ligation. Despite these interventions, 30% or more of patients with variceal hemorrhage die as a direct consequence of hemorrhage such as hypovolemic shock, hepatic coma, or other complications. Furthermore, more than 50% of patients who survive a first variceal bleed have recurrent hemorrhage within 1 year, and this carries a mortality rate similar to that of the first episode. Thus, patients with risk factors for hemorrhage, including large varices, elevated hepatic venous pressure gradient, previous bleeding, and advanced liver disease may be treated prophylactically with beta-blockers to reduce portal blood flow and with endoscopic variceal ligation. Despite the frequency and risks of variceal hemorrhage, it is important to recognize that cirrhosis patients with small varices that have never bled are at relatively low risk for bleeding and death, and that, even when varices are present, they are only one of several causes of hematemesis.

Barrett Esophagus

Barrett esophagus is a complication of chronic GERD that is characterized by intestinal metaplasia within the esophageal squamous mucosa. The incidence of Barrett esophagus is rising, and it is estimated to occur in as many as 10% of individuals with symptomatic GERD. Barrett esophagus is most common in white males and typically presents between 40 and 60 years of age. *The greatest concern in Barrett esophagus is that it confers an increased risk of esophageal adenocarcinoma.* Genomic sequencing of biopsies involved by Barrett esophagus has revealed the presence of mutations that are shared with esophageal adenocarcinoma, in keeping with the idea that Barrett esophagus is a precursor lesion to cancer. Potentially oncogenic mutations are more numerous when biopsies demonstrate dysplasia, which is detected in 0.2% to 2% of persons with Barrett esophagus each year. The presence of dysplasia, a preinvasive change, is associated with prolonged symptoms, longer segment length, increased patient age, and Caucasian race. Although the vast majority of esophageal adenocarcinomas are associated with Barrett esophagus, it is important to remember that most individuals with Barrett esophagus do not develop esophageal tumors.

MORPHOLOGY

Barrett esophagus can be recognized as one or several tongues or patches of red, velvety mucosa extending upward from the gastroesophageal junction. This metaplastic mucosa alternates with residual smooth, pale squamous (esophageal) mucosa and interfaces with light-brown columnar (gastric) mucosa distally (Fig. 17-7*A, B*). High-resolution endoscopes have increased the sensitivity of Barrett esophagus detection. This has led to subclassification of Barrett esophagus as long segment, which involves 3 cm or more, or short segment, in which less than 3 cm is involved. Available data suggest that the risk of dysplasia correlates with length of esophagus affected.

Diagnosis of Barrett esophagus requires endoscopic evidence of metaplastic columnar mucosa above the gastroesophageal junction. Microscopically, intestinal-type metaplasia is seen as replacement of the squamous esophageal epithelium with goblet cells. These are diagnostic of Barrett esophagus, and have distinct mucous vacuoles that stain pale blue by hematoxylin and eosin and impart the shape of a wine goblet to the remaining cytoplasm (Fig. 17-7*C*). Non-goblet columnar cells, such as gastric type foveolar cells, may also be present. However, whether the latter are sufficient for diagnosis is controversial.

When **dysplasia** is present, it is classified as low grade or high grade. Atypical mitoses, nuclear hyperchromasia, irregularly clumped chromatin, increased nuclear-to-cytoplasmic ratio, and a failure of epithelial cells to mature as they migrate to the esophageal surface are present in both grades of dysplasia (Fig. 17-8*A*). Gland architecture is frequently abnormal and is characterized by budding, irregular shapes, and cellular crowding. High-grade dysplasia (Fig. 17-8*B*) exhibits more severe cytologic and architectural changes. With progression, epithelial cells may invade the lamina propria, a feature that defines intramucosal carcinoma.

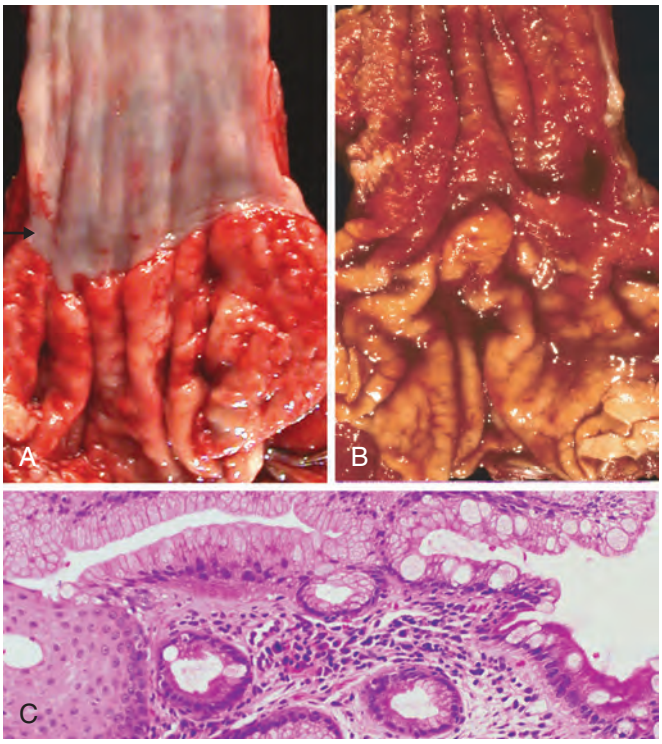

Figure 17-7 Barrett esophagus. **A,** Normal gastroesophageal junction. **B,** Barrett esophagus. Note the small islands of residual pale squamous mucosa within the Barrett mucosa. **C,** Histologic appearance of the gastroesophageal junction in Barrett esophagus. Note the transition between esophageal squamous mucosa (left) and Barrett metaplasia, with abundant metaplastic goblet cells (right).

Clinical Features. **Barrett esophagus can only be identified thorough endoscopy and biopsy, which are usually prompted by GERD symptoms.** Once diagnosed, the best course of management is a matter of debate. Many support periodic endoscopy with biopsy, for dysplasia surveillance. However, randomized trials have failed to demonstrate that surveillance improves patient survival. Furthermore, uncertainties regarding the potential of dysplasia, particularly low grade, to regress spontaneously and limited information on the risk of progression complicate clinical decisions.

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