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 would explain the higher healing rates compared with Bianchi Porro's experience. It is more probable that our patients were either a mixture of *H pylori* positive and negative with ulcers or mostly *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 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,[5] 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 subsequent 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's data certainly give support.

At the present time omeprazole is the anti-ulcer drug that provides the most striking results in the treatment of resistant duodenal ulcers.[6] Its efficacy has generally been related to sustained and powerful acid inhibition, but the drug is also known to exert a clearing effect on *H pylori*, if not to eradicate the micro-organism.[7] 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 six of 15 refractory 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 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 1989[1 2] duodenal gastric metaplasia completely disappeared or became minimal in eight of 11 (73%) patients with healed duodenal ulcers after one year's maintenance on 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 11 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 and they were only very rarely seen when there was minimal gastric metaplasia.[3 4]

These findings would suggest that longterm maintenance treatment with sucralfate, by enhancing mucosal resistance to *H pylori*, may be an alternative means of eliminating the organism and reducing the relapse rate.[5]

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EDITOR,—Professor Bianco Porro *et al* (*Gut* 1993; **34**: 466–9) have shown that eradication of *Helicobacter pylori* in patients with duodenal ulcer disease resistant to H_2 blockers provides effective healing with low relapse rates at one year. They also state that single drug treatment with colloidal bismuth subcitrate or omeprazole heals at least 80% of these resistant ulcers within four weeks.

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, cimetidine 400 mg twice daily with sucralfate 1 gm four times daily, ranitidine 150 mg twice daily, pirenzepine 50 mg three times daily and omeprazole 60 mg once daily, but none produced symptomatic relief. A further gastroscopy again reported 'salt and pepper' duodenitis while quadrantic antral biopsy specimens showed chronic inflammation and *H pylori* like organisms. He was then treated with colloidal bismuth citrate 120 mg four times daily for two months with amoxycillin 500 mg four times daily and metronidazole 400 mg three times daily for the initial two weeks. He had an excellent symptomatic response and remains asymptomatic one year after completing the course of treatment.

H pylori is probably a significant factor in the cause of erosive duodenitis in this patient. Symptomatic erosive duodenitis seems less responsive than duodenal ulcers to gastric acid inhibition.[1 2] Persistent duodenitis may also be a marker for early relapse of duodenal ulceration.[3] Eradication regimens for *H pylori* should be considered at an early stage of treatment of symptomatic erosive duodenitis.

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Reply

EDITOR,—Thank you for giving us the opportunity of replying to the comments on our article.

Dr Guslandi raises two important points in his letter: firstly, the lack of a group of refractory duodenal ulcers treated with CBS alone and secondly, the possibility of reasons other than anti-*H pylori* activity, for the superiority of triple therapy over sucralfate alone. We share his concern about the possibility that CBS alone may be superior to sucralfate in these patients and that it would have been interesting to include such a treatment group in the trial. Unfortunately, comparative trials between the two drugs in refractory duodenal ulcer patients are lacking, and only one controlled study comparing CBS with sucralfate in non-resistant patients exists to date.[1]

As far as the reasons for the high efficacy of the triple therapy in refractory duodenal ulcers are concerned, we feel that this is mainly because of its anti-*H pylori* activity. Indeed, the rate of healing was significantly higher in those patients where *H pylori* was eradicated after treatment than in those who had a persistent infection. This view is also supported by two recent trials in non-resistant duodenal ulcers, which report that adding antibiotics to an anti-ulcer regimen accelerates the healing of ulcers.[2 3] Therefore, in our opinion, an eradication regimen should be considered as the treatment of choice in the presence of an *H pylori* positive refractory duodenal ulcer, as it is capable not only of healing the anatomical lesion but also of changing its natural history. Tovey *et al* place great emphasis upon the shortness of initial acute treatment (four weeks) to explain the low healing activity of sucralfate alone, but we feel that in most of the existing trials on refractory duodenal ulcers the length of short term treatment was predetermined at four weeks as it is clear that prolonging the treatment (whatever drug is