It is now recognized that there is a two-way interaction between *Helicobacter pylori* gastritis and gastric physiology. *H. pylori* gastritis can alter gastric physiology, leading to increased or decreased acid secretion depending on the pattern of gastritis present. These changes in physiology are related to the disease outcome, with increased acid secretion leading to duodenal ulcer disease and reduced acid secretion being a risk factor for gastric cancer. Gastric acid secretion also affects the pattern of gastritis induced by the infection, with low acid secretion leading to a pangastritis and possibly atrophy. This two-way interaction between *H. pylori* gastritis and acid secretion is likely to be involved in the evolution of the disease. It also can explain the more potent reduction of acid secretion produced by proton-pump inhibitors in *H. pylori*-positive subjects as well as rebound acid hypersecretion being more marked in *H. pylori*-negative subjects.

OVERVIEW OF THE NORMAL STRUCTURE AND FUNCTION OF THE STOMACH

Understanding the effects of *H. pylori* infection on gastric functions requires some knowledge of gastric physiology. The stomach consists of two distinct regions that differ morphologically and functionally (*Fig. 1*). The proximal two thirds of the stomach is referred to as the body region. This region contains the acid-secreting parietal cells and, in close proximity to them, the histamine-releasing enterochromaffin-like (ECL) cells. The distal one third of the stomach does not contain any acid-secreting cells but contains G cells, which release the hormone, gastrin. In close proximity to the antral G cells are somatostatin-producing...
D cells.

Figure 1.
Role of gastrin in the regulation of acid secretion. Protein components of food stimulate the G cells in antral mucosa to release gastrin. The hormone circulates and stimulates the body region of the stomach to secrete acid. This occurs by gastrin stimulating release of histamine from enterochromaffin-like (ECL) cells that then activates the H2 receptors on the acid producing parietal cells (P). Overproduction of acid is prevented by low antral pH inhibiting gastrin release. This inhibitory control is mediated by release of somatostatin from D cells situated in close proximity to the G cells.

The thought, sight, smell, or taste of food all stimulate acid secretion. This stimulation is referred to as the cephalic phase of acid secretion and is activated by the vagus nerve. When food enters the stomach, the protein component stimulates the antral G cells to release gastrin, which circulates and stimulates the proximal body region to secrete acid. The gastrin stimulates the body mucosa to secrete acid by activating receptors on the ECL cells, which then release histamine, which, in turn, stimulates the acid-producing parietal cells by their H2 receptors. Gastrin also exerts trophic effects on the acid-secreting mucosa, and this effect is most marked on the ECL cells.

The amount of gastrin released by the antral G cells is regulated by intragastric pH, and this serves as a negative feedback control to prevent hypersecretion of acid. When the pH of gastric juice falls, this inhibits further release of gastrin. This inhibitory influence of intragastric acid is mediated through it stimulating the release of somatostatin from the D cells situated close to the antral G cells.

When a meal is consumed, the protein component of the meal increases acid secretion in two ways. First, the protein stimulates the G cells directly to release gastrin. Second, the buffering effect of the protein raises intragastric pH and removes the somatostatin-mediated inhibition of gastrin release. The increase in gastrin stimulates increased acid secretion. The increased acid secretion eventually overcomes the buffering effect of the food, resulting in lowering of intragastric pH again. Once this lowering occurs, the release of gastrin is inhibited, and acid secretion falls. This inhibition of gastrin release prevents prolonged and excessive secretion of acid, which could be injurious to the mucosa.

EFFECTS OF HELICOBACTER PYLORI GASTRITIS ON GASTRIC ACID SECRETION
Helicobacter pylori

exerts diverse effects on gastric acid secretion. It can result in increased acid secretion, decreased acid secretion, or no
overall change in gastric acid secretion. The effect of Helicobacter pylori infection on acid secretion depends on the pattern of
 gastritis induced by the infection. In particular, the effect depends on the distribution of the gastritis between the antral
 and body region of the stomach and on the degree of atrophy of the mucosa produced by the gastritis. Nonatrophic
 antral-predominant gastritis results in increased acid secretion, and this is seen in patients who develop duodenal
 ulceration. An atrophic pangastritis involving the body region of the stomach as well as the antral region results in
 reduced gastric acid secretion, and this pattern is seen in subjects who develop proximal gastric ulcers and gastric
 cancer. In most patients with Helicobacter pylori

infection, there is relatively little atrophy, and the inflammation is more marked in the antrum but also involves to some
extent the body mucosa. This pattern of gastritis results in no overall change in gastric acid secretion. The mechanism
by which these different patterns of gastritis result in different disturbances in gastric function is examined in more detail.

