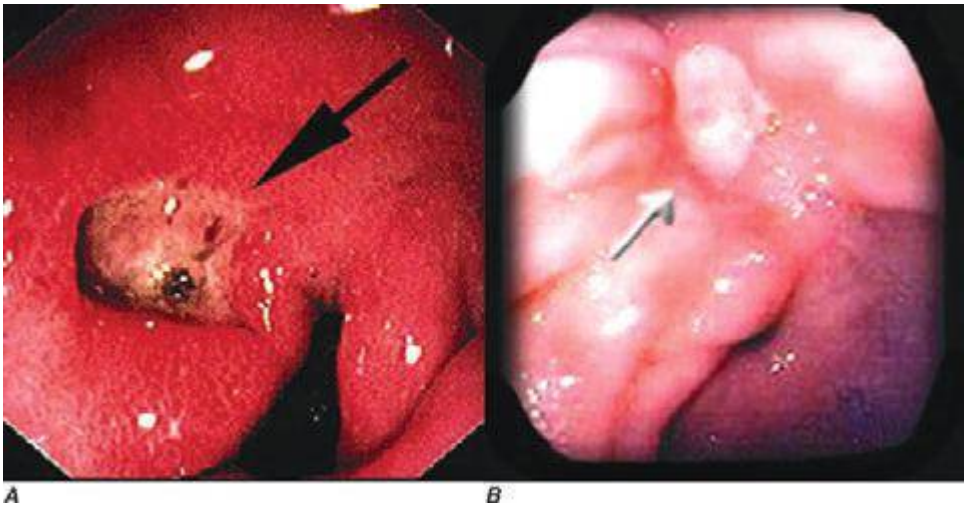

FIGURE 348-11 **Endoscopy demonstrating** (***A***) a benign duodenal ulcer and (***B***) a benign gastric ulcer.