LETTERS TO THE EDITOR

Sucralfate, bismuth subcitrate, and gastric HCO3 in humans

Sir,-The study by Shorrock et al1 shows a stimulatory effect of sucralfate on gastric bicarbonate secretion in humans. This confirms our previous findings published in 19852 in a paper unfortunately not quoted in Shorrock’s reference list. Yet the two studies somehow complement each other. Shorrock’s experiment was performed in acute conditions and in healthy volunteers, while our study was carried out in duodenal ulcer patients treated with sucralfate for six weeks. Basal gastric bicarbonate secretion, assessed by means of Feldman’s method,1 proved to be normal in all subjects before treatment and was significantly increased (p<0.01) at the end of the therapeutic course in both healed and unhealed patients.

The final assessment of gastric HCO3, was made 12 hours after the last dose of sucralfate. In Shorrock’s study the increase in alkaline secretion by sucralfate appears to be transient. Our data suggest either that the lack of effect of bismuth or that the lack of effect of bismuth subcitrate on gastric bicarbonate secretion in vivo is due to the method used, which may be not adequate suppression of bicarbonate secretion in human stomach. Our data suggest that the lack of effect of bismuth subcitrate on gastric bicarbonate secretion in vivo is due to the method used, which may be not adequate suppression of bicarbonate secretion in human stomach. Indeed, we have previously published3,4 studies which show a good correlation between infused and recovered bicarbonate in our perfusion system.5

We found that the dose of H, receptor antagonist used in our studies was adequate at keeping luminal pH between 6.4 and 7.1 when most of the gastric bicarbonate is in the free form and not as CO3-. The effect of PGE2 was not investigated in this study, having previously been shown not to stimulate bicarbonate secretion in the stomach.6

At concentrations of colloidal bismuth subcitrate (CBS) of 10 mg/ml we found no stimulation of gastric alkaline secretion (we have never studied effects on duodenal secretion in humans), which is clearly at variance with Dr Konturek’s comment.7 In a steady state perfusion system with a compound displaying negligible absorption, we have found that CBS adjunction to the bicarbonate so that the mucosa should remain more or less constant and is independent of perfusion rates. In our experiments we have tried to use concentrations of CBS which are likely to occur in the stomach after conventional oral doses of De-Nol. Higher concentrations are not only irrelevant but may in fact damage the mucosa, resulting in passive diffusion of bicarbonate as observed by us in vitro with carbamoxine.8

The conclusions of our study are therefore as stated.