**Helicobacter pylori and Gastric Acid Hypersecretion in Duodenal Ulcer Patients**

In duodenal ulcer patients, Helicobacter pylori infection colonizes the entire stomach. The inflammation of the underlying mucosa induced by the infection is more
marked in the distal antral region with relative sparing of the acid-secreting body region of the stomach. The gastritis is accompanied by little, if any, atrophy of the mucosa. This pattern of gastritis results in increased release of
 gastrin, and this is accompanied by increased acid secretion.

The increased release of gastrin from the antral mucosa is evident under resting conditions and becomes more
pronounced after ingestion of a protein-containing meal or after stimulation by the intravenous administration of
 gastrin-releasing peptide (Fig. 2). There are two biologically active circulating forms of gastrin, G17 and G34.

G17 is the form of the hormone that increases after ingestion of a meal, and this form is increased in Helicobacter pylori infection. The hypergastrinemia resolves within 2 to 14 days of starting anti-Helicobacter pylori therapy, indicating that it is caused by the
infection.
Figure 2. Gastrin (A) and acid response (B) to stimulation with gastrin releasing peptide in *H. pylori* negative healthy volunteers and duodenal ulcer patients before and at 1 month and 1 year after eradication of the infection. Results are mean values for 10 patients. Note that the increased gastrin resolves more quickly than the increased acid. This can be explained by the increased gastrin inducing ECL cell hyperplasia, which will persist for some time after resolution of the hypergastrinaemia.

There has been considerable interest in the underlying mechanism by which *H. pylori* infection results in increased gastrin release by the antral mucosa. The release of gastrin normally is inhibited by low intraluminal pH, and this influence is exerted through the release of somatostatin from D cells situated close to the G cells. [14] [21] [22] [23] [27] [48] [52] [58] [60] The exaggerated gastrin response in *H. pylori*-infected duodenal ulcer subjects is mainly secondary to impairment of this somatostatin-mediated acid inhibitory control of gastrin release. In *H. pylori*-infected subjects, the concentration of somatostatin and of messenger RNA for somatostatin is reduced within the antral mucosa. [18] [25] [43] [49] [54] The mechanism by which the infection or accompanying inflammation results in this depletion of somatostatin is unclear. It may be related to the ammonia produced by the organism's urease activity elevating antral surface pH. [34] This effect of ammonia would reduce the stimulation of the D cell by intraluminal acid and result in hypofunction and atrophy of the D cells. [26]

It is well recognized that reduction of intragastric acidity by proton-pump inhibitor therapy or by pernicious anemia results in depletion of antral somatostatin concentrations. [1] Inhibition of acid secretion by proton-pump inhibitors also reproduces the exaggerated response to gastrin-releasing peptides seen in *H. pylori*-infected subjects. [19]

Other factors may contribute to the exaggerated antral gastrin release caused by *H. pylori* in duodenal ulcer patients. It
is possible that cytokines overproduced by the inflammatory infiltrate may modify antral neuroendocrine function. Bioactive substances produced by the bacterium itself might have an effect on G and D cell regulation.

Functional studies have confirmed that *H. pylori* infection impairs the acid-mediated inhibitory control of gastrin release. Tarnaski et al have shown that inhibition of gastrin release by intragastric acid is impaired in subjects with *H. pylori* infection. There is substantial morphologic and functional evidence that the exaggerated gastrin response induced by *H. pylori* infection is secondary to impairment of the acid-stimulated, somatostatin-mediated acid inhibitory control.

The increased gastrin release produced by *H. pylori* infection in duodenal ulcer patients with nonatrophic antral-predominant gastritis is accompanied by increased acid secretion. Basal acid output is increased approximately twofold, and this resolves after eradication of the infection. The acid response to stimulation with gastrin-releasing peptide also is markedly increased in *H. pylori*-infected duodenal ulcer patients, and this response decreases after eradication of the infection (see Fig. 2). The increased acid secretion results in an increased duodenal acid load, and this resolves after treatment of the infection. Some, but not all, studies have shown that eradicating *H. pylori* infection also results in a fall in the maximal acid output in duodenal ulcer patients.

