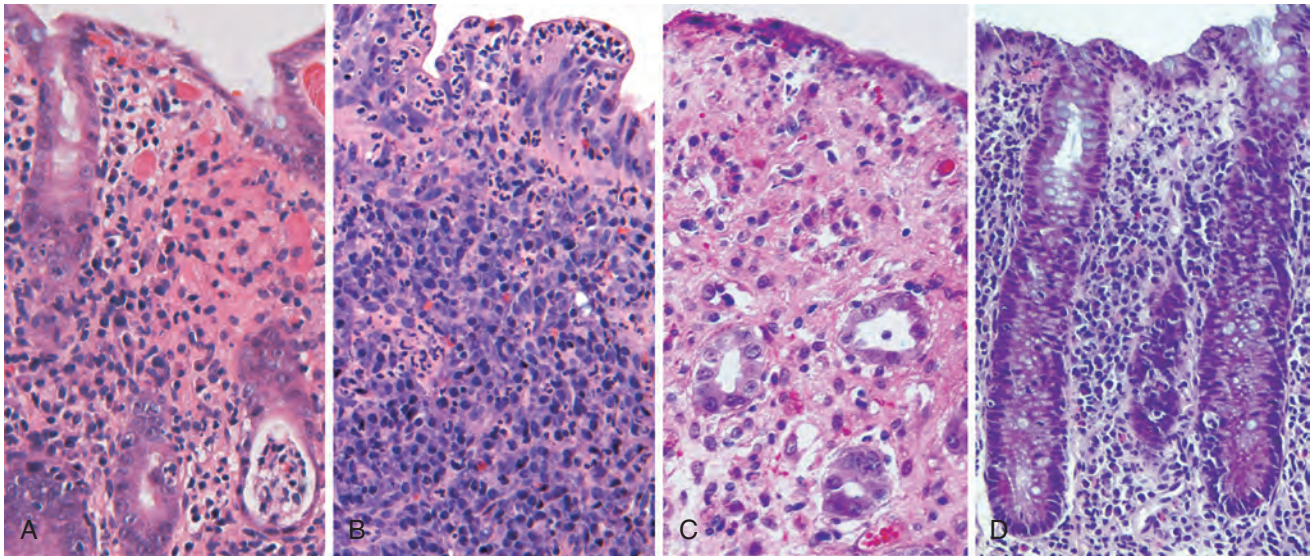

Figure 17-28 Bacterial enterocolitis. **A,** *Campylobacter jejuni* infection produces acute, self-limited colitis. Neutrophils can be seen within the surface and crypt epithelia and a crypt abscess is present at the lower right. **B,** In *Yersinia* infection the surface epithelium can be eroded by neutrophils and the lamina propria is densely infiltrated by sheets of plasma cells admixed with lymphocytes and neutrophils. **C,** Enterohemorrhagic *E. coli* O157:H7 results in an ischemia-like morphology with surface atrophy and erosion. **D,** Enteroinvasive *E. coli* infection is similar to other acute, self-limited colitides such as those caused by *Campylobacter jejuni*. Note the maintenance of normal crypt architecture and spacing, despite abundant intraepithelial neutrophils.

Pathogenesis. *Shigella are resistant to the harsh acidic environment of the stomach, thereby explaining the extremely low infective dose.* Once in the intestine, organisms are taken up by M, or microfold cells. These are epithelial cells, which are specialized for sampling and presentation of luminal antigens. *Shigella* proliferate intracellularly, escape into the lamina propria, and are phagocytosed by macrophages, in which they induce apoptosis. The ensuing inflammatory response damages surface epithelia and allows *Shigella* within the intestinal lumen to gain access to the basolateral membranes of colonic epithelial cells, which is the preferred domain for invasion. All *Shigella* spp. carry virulence plasmids, some of which encode a type III secretion system capable of directly injecting bacterial proteins into the host cytoplasm. *S. dysenteriae* serotype 1 also release the Shiga toxin Stx, which inhibits eukaryotic protein synthesis, resulting in host cell damage and death.

MORPHOLOGY

Shigella infections are most prominent in the left colon, but the ileum may also be involved, perhaps reflecting the abundance of M cells in the dome epithelium over the Peyer patches. The mucosa is hemorrhagic and ulcerated, and pseudomembranes may be present. The histology of early cases is similar to other acute self-limited colitides, such as *Campylobacter* colitis, but because of the tropism for M cells, aphthous-appearing ulcers similar to those seen in Crohn disease may occur. The potential for confusion with chronic inflammatory bowel disease is significant, particularly if there is distortion of crypt architecture.

Clinical Features. After an incubation period of up to 1 week, *Shigella* causes self-limited disease characterized by about 1 week of diarrhea, fever, and abdominal pain. The initially watery diarrhea progresses to a dysenteric phase in approximately 50% of patients, and constitutional symptoms can persist for as long as 1 month. The subacute presentation that develops in a minority of adults is characterized by several weeks of waxing and waning diarrhea that can mimic new-onset ulcerative colitis. While duration is typically shorter in children, severity is often much greater. Confirmation of *Shigella* infection requires stool culture.

Complications of *Shigella* infection are uncommon and include a triad of sterile reactive arthritis, urethritis, and conjunctivitis that preferentially affects HLA-B27-positive men between 20 and 40 years of age. Hemolytic-uremic syndrome, which is typically associated with *enterohemorrhagic E. coli* (EHEC), may also occur after infection with *S. dysenteriae* serotype 1 that secrete Shiga toxin (Chapter 20). Toxic megacolon and intestinal obstruction are uncommon complications. Antibiotic treatment shortens the clinical course and reduces the duration of organism shedding in stools, but antidiarrheal medications are contraindicated because they can prolong symptoms and delay *Shigella* clearance.

Salmonella

Salmonella, which are classified within the Enterobacteriaceae family of gram-negative bacilli, are divided into *Salmonella typhi*, the causative agent of typhoid fever (discussed in the next section) and nontyphoid *Salmonella*. The latter are the causative agent of salmonellosis, which is usually due to *S. enteritidis*; more than 1 million cases occur each year in the United States, and the prevalence is even greater in developing countries. Infection is most common in young children and older adults, with peak incidence in the summer and fall. Salmonella may also cause food poisoning by ingestion of contaminated food, particularly raw or undercooked meat, poultry, eggs, and milk. Centralized food processing can lead to large