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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 earlier⁵ 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 H₂-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 dicitrato 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

CP eradication from the gastric antrum or the duodenal bulb.

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Bile acid malabsorption in progressive systemic sclerosis

SIR,—We have read with interest the paper by Stellaard and coworkers¹ on the increase of unconjugated serum bile acids in patients suffering from progressive systemic sclerosis (PSS). We have recently studied the serum pattern of primary bile acids in PSS-patients and the results have been partly reported on the journal of the Italian Association of Rheumatology² and at international symposia.^{3,4}

Our data confirm that a bile acid malabsorption is, in these patients, much more frequent than it is usually believed. We also think that malabsorption can not be caused only by bowel bacterial overgrowth. Very often the clinical picture of malabsorption is not reversed by wide spectrum antibiotics and in most patients steatorrhoea persists despite treatment.

In a group of 11 patients with 'classical' PSS (ARA criteria) we evaluated serum conjugated primary bile acids concentrations (by RIA) after an overnight fast and during three hour after a standard semiliquid meal (450 cal.). Postprandial curves were compared with 10 curves from sex and age matched healthy controls. Fasting serum concentrations of chenodeoxycholic acid conjugated (CDCA) were similar in the two groups [$1.76 (1.0) \mu\text{mol/l}$ in controls, $1.42 \pm 0.75 \mu\text{mol/l}$ in PSS], while the concentrations of cholic acid conjugated (CCA) were lower in the PSS-group [$0.67 (0.23) \nu 0.48 (0.17) \mu\text{mol/l}$, $p < 0.05$]. A similar pattern was exhibited by the postprandial increases postprandial maximal peaks of CDCA were almost normal in patients with PSS [$6.59 (2.18)$ in controls, $5.19 (2.99)$ in PSS], while CCA peaks were strongly reduced in PSS-patients [$3.36 (0.65) \nu 1.35 (0.44)$, $p < 0.001$].

Similarly, the area under the curve calculated for serum post-prandial concentrations of CDCA was not significantly different in the two groups, while for CCA concentrations, the area was significantly smaller in PSS-patients ($p < 0.01$).

In five patients we also studied the abdominal retention of $^{75}\text{SeHCAAT}$ (^{75}Se -homocholic acid taurine) at the fourth and the seventh day after the ingestion of a 370 kBq ($10 \mu\text{Ci}$) capsule (Amersham). PSS-patients exhibited strongly reduced $^{75}\text{SeHCAAT}$ abdominal retention (at seventh day: mean 5.53%, range 1-14) when compared with five sex and aged matched healthy controls (mean 23.30%, range 11.4-33). The results of $^{75}\text{SeHCAAT}$ -test were in good agreement with serum postprandial patterns of CCA.

Because it is well known that negligible bacterial deconjugation of SeHCAAT occurs *in vivo*, bowel bacterial overgrowth does not seem to be important in determining bile acid malabsorption in PSS.

Our data are consistent with a bile acid malabsorption of first type, very similar to that found to be related to Crohn's disease or to ileal resection⁵ and the severity of malabsorption may depend on the extent of ileal involvement in PPS.

In conclusion, we suggest that fasting and postprandial serum concentrations of CCA and $^{75}\text{SeHCAAT}$ abdominal retention test represent good markers of ileal disease in these patients. We therefore agree with Stellaard and coworkers, that further studies should be done to investigate the specific role of bacterial overgrowth in PSS, particularly with cholesteryl-glycine-1- ^{14}C breath test.

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