Although the fall in serum gastrin occurs within a few days of starting anti-*H. pylori* therapy, the fall in acid secretion occurs more slowly, being most evident 6 months after eradicating the infection. This delayed resolution of the acid hypersecretion may be explained by the hypergastrinemia causing acid hypersecretion by two distinct mechanisms. The oxyntic mucosa is stimulated directly to secrete acid, and the increased acid induced by this mechanism resolves simultaneously with resolution of the hypergastrinemia. Hypergastrinemia also exerts trophic effects on the oxyntic mucosa, however, resulting in hyperplasia and hyperfunctioning of the histamine-releasing ECL cells. These changes take weeks or months to reverse after removing the trophic stimulus and result in some persisting acid hypersecretion for some time after resolution of the hypergastrinemia. Gastrin also exerts trophic effects on the parietal cells, and this may contribute to the increased maximal acid output characteristic of patients with the duodenal ulcer pattern of *H. pylori* gastritis.

Long before the discovery of *H. pylori* infection, several disturbances of gastric function were recognized to be present in duodenal ulcer patients and were thought to be important in the cause of the disease. Such patients were recognized to secrete excessive amounts of acid under fasting conditions and to have exaggerated acid response to a meal. Duodenal ulcer patients were recognized to have a larger capacity to secrete acid under maximal stimulation by gastrin, and this was shown to be due to them having an increased number of parietal cells. Further studies indicated that duodenal ulcer patients had impaired acid inhibitory control of gastrin release. It is now recognized that most of these previously recognized abnormalities of gastric function observed in duodenal ulcer patients can be explained by the effects of *H. pylori* infection.

**Summary of the Role of Helicobacter pylori in Pathophysiology of Duodenal Ulcer Disease**

The role of *H. pylori* infection in the pathophysiology of duodenal ulcer disease is summarized in Figure 3. The nonatrophic antral-confined gastritis disrupts the acid-mediated control of gastrin release. The increased antral gastrin release stimulates the healthy body region of the stomach to secrete excessive amounts of acid. The hypergastrinemia also increases the capacity of the oxyntic mucosa-secreted acid by its trophic effects on the ECL cells and parietal cells. The increased acid secretion results in an increased duodenal acid load. This increased acid load results in progressive damage to the duodenal mucosa, resulting in the development of gastric metaplasia within the duodenal bulb. The infection then is able to colonize the patches of metaplasia within the duodenum. The combination of the increased duodenal acid load and the local damage to the duodenal mucosa by the infection eventually results in mucosal ulceration. Eradicating the infection reduces the acid load on the mucosa and removes any local effects of the bacterium. Consequently, eradicating *H. pylori* results in the long-term cure of the ulcer disease.
Duodenal ulcer patients have a nonatrophic antral predominant *H. pylori* gastritis. This stimulates the antral G cells to secrete excess gastrin, which circulates and stimulates the healthy body region of the stomach to secrete excess acid. The resulting increased duodenal acid load eventually leads to duodenal ulceration.

**HELICOBACTER PYLORI INFECTION AND GASTRIC ACID HYPOSECRETION**

*H. pylori* infection also can result in a pattern of *H. pylori* gastritis that is different from that seen in duodenal ulcer patients and that results in entirely different changes in gastric physiology. This different pattern of gastritis is seen in patients who develop gastric cancer or proximal gastric ulcers. It differs from that seen in the duodenal ulcer patients by having evidence of atrophy of the antral and the body mucosa. It also differs in that the inflammation extends into the acid-secreting body mucosa, resulting in a pangastritis or body-predominant gastritis. This atrophic pangastritis results in hyposecretion of gastric acid or complete achlorhydria. The mechanism by which this pattern of gastritis results in the hypochlorhydria is due to its effects on the function of the antral mucosa and the body mucosa.

**Influence of Helicobacter pylori-Induced Atrophy and Inflammation of the Body Mucosa on Gastric Function**

In subjects with *H. pylori*-induced inflammation and atrophy of the body mucosa, the ability to secrete acid in response to gastrin stimulation is reduced markedly or completely absent (Fig. 4) (Figure Not Available). Eradicating the infection in these subjects results in variable degrees of recovery of gastric acid secretion. This recovery of acid secretion coincides with resolution of the inflammation of the body mucosa with little evidence of any resolution of the atrophy. The recovery of acid secretion can occur within a few days of starting the anti-*H. pylori* therapy, and this corresponds with the timing of resolution of the polymorphonuclear cell component of the inflammation.

