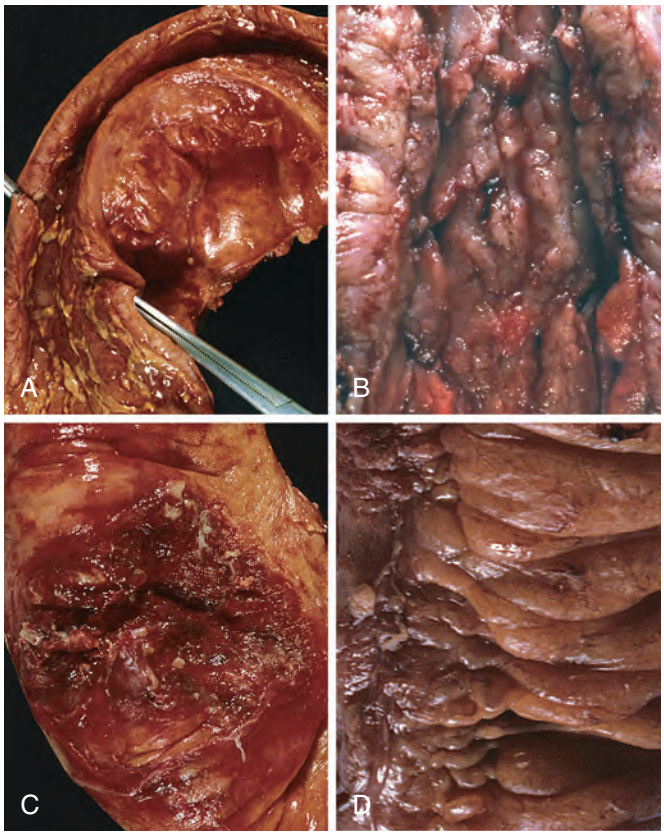

Figure 17-34 Gross pathology of Crohn disease. **A,** Small-intestinal stricture. **B,** Linear mucosal ulcers, which impart a cobblestone appearance to the mucosa, and thickened intestinal wall. **C,** Perforation and associated serositis. **D,** Creeping fat.

MORPHOLOGY

Crohn disease may occur in any area of the GI tract, but the most common sites involved at presentation are the terminal ileum, ileocecal valve, and cecum. Disease is limited to the small intestine alone in about 40% of cases; the small intestine and colon are both involved in 30% of patients; the remainder have only colonic involvement. The presence of multiple, separate, sharply delineated areas of disease, resulting in **skip lesions**, is characteristic of Crohn disease and may help in the differentiation from ulcerative colitis. Strictures are common in Crohn disease, but do not generally develop in ulcerative colitis (Fig. 17-34*A*).

The earliest lesion, the **aphthous ulcer**, may progress, and multiple lesions often coalesce into elongated, serpentine ulcers oriented along the axis of the bowel (Fig. 17-34*B*). Edema and loss of the normal mucosal texture are common. Sparing of interspersed mucosa, a result of the patchy distribution of Crohn disease, results in a coarsely textured, **cobblestone** appearance in which diseased tissue is depressed below the level of normal mucosa (Fig. 17-34*B*). **Fissures** frequently develop between mucosal folds and may extend deeply to become fistula tracts or sites of perforation (Fig. 17-34*C*). The intestinal wall is thickened and rubbery as a consequence of transmural edema, inflammation, submucosal fibrosis, and hypertrophy of the muscularis propria, all of which contribute to stricture formation (Fig. 17-34*A*). In cases with extensive transmural disease, mesenteric fat frequently extends around the serosal surface **(creeping fat)** (Fig. 17-34*D*).

The microscopic features of active Crohn disease include abundant neutrophils that infiltrate and damage crypt epithelium. Clusters of neutrophils within a crypt are referred to as **crypt abscesses** and are often associated with crypt destruction. Ulceration is common in Crohn disease, and there may be an abrupt transition between ulcerated and adjacent normal mucosa. Even in areas where gross examination suggests diffuse disease, microscopic pathology can appear patchy. Repeated cycles of crypt destruction and regeneration lead to **distortion of mucosal architecture**; the normally straight and parallel crypts take on bizarre branching shapes and unusual orientations to one another (Fig. 17-35*A*). Epithelial metaplasia, another consequence of chronic relapsing injury, often takes the form of gastric antral-appearing glands, and is called pseudopyloric metaplasia. **Paneth cell metaplasia** may also occur in the left colon, where Paneth cells are normally absent. These architectural and metaplastic changes may persist even when active inflammation has resolved. Mucosal atrophy, with loss of crypts, may occur after years of disease. **Noncaseating granulomas** (Fig. 17-35*B*), a hallmark of Crohn disease, are found in approximately 35% of cases and may occur in areas of active disease or uninvolved regions in any layer of the intestinal wall (Fig. 17-35*C*). Granulomas may also be present in mesenteric lymph nodes. Cutaneous granulomas form nodules that are referred to as **metastatic Crohn disease** (a misnomer since there is no cancer). The absence of granulomas does not preclude a diagnosis of Crohn disease.

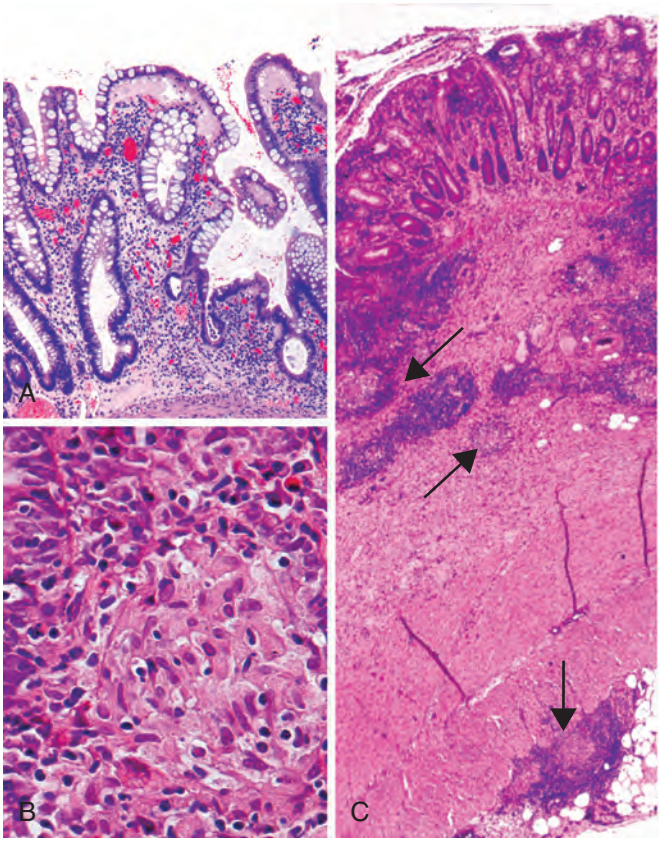

Figure 17-35 Microscopic pathology of Crohn disease. **A,** Haphazard crypt organization results from repeated injury and regeneration. **B,** Noncaseating granuloma. **C,** Transmural Crohn disease with submucosal and serosal granulomas (arrows).