Rarely, chronic GERD is punctuated by attacks of severe chest pain that may be mistaken for heart disease. Treatment with proton pump inhibitors, which have replaced $H_2$ histamine receptor antagonists, to reduce gastric acidity typically provides symptomatic relief. While the severity of symptoms is not closely related to the degree of histologic damage, the latter tends to increase with disease duration. Complications of reflux esophagitis include ulceration, hematemesis, melena, stricture development, and Barrett esophagus.

Hiatal hernia can give rise to symptoms, such as heartburn and regurgitation of gastric juices, that are similar to those of GERD. It is characterized by separation of the diaphragmatic crura and protrusion of the stomach into the thorax through the resulting gap. Congenital hiatal hernias are recognized in infants and children, but many are acquired in later life. Hiatal hernia is symptomatic in fewer than 10% of adults, but can be a cause of lower esophageal sphincter incompetence.

## Eosinophilic Esophagitis

The incidence of eosinophilic esophagitis is increasing markedly. Symptoms include food impaction and dysphagia in adults and feeding intolerance or GERD-like symptoms in children. The cardinal histologic feature is large numbers of intraepithelial eosinophils, particularly superficially (Fig. 17-5*B*). Their abundance can help to differentiate eosinophilic esophagitis from GERD, Crohn disease, and other causes of esophagitis. In addition, unlike patients with GERD, acid reflux is not prominent and high doses of proton pump inhibitors usually do not provide relief. *The majority of individuals with eosinophilic esophagitis are atopic* and many have atopic dermatitis, allergic rhinitis, asthma, or modest peripheral eosinophilia. Treatments include dietary restrictions to prevent exposure to food allergens, such as cow's milk and soy products, and topical or systemic corticosteroids.

## Esophageal Varices

Venous blood from the GI tract passes through the liver, via the portal vein, before returning to the heart. This circulatory pattern is responsible for the first-pass effect in which drugs and other materials absorbed in the intestines are processed by the liver before entering the systemic circulation. Diseases that impede this flow cause portal hypertension and can lead to the development of esophageal varices, an important cause of esophageal bleeding.

***Pathogenesis.*** **Portal hypertension results in the development of collateral channels at sites where the portal and caval systems communicate.** These collateral veins allow some drainage to occur, but at the same time they lead to development of congested subepithelial and submucosal venous plexi within the distal esophagus and proximal stomach. These vessels, termed *varices,* develop in the vast majority of cirrhotic patients, most commonly in association with alcoholic liver disease. Worldwide, hepatic schistosomiasis is the second most common cause of varices. A more detailed consideration of portal hypertension is given in Chapter 18.

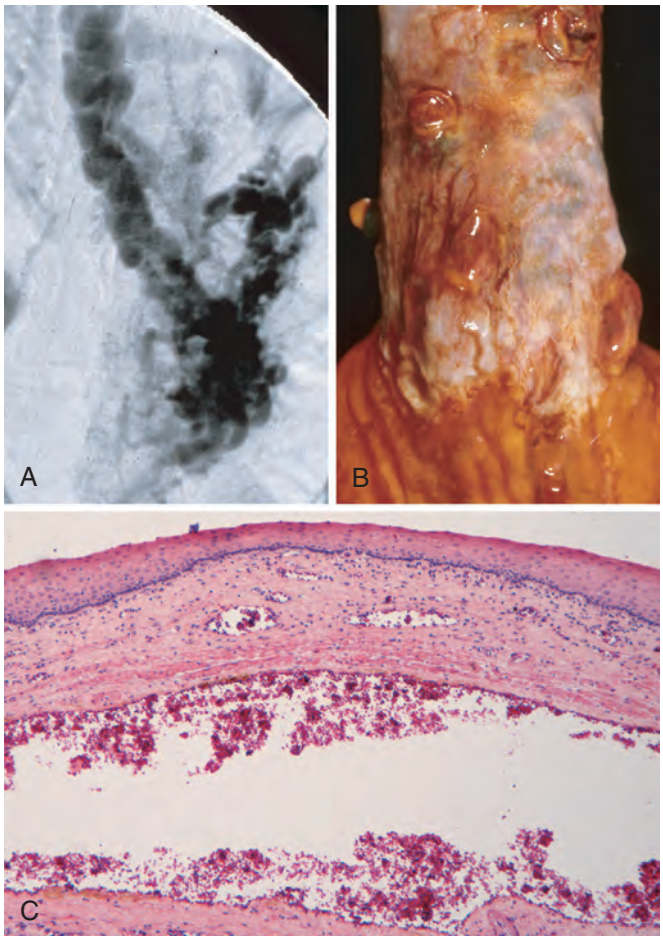


**Figure 17-6** Esophageal varices. **A,** Although no longer used as a diagnostic approach, this angiogram demonstrates several tortuous esophageal varices. **B,** Collapsed varices are present in this postmortem specimen corresponding to the angiogram in **A**. The polypoid areas represent previous sites of variceal hemorrhage that have been ligated with bands. **C,** Dilated varices beneath intact squamous mucosa.

### MORPHOLOGY

Varices are tortuous dilated veins lying primarily within the submucosa of the distal esophagus and proximal stomach (Fig. 17-6*A*). Venous channels directly beneath the esophageal epithelium may also become massively dilated. Varices may not be grossly obvious in surgical or postmortem specimens, because they collapse in the absence of blood flow (Fig. 17-6*B*) and are obscured by the overlying mucosa (Fig. 17-6*C*). Variceal rupture results in hemorrhage into the lumen or the esophageal wall, in which case the overlying mucosa appears ulcerated and necrotic. If rupture has occurred in the past, venous thrombosis, inflammation, and evidence of prior therapy may also be present.

***Clinical Features.*** Gastroesophageal varices are present in nearly half of the patients with cirrhosis, and 25-40% of patients with cirrhosis develop variceal bleeding. Approximately 12% of previously asymptomatic varices bleed each year. Variceal hemorrhage is an emergency that can be treated medically by inducing splanchnic vasoconstriction or endoscopically by sclerotherapy (injection of thrombotic agents), balloon tamponade, or variceal