These observations suggest that the impaired function of the body mucosa is an effect of the acute inflammatory infiltrate or of something produced by the bacterium itself. The latter explanation seems unlikely because the density of colonization of the body mucosa in such subjects is considerably less than that in duodenal ulcer patients in whom gastric acid hyposecretion is not seen. The inflammation of the oxyntic mucosa seems the more likely explanation for its impaired function. Studies several years ago, before the recognition of *H. pylori* infection, had recognized that...
inflammation of the body mucosa in the absence of atrophy could inhibit gastric acid secretion. The inflammatory response induced by *H. pylori* results in increased local production of interleukin-1beta, which exerts powerful inhibitory effects on gastric acid secretion. In subjects with atrophic pangastritis, there is usually atrophy of the antrum as well as of the body mucosa. This atrophy of the antral mucosa results in depletion of gastrin-producing G cells, and this impairs the ability to secrete gastrin. The serum gastrin levels in *H. pylori*-infected subjects with or without evidence of atrophy of the antral mucosa were examined. In those with antral atrophy, the gastrin level is consistently lower than in those without atrophy when corrected for intragastric acidity (Fig. 5). The ability of the gastrin concentration to rise in response to secretion of gastrin pH was impaired.

![Figure 5](http://home.mdconsult.com/das/article/body/67257502-2/jorg=clinics&s...

It can be seen that in patients with an *H. pylori*-induced atrophic pangastritis, there are three separate factors producing gastric acid hypersecretion. The first is the loss of G cells because of atrophy of the antral mucosa, resulting in impaired gastrin response. The second is the loss of acid-secreting parietal cells because of the atrophy of the body mucosa. The third is the inflammation of the body mucosa impairing the functioning of the remaining parietal cells. Eradicating *H. pylori* infection results in early resolution of the acute inflammation of the body mucosa, and this is accompanied by varying degrees of recovery of acid secretion. The extent of recovery in acid secretion depends on the extent of the atrophy and irreversible loss of G cells and parietal cells.

**Spectrum of Changes in Gastric Morphology and Function Induced by Helicobacter pylori Infection**

*H. pylori* infection induces a spectrum of changes in gastric morphology and gastric function. At the one end of the spectrum, there is the nonatrophic antral-predominant, body-sparing *H. pylori* gastritis, that is characteristic of duodenal ulcer patients and that produces marked acid hypersecretion. At the other end of the spectrum is the atrophic pangastritis or body-predominant gastritis that is characteristic of gastric cancer patients and that induces marked acid hyposecretion. Most subjects with *H. pylori* infection fall somewhere between these two extremes with respect to gastric morphology and gastric function.

The fact that the morphologic pattern of gastritis determines the change in gastric function makes it essential that the pattern of gastritis is carefully documented in any study of the effect of *H. pylori* infection on gastric function. Early studies considered *H. pylori* infection to be a single entity and were unable to detect any association between the infection and changes in gastric acid secretion. This inability is explained by the fact that these studies included some patients with the pattern of *H.*
pylori gastritis that was reducing acid secretion and other patients with the pattern of gastritis that was increasing acid secretion, and these diverse effects were resulting in no change in mean acid output.

EFFECTS OF GASTRIC ACID SECRETION ON HELICOBACTER PYLORI GASTRITIS

It is now recognized that there is a two-way interaction occurring between H. pylori gastritis and gastric acid secretion. As discussed earlier, H. pylori gastritis exerts important effects on gastric acid secretory function. It has been recognized that acid secretion also can affect H. pylori gastritis, however.

The recognition that gastric acid secretion status modifies H. pylori gastritis is based mainly on observations after inhibition of acid secretion with proton-pump inhibitor therapy. Shortly after starting this treatment, there is a marked change in the distribution of the gastritis within the stomach. It changes from being antral predominant to becoming a pangastritis or a body-predominant gastritis. This redistribution of the gastritis mainly is due to the development of an increased inflammatory response to the bacterium within the body region of the stomach, rather than merely a redistribution of the infection itself. This situation is shown by the fact that the density of H. pylori colonization of the body mucosa in duodenal ulcer patients before omeprazole therapy is greater than it is with omeprazole. This redistribution of the gastritis mainly is due to the development of an increased inflammatory response to the bacterium within the body region of the stomach, rather than merely a redistribution of the infection itself. This situation is shown by the fact that the density of H. pylori colonization of the body mucosa in duodenal ulcer patients before omeprazole therapy is greater than it is with omeprazole.

