known of the possible role of *H pylori* in duodenal ulcer disease and the *H pylori* state of those patients cannot be determined retrospectively. It is possible that most subjects in our series were *H pylori* positive, which does not help to explain the reasons for this discrepancy.

By far the most intriguing question raised by Bianchi Porro’s study is whether resistance to standard anti-ulcer treatment can be the presence of *H pylori*. In Wagner’s study with bismuth subsaliclylate, 14% of duodenal ulcers did not heal despite *H pylori* clearance and 65% of healed ulcers had persistent *H pylori* infection, suggesting that this might not be the case.

Eradication by means of a more complete treatment regimen rather than mere clearance of the micro-organism may have a bearing on the higher relapse rate but can hardly account for the superior effects in the short term. A role for *H pylori* in some cases of refractory duodenal ulcers remains, however, an attractive hypothesis to which Professor Bianchi Porro gave prominence.

At the present time omeprazole is the anti-ulcer drug that provides the most striking results in the treatment of resistant duodenal ulcers. Its efficacy has generally been related to sustained and profound acid inhibition, but the drug is also known to exert a clearing effect on *H pylori*, if not to eradicate the micro-organism. Further studies are needed to discriminate between the roles of acid suppression and *H pylori* inhibition in the successful use of omeprazole for refractory duodenal ulcers.

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**EDITOR,—** Professor Bianchi Porro et al are to be congratulated on their interesting paper (Gut 1993; 34: 466–9). It is not surprising that only 14% of refractory duodenal ulcers were healed after four weeks’ treatment with sucralfate 4 g/day. Non-refractory ulcers require some weeks’ or up to 12 weeks’ treatment for healing. It is interesting that, in the two patients with unhealed ulcers after four weeks’ treatment with bismuth subcitrate plus amoxycillin and tinidazole, both healed with a further four weeks’ treatment with sucralfate. It is known that sucralfate has no direct action on Helicobacter pylori. *H pylori* however, cannot exist in the duodenal mucosa in the absence of gastric metaplasia. In the small study we reported in 19891 duodenal gastric metaplasia completely disappeared or became minimal in eight of 11 (73%) patients with healed duodenal ulcers after one year’s maintenance treatment with sucralfate 1 g twice daily. This compared with only five of 14 (34%) of patients who had been on one year’s maintenance with cimetidine. In the subsequent two years, two of five in the sucralfate group relapsed, compared with nine of 13 in the cimetidine group. In the absence of gastric metaplasia, no *H pylori* organisms were seen by light or electron microscopy in the duodenal mucosa. They were only very rarely seen when there was minimal gastric metaplasia.12 These findings would suggest that longterm maintenance treatment with sucralfate, by enhancing mucosal resistance to *H pylori*, may be an alternative method of eliminating the organism and reducing the relapse rate.1

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