colegues themselves seem to rule out the hypothesis that gastric metaplasia in duodenal ulcer patients is secondary to the H pylori induced augment of gastric acid production with subsequent greater duodenal acidity. 3 As it is well known that H pylori infection is usually present in all patients bearing duodenal ulcer, all these patients should be acid hypersecretors, but this is true only in about one third of cases. 4 Moreover, several studies on the effect of both medical and surgical antisecretory measures have failed to show any reversal of gastric metaplasia in the duodenum. 5,6 Unfortunately, neither Khulusi et al nor Noach et al. reported data on the acid secretory patterns before and after H pylori eradication in the patients in whom they, respectively, showed regression or unchanged gastric metaplasia in duodenum. The knowl-

dge of this variable would have been of great help in interpreting the role of acid in the reversal or not of duodenal gastric metaplasia in the patients where H pylori has been eradicated. We recently studied the circadian pattern of gastric acidity in H pylori positive duodenal ulcer patients with (n = 24) and without (n = 14) gastric metaplasia in duodenum. 8 Although the acidity of both groups was significantly higher than normal, there was no significant difference between them as regards the mean (SD) pH values and 30-min (1-56 (0.35) e 1-44 (0.37)). These findings raise further doubts on the responsibility of hyperacidity in the induction of gastric type epithelium in duodenal mucosa and seem to support the conclusion by Khulusi et al. that factors other than acid, and possibly also the reduction of duodenal metaplasia, are implicated in the partial reversal of gastric metaplasia after eradication of H pylori.

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Editor—We are grateful for the comments of Savarino et al. The pathogenesis of gastric metaplasia is indeed a complex issue. Our paper (Gut 1995; 36: 193–7) and other work recently completed in our department suggests that H pylori is responsible for extending pre-existing gastric metaplasia, probably as a result of increased duodenal inflammation. The study by Noach et al. did show a trend towards reduction in gastric metaplasia with H pylori eradication but their small study size had insufficient power to detect a significant reduction. Hence their results cannot be considered to ‘contrast’ with our own although their interpretation does. We have recently studied the role of acid in the pathogenesis of gastric metaplasia. In a double blind placebo controlled trial we found that profound and prolonged acid suppression with omeprazole does reduce the extent of gastric metaplasia without affecting the severity of duodenitis. Taken together, it seems that H pylori extends gastric metaplasia by provoking duodenitis and that acid also extends gastric metaplasia but by a mechanism independent of inflammation.

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Helicobacter pylori and gastric metaplasia of the duodenum

Editor—Professor Northfield’s group (Gut 1995; 36: 193–7) found a significant decrease in gastric metaplasia in the duodenal bulb after Helicobacter pylori eradication. We have three questions:

(1) Duodenal biopsy specimens were examined by two histopathologists. Were they independent, did their results agree, and if not, whose were used?

(2) The extent of gastric metaplasia in the duodenal bulb biopsy specimens was assessed as a percentage of the total duodenal epithelial surface. How was this technique validated? What were the intra- and interobserver variation of these measurements?

(3) Why were six of 32 (19%) patients with duodenal ulcer without gastric metaplasia or duodenitis in the duodenal bulb?

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Reply

Editor—We thank Dr Baron’s group for their questions, in reply:

(1) The assessment of histological sections was carried out by two histopathologists unaware of the treatment status of the subject and working together on the same sections. The grading of gastric metaplasia and duo-
ndenitis was based on their mutually agreed values.

(2) The extent of gastric metaplasia in the duodenal bulb biopsy specimens was assessed as ‘total epithelial surface present’ in the biopsy sections themselves. We validated the results on 30 randomly selected sections by a morphometric technique using an interactive image analysis system (OsteoMeasure, Osteometrics, Atlanta, USA). There was a close correlation between the semiquantita-

tive values given in the paper and quantitative measurements (r = 0.89, p < 0.001).

(3) Duodenitis and gastric metaplasia occur in close proximity to duodenal ulcers. There are a few data on patchy distribution, however, and can be absent from biopsy specimens, especially if the samples are obtained from specific sites of the endoscopic appearance.

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