The degree of inflammation of the body mucosa is much more marked on omeprazole, however. The secretion of acid by the body mucosa seems to prevent it from developing inflammation in response to the presence of H. pylori, and this protection is lost when the acid secretion is turned off. The mechanism by which acid secretion protects the body mucosa from developing gastritis is unclear. It may be that the secretion of acid simply flushes away toxic bacterial products, preventing them from reaching the mucosal surface. Alternatively the low local pH associated with high acid secretion may maintain the ammonia produced by H. pylori in the ionized form, which cannot penetrate the epithelial cells and induce toxic effects. Inhibition of acid secretion and elevation of local pH allow a proportion of the ammonia to remain unionized and able to exert toxic effects.

In addition to changing the distribution of the gastritis, profound inhibition of gastric acid secretion and elevation of intragastric pH reduces the density of colonization throughout the stomach. In a percentage of patients, inhibition of acid secretion results in eradication of the infection. H. pylori appears to have evolved in such a way as to allow it to thrive in a relatively acid environment, and a neutral environment is less hospitable. Profound inhibition of acid secretion allows other bacteria to colonize the stomach, and these compete with H. pylori for this location.

There is some evidence that proton-pump inhibitor therapy increases the tendency to develop atrophy of the body mucosa in H. pylori-infected subjects. The studies showing this have been criticized because of lack of adequate controls, however, and it remains unclear whether the omeprazole treatment merely is redistributing the occurrence of atrophy from the antrum to the body and not increasing the overall incidence of atrophy throughout the stomach.

IMPLICATIONS OF THE INTERACTION BETWEEN HELICOBACTER PYLORI INFECTION AND GASTRIC ACID SECRETION

The two-way interaction between H. pylori gastritis and gastric function is likely to be relevant to many clinically important issues, including the determination of the outcome of H. pylori infection, i.e., whether it induces duodenal ulceration or gastric cancer. The two-way interaction also is important in determining the efficacy of proton-pump inhibitor therapy and of the likelihood of rebound acid hypersecretion after discontinuation of the treatment.

Determinants of Outcome of Helicobacter pylori Infection

As discussed earlier, H. pylori infection can result in different patterns of gastritis, resulting in different disturbances of gastric physiology and different disease outcomes. The reason for these different responses and outcomes is unclear. The two-way interaction between H. pylori gastritis and gastric acid secretion is likely to be central to the evolution of the disease, however.
A subject's premorbid acid-secretory status may influence the outcome of *H. pylori* infection. It is well recognized that the level of gastric acid secretion in normal uninfected subjects varies considerably. A premorbid high acid output may protect a subject from developing body gastritis on contracting *H. pylori* infection. The high acid secretion results in the subject developing an antral-predominant, body-sparing gastritis. This pattern of gastritis results in increased antral gastrin release, and the gastrin stimulates the healthy uninfamed body region of the stomach to secrete excessive acid. Subjects with a premorbid high acid secretion develop even higher acid secretion on developing *H. pylori* infection and progress to develop duodenal ulceration.

Most studies have shown that eradication of *H. pylori* infection results in relatively little fall in the maximal acid output. This situation would support the high maximal output being the characteristic of that patient rather than merely secondary to the effect of *H. pylori*-induced hypergastrinemia on the parietal cell mass. Smoking and male sex are associated with high gastric acid secretion, and both of these are important risk factors for the development of duodenal ulcer disease in *H. pylori*-infected subjects. All these observations suggest that subjects with a high acid output because of gastric or environmental factors would develop a body-sparing gastritis, leading to a further increase in acid secretion and duodenal ulceration.

The propensity of a subject to develop atrophy would affect their gastric acid secretory function and the distribution of gastritis and outcome of the infection. If a subject initially develops a nonatrophic antral-predominant gastritis, the increased gastrin released by the antrum stimulates increased acid secretion, and this helps protect the body mucosa from developing. If for any reason the inflammation of the antrum leads to atrophy of the antral mucosa, this results in loss of the gastrin-producing antral G cells and a fall in the gastric level. This fall in gastrin results in a fall in acid secretion, and this allows the inflammation to extend up into the body mucosa. This is one process by which a subject may progress from an antral-predominant nonatrophic gastritis to an atrophic pangastritis. The propensity to develop atrophy of the antrum in the first place may be related to genetically determined response to the infection or lack of antioxidants in the diet, both of which are recognized to be associated with the development of atrophy and gastric cancer.

