Correspondence

Raised serum concentrations of pancreatic enzymes in smokers

Sir,—We have read with interest the recent article by Dubick et al. about the enhanced pancreatic serum enzyme response after secretin in a control population of smokers compared with non-smokers. A rapid single injection of 75 CU secretin (Kabi Vitrum Sweden) resulted in increased serum enzyme concentrations of amylase, immunoreactive cationic trypsinogen and immunoreactive elastase 2 in 19 heavy smokers while none of these changes occurred in 13 non-smokers. These results are in part at variance with our experience and with some reports from the literature.3–4

The factors responsible for the serum enzyme response after secretin are ill defined.1,3 One of the main reasons for the controversies about the serum evocative tests in the literature over the last 30 years is the lack of a standardised study protocol and the lack of agreement about criteria of what is normal ('gold standard'). Variables like preparation and dose of secretin, selection of control population and of enzyme parameters may influence the test results in normals. In our recent study we investigated the serum evocative test in a control population of 19 smokers (defined as >20 cigarettes/day for >5 years; COHb: 3.5±2.3%), 19 non-smokers (no smoking for >3 years; COHb: 1.4±0.7%) and 10 patients with hypoxemia caused by chronic pulmonary disease (defined as pO2 <8.8 kPa).2 No participant had clinical or biochemical evidence for pancreatic disease (faecal chymotrypsin test >120 μg/g), hepatopathy, renal insufficiency, diabetes or excessive alcohol intake (>40 g/day). A bolus injection of 1 CU/kg secretin (secretolin® Hoechst) was given and serum enzyme concentrations of amylase, P-isoamylase, immunoreactive trypsin and immunoreactive lipase were determined before, 15 and 30 minutes after injection. Mean values of all enzymes were significantly higher after stimulation compared to basal levels in the three groups. In all groups, the enzyme response to secretin was more pronounced for trypsin and lipase than for amylase or P-isoamylase. A lack of serum enzyme response, however,—for example, no significant increase of serum immunoreactive cationic trypsinogen and immunoreactive lipase values was found in smokers in 26% and 36%, in non-smokers in 47% and 58% and in patients with hypoxemia in 10% and in 30%, respectively. Thus in our experience 'non-responders' are rather common in a normal control population for unknown reasons and the specificity of the evocative test for detection of advanced chronic pancreatitis is low (no serum enzyme response—immunoreactive cationic trypsinogen, lipase, P-isoamylase—after secretion or bombesin stimulation has been found typically in severe exocrine insufficiency5).

On the other hand, the results of Dubick et al.6 and Baldin et al.7 indicate that in normal non-smokers there is no enzyme response after secretin for immunoreactive cationic trypsinogen, P-isoamylase, and/or immunoreactive elastase-2 in contrast with the smoking group. This is at variance with results in non-smokers in our series with a marked increase of serum immunoreactive cationic trypsinogen and immunoreactive lipase in 53% and 42% respectively (with peak values of concentrations of immunoreactive cationic trypsinogen up to 7300 ng/ml and immunoreactive lipase up to 650 μg/l). Similar results have been reported previously, which indicate that no consistent postsecretin serum enzyme pattern for a normal non-smoking control population has been established.

In view of these controversies, the results of Dubick et al. are interesting but not convincing. Additional data are needed. According to Florholmen et al. no significant serum enzyme response (for immunoreactive cationic trypsinogen, P-isoamylase) is observed in non-smokers after a low dose secretin infusion (0.3 CU/kg/h) compared with high secretin doses (1.0 and 3.0 CU/kg/h) which regularly elicit a significant increase of serum immunoreactive cationic trypsinogen (and P-isoamylase with 3.0 CU/kg). In order to show more convincingly the influence of smoking on the serum enzyme response, the experiments of Dubick et al. should be repeated with graded doses of secretin, particularly with more physiological low doses.

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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 I was disappointed, however, not to find any mention of this. Comparable findings have been reported earlier2 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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References

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 DeNo1 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