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Tripotassium dicitrato bismuthate on unhealed duodenal ulcers

SIR,—I read with great interest the work of Bianchi Porro and coworkers.[1] In the light of the rapidly accumulating evidence of association between peptic ulcer disease and *Campylobacter pylori*[2-4] I was disappointed, however, not to find any mention of this. Comparable findings have been reported earlier[5] and raised the question whether *Campylobacter pylori* is responsible for those cases of peptic ulcer disease that tend to become chronic and recurrent. It would have been interesting to know whether the results of this study correlate with eradication of *Campylobacter pylori* from the stomachs of the patients.

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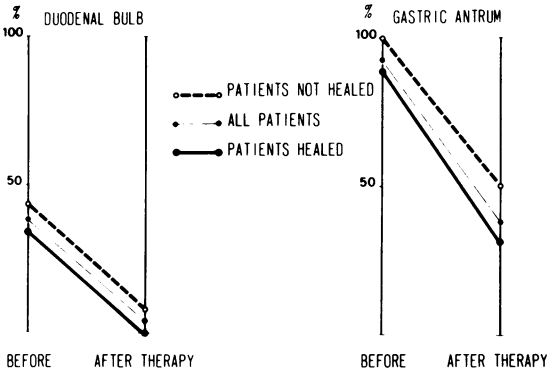
Reply

SIR,—We thank Dr Konikoff for his letter regarding our article (*Gut* 1987; **28:** 807–911). We agree with his interest to evaluate in future clinical trials, on duodenal ulcer resistant to H_2-blockers, the role of *Campylobacter pylori* (CP) colonisation in developing refractoriness. Unfortunately, this could not be done in our study, because when the trial was planned (August 1984), a reliable method to detect CP in our laboratory was not yet available.

Indirect information on this topic, however, can be drawn from our recent experience on the relationship between eradication of campylobacter from the antrum and the duodenal bulb and duodenal ulcer healing induced by tripotassium dicitrate bismuthate (TDB); 49 patients with active duodenal ulcer have been treated with DeNol 480 mg/d for four weeks; the presence of campylobacter in single biopsy specimens from duodenal bulb and gastric antrum was investigated by CLO test which has been documented to be an accurate predictor of the presence of CP;[1,2] the test was done on each patient at the initial and the follow up endoscopy.



Ninety two per cent and 37% of patients, respectively, had a positive antral and duodenal CLO test on study admission; after four weeks of treatment, the corresponding percentages of positivity were 37% and 2%, respectively. Evaluating the percentage of campylobacter eradication separately in healed and non-healed patients, we have not found any significant difference between the two groups. In fact, in non-healed ulcers antral CP positive specimens were 100% and 50% before and after treatment, while duodenal CP positive biopsies were 43% and 7%, respectively; the corresponding figures in healed ulcers were 89% and 31% for the antrum and 34% and 0% for the duodenal bulb (Figure). These findings suggest that the healing process, at least in responder duodenal ulcers, is not clearly related to