**Clinical Relevance of the Interaction Between Helicobacter pylori Gastritis and Acid Secretory Function During Proton-Pump Inhibitor Therapy**

The two-way interaction between *H. pylori* gastritis and acid secretion may explain the more profound inhibition of acid secretion occurring during proton-pump inhibitor therapy in *H. pylori*-positive subjects. The degree of elevation of intragastric pH is significantly higher during proton-pump inhibitor therapy in *H. pylori*-positive versus *H. pylori*-negative subjects. Further studies indicated that this was a direct effect of the infection because eradicating the infection lowered the intragastric pH during omeprazole therapy. Initially, it was proposed that the more marked elevation of intragastric pH was due to the ammonia produced by *H. pylori* urease activity neutralizing gastric acid. Studies in the authors' unit showed that a degree of inhibition of acid secretion by proton-pump inhibitor therapy was much more marked in *H. pylori*-positive versus *H. pylori*-negative subjects, however.

These studies indicated that the infection was accentuating the acid-inhibitory effect of the proton-pump inhibitor therapy.

The accentuated inhibition of acid secretion achieved by proton-pump inhibitors in *H. pylori*-infected subjects can be explained by the interaction between acid secretion and *H. pylori* gastritis. The inhibition of acid secretion in *H. pylori*-infected subjects results in the development of inflammation of the body mucosa. This inflammation illustrates acid secretion affecting *H. pylori* gastritis. Inflammation of the body mucosa impairs its ability to secrete acid, however. This situation exemplifies the effect of the gastritis on acid secretion. This development of body gastritis and the effect of body gastritis on acid secretion can explain the more profound inhibition of acid secretion in *H. pylori*-infected subjects during proton-pump inhibitor therapy. In *H. pylori*-negative subjects, there is only the pharmacologic effect of the drug inhibiting acid secretion. In *H. pylori*-positive subjects, there is the pharmacologic effect of the drug and the impairment of acid secretory function resulting from the body gastritis that develops because of the low acid secretion.

**Helicobacter pylori Status and Rebound Acid Hypersecretion After Proton-Pump Inhibitor Therapy**

It has been recognized that proton-pump inhibitor therapy results in marked rebound acid hypersecretion in *H. pylori*-negative but not *H. pylori*-positive subjects (Fig. 6). In *H. pylori*-negative subjects, the severity of the
rebound acid hypersecretion is related to the degree of elevation of intragastric pH and elevation of gastrin during proton-pump inhibitor therapy. The rebound acid hypersecretion is thought to be due to hypergastrinemia, resulting in ECL cell hyperplasia, which persists for several weeks after stopping the medication.

![Graph showing Basal Acid Output (mmol/l) for H. pylori Negative and Positive subjects.](image)

Figure 6. There is marked rebound basal acid hypersecretion following discontinuation of proton pump inhibitors in H. pylori negative but not positive subjects. The rebound acid hypersecretion is probably explained by the hypergastrinemia during proton pump inhibitor therapy producing ECL cell hyperplasia that persists for some time after stopping medication. The absence of rebound acid hypersecretion in the H. pylori positive subjects can be explained by the H. pylori body gastritis preventing acid hypersecretion.

The absence of rebound acid hypersecretion in H. pylori-positive subjects is surprising because these subjects have greater elevation of intragastric pH during proton-pump inhibitor therapy and a more marked rise in gastrin. Consequently, these subjects should develop a greater degree of ECL cell hyperplasia because of proton-pump inhibitor therapy, resulting in greater rebound acid hypersecretion. The absence of rebound acid hypersecretion in H. pylori-infected subjects is likely to be explained by the development of body gastritis during treatment and this gastritis persisting for some time after stopping the treatment, however. The body gastritis impairs the ability of the body mucosa to secrete acid and prevents its producing excess acid despite the development of ECL cell hyperplasia. This explanation for the absence of rebound acid hypersecretion in H. pylori-infected subjects suggests that eradication of H. pylori infection at the time of discontinuing proton-pump inhibitor therapy might result in severe rebound acid hypersecretion, and early data suggest that this is the case.

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