stomach. The erythematous stripes are created by ectatic mucosal vessels. Histologically, the antral mucosa shows reactive gastropathy with dilated capillaries containing fibrin thrombi. While most often idiopathic, GAVE can also be associated with cirrhosis and systemic sclerosis. Patients may present with occult fecal blood or iron deficiency anemia.

Chronic Gastritis

The most common cause of chronic gastritis is infection with the bacillus *H. pylori*. Autoimmune gastritis, the most common cause of diffuse atrophic gastritis, represents less than 10% of cases of chronic gastritis, but is the most common form of chronic gastritis in patients without *H. pylori* infection. However, it is important to recognize that longstanding *H. pylori* infection can also result in atrophic gastritis, typically in a multifocal rather than diffuse pattern. Less common causes of chronic gastritis include radiation injury, chronic bile reflux, mechanical injury (e.g. an indwelling nasogastric tube), and involvement by systemic diseases, such as Crohn disease, amyloidosis, or graft-versus-host disease.

In contrast to acute gastritis, the symptoms associated with chronic gastritis are typically less severe but more persistent. Nausea and upper abdominal pain are typical, sometimes with vomiting, but hematemesis is uncommon.

Helicobacter pylori Gastritis

H. pylori are spiral-shaped or curved bacilli present in gastric biopsy specimens of almost all patients with duodenal ulcers as well as most individuals with gastric ulcers or chronic gastritis. Acute *H. pylori* infection does not produce sufficient symptoms to come to medical attention in most cases; it is the chronic gastritis that ultimately causes the individual to seek treatment. *H. pylori* organisms are present in 90% of individuals with chronic gastritis affecting the antrum.

Epidemiology. In the United States, *H. pylori* infection is associated with poverty, household crowding, limited education, African American or Mexican American ethnicity, residence in rural areas, and birth outside of the United States. Humans are the primary carriers, suggesting that transmission is primarily by the fecal-oral route. Infection is typically acquired in childhood and persists for life without treatment. Improved sanitation in the United States likely explains the marked reduction in *H. pylori* infection rates among younger people that has resulted in a cohort effect. For example, the prevalence of *H. pylori* infection in those younger than 12 years old is less than 15% relative to the 50% to 60% prevalence in those older than 60 years of age. Accordingly, colonization rates vary from less than 10% to more than 80% worldwide, as a function of age and geography.

***Pathogenesis. H. pylori* infection most often presents as a predominantly antral gastritis with normal or increased acid production.** Local gastrin production may be increased, but hypergastrinemia (increased serum gastrin) is uncommon. When inflammation remains limited to the antrum, increased acid production results in greater risk of duodenal peptic ulcer (see later). In other patients gastritis may progress to involve the gastric body and fundus. This *multifocal atrophic gastritis* is associated with patchy mucosal atrophy, reduced parietal cell mass and acid secretion, intestinal metaplasia, and increased risk of gastric adenocarcinoma. Thus, there is an inverse relationship between duodenal ulcer and gastric adenocarcinoma that correlates with the pattern of gastritis. The bacterial and host factors that determine which pattern develops in an individual patient are discussed later.

H. pylori organisms have adapted to the ecologic niche provided by gastric mucus. Its virulence is linked to the following factors:

- *Flagella*, which allow the bacteria to be motile in viscous mucus
- *Urease*, which generates ammonia from endogenous urea and thereby elevates local gastric pH and enhances bacterial survival
- *Adhesins* that enhance bacterial adherence to surface foveolar cells
- *Toxins*, such as cytotoxin-associated gene A *(CagA)*, that may be involved in disease progression

Variation in these and other bacterial factors are strongly linked to outcome. For example, *CagA* gene and the associated 20 gene pathogenicity islands are present in 50% of *H. pylori* isolates overall but in 90% of *H. pylori* isolates found in populations with elevated gastric cancer risk. This may, in part, be because CagA expressing strains can effectively colonize the gastric body and cause multifocal atrophic gastritis.

Host factors also play an important role in the outcome of *H. pylori* infection. Genetic polymorphisms that lead to increased expression of the proinflammatory cytokines tumor necrosis factor (TNF) and interleukin-1β (IL-1β) or decreased expression of the antiinflammatory cytokine interleukin-10 (IL-10) are associated with development of pangastritis, atrophy, and gastric cancer. Iron deficiency may also be a risk factor for *H. pylori*–associated gastric cancer. The course of *H. pylori* gastritis is, therefore, the result of interplay between gastroduodenal mucosal defenses, inflammatory responses, and bacterial virulence factors.

MORPHOLOGY

Gastric biopsy specimens generally demonstrate *H. pylori* in infected individuals. The organism is concentrated within the superficial mucus overlying epithelial cells in the surface and neck regions. The distribution can be irregular, with areas of heavy colonization adjacent to those with few organisms. In extreme cases, the organisms carpet the luminal surfaces of foveolar and mucous neck cells, and can even extend into the gastric pits. Organisms are most easily demonstrated with special stains (Fig. 17-12A). *H. pylori* display tropism for gastric epithelia and are generally not found in association with intestinal metaplasia or duodenal epithelium.

Within the stomach, *H. pylori* are most often found in the antrum (Table 17-2). Although there is often concordance between colonization of the antrum and cardia, infection of the