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 negative, 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-H pylori treatment can be related to the presence of H pylori. In Wagner's study with bismuth subsalicylate, 14% of duodenal ulcers did not heal despite H pylori clearance and 65% of healed ulcers had persistent H pylori infection,7 suggesting that this might not be the case.

Eradication by means of a more complete treatment regimen rather than mere clearance of the microorganism may have a bearing on the rate of initial clearance and 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 gives shape.

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 suppression of acid inhibition, but the drug is also known to exert a clearing effect on H pylori, if not to eradicate the microorganism.7 Further studies are needed to discern the role 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 30% of duodenal ulcers were healed after four weeks' treatment with sucralfate 4 g/day. Non-refractory ulcers require six weeks' or up to 12 weeks' treatment for healing. It is interesting that, in the 10 patients with unhealed ulcers after four weeks' treatment with bismuth subcitrate plus amoxicillin 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, once 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, of the nine in the sucralfate relapse group, only one had a clear cut relapse. Thus, 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.14 These findings suggest that longer maintenance treatment with sucralfate, by enhancing mucosal resistance to H pylori, may be an alternative to eradication of the organism and reducing the relapse rate.15

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Helicobacter pylori positive resistant duodenal ulcers.

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