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-ulcer 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, 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 ulcer 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 points.

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 supposed and proven capacity for 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 discern the roles of acid suppression and H pylori infection in the successful use of omeprazole for refractory duodenal ulcers.

G MUSLANDI
Gastrointestinal Unit, S Raffaele Hospital, Via Olgiata 60, 20132 Milan, Italy


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 65% 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 two patients with unhealed ulcers after four weeks’ treatment with bismuth subsalicylate 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. It is possible that colonization with H pylori may be an alternative mechanism for maintaining the organism and reducing the relapse rate.

F ITOVEY
B A BAKER
P A JAYARAJ
Y CHU YIU
Department of Surgery, University College and Middlesex School of Medicine, University College London, London WC1E 6AU


EDITOR—Professor Bianchi Porro et al (Gut 1993; 34: 466-9) have shown that eradication of Helicobacter pylori in patients with duodenal ulcer disease resistant to H. pylori blockers provides effective healing with low relapse rates at one year. They also state that single drug treatment with colloidal bismuth subsalicylate or omeprazole heals at least 80% of these resistant ulcers within one year.

We describe a 30 year old man with an 11 year history of epigastric pain unresponsive to drug treatment. He smoked 20 cigarettes daily, and there was no history of ingestion of non-steroidal anti-inflammatory drugs or excessive alcohol intake. The three gastroscopies performed during this period reported either erosive duodenitis or ‘salt and pepper’ duodenitis. Duodenal brushings did not show any pathogens. Treatment regimens included colloidal bismuth citrate 120 mg four times daily, cinamidine 400 mg twice daily with sucralfate 1 g four times daily, ranitidine 150 mg twice daily, prezenepine 50 mg three times daily and omeprazole 60 mg once daily, but no pro-