LETTERS TO THE EDITOR

Postprandial mesenteric blood flow

Sir,—We read with interest the study by Sieber et al1 in which they used Doppler ultrasound to measure superior mesenteric arterial blood flow in response to liquid test meals given orally and intraduodenally (with and without atropine), and the additional effects of the infusion of combinations of cholecystokinin octapeptide, secretin, gastrin, glucagon, and, glucagon, cholecystokinin octapeptide. All occasions and hormones in mediating splanchic readings have only subjects this, support and No postprandial combinations of that fasting superior mesenteric arterial flow were examined of the intraduodenal test meal. Furthermore, however, that Lee et al2 using indocyanine dye chlormethyl showed that supraphysiological infusions of 10 and 20 ng/kg/minute glucagon caused no systemic haemodynamic or total hepatic blood flow changes in a group of alcoholic subjects. Azyny blood flow, however (and hence superior mesenteric flow), rose significantly in those with well compensated cirrhosis, suggesting that at supraphysiological doses (similar to those used by Sieber et al) glucagon may be a splanchnic vasodilator.2

(4) The authors state that blood pressure and pulse were monitored throughout the experiments but apart from incomplete data for the intraduodenal test meal with and without atropine, they present no information of blood pressure or pulse rate changes in response to any of their meals or infusion experiments. The blood and pulse pressure response to meals varies with both the age of the subject and meal composition, and these data should have been included or discussed in the current study.

(5) Atropine was shown to attenuate the postprandial hyperaemic response to the meal, suggesting that the cholinergic nervous system has a role in this change. The mechanism for the postprandial response is, however, likely to be multifactorial and may also involve β adrenergic3 and peptidergic mechanisms. Neurotensin4,5 is an intramural polypeptide,6 and calcitonin gene related peptide7 are known to be powerful splanchnic vasodilators and may also be involved in postprandial splanchnic vasodilatation.

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Reply

Sir,—Dr Wells raises important points about our recent publication,1 especially about the methodology used to assay superior mesenteric artery blood flow (SMABF). Several comments need clarification, however, so we would like to reply in the same order as given in the letter:

(1) We used a 3-5 MHz sector scanner for diameter measurements (and not a 3 MHz Doppler flowmeter as stated in the letter). This sector probe was combined with a pulsed Doppler flow meter (3-0 MHz). Diameters were presented as mean (SEM) cm units. The statement that we were reporting two decimal points is therefore rather misleading.

In our experience, the superior mesenteric artery has a circular anatomy which is an advantage compared with the portal vein where the determination of the cross sectional area of the vessel is complicated by an ellipsoidal vessel shape.

(2) We are puzzled by the statement that the increases in postprandial SMABF reported in our paper were far higher than in the published reports: similar measurements have been reported by others8 with comparable meals and caloric loads. On the other hand, the reports cited by Dr Wells are hardly comparable.

In one study, a new technique was employed to quantify SMABF (dye dilution). In a second, a lower caloric load was tested and in the study of Qamar8 only fasting SMABF was measured.

Nine subjects participated in this study. For all oral food experiments as well as the hormone studies, the same six volunteers were studied. For the experiments involving intraduodenal food application, 3 additional subjects were


Heliobacter pylori infection in healthy people

Sir,—We have recently published the results of an epidemiological study in Gut reporting discrepancies between active Heliobacter pylori (Hp) infection determined by means of the "C-urea breath test and the prevalence of anti-Hp antibodies in healthy volunteers. Further developments in serological tests make it necessary to report additional information and to reconsider our conclusions based on the serological data presented in the paper.

A systemic humoral immune response to H pylori has been searched for in many studies (including several serological tests), some of which have become commercially available. They have all in common that whole bacterial cells were primarily used as antigen (acid glycin extracts or sonicated bacterial cells, especially Campylobacter jejuni). False positive serological test results can therefore not be excluded. Thus new serological tests using purified high molecular outer membrane proteins of H pylori and urease as antigen were developed. These second generation serological tests may be more specific for H pylori infection.

We have investigated the sensitivity and specificity of several different serological tests in a population of patients in whom the presence or absence of H pylori infection was unequivocally established. These patients all had upper gastrointestinal tract endoscopy with antral mucosal biopsies that were used for microbiological Hp testing (Hemocult and a quick urease test (CLO test) and they all underwent a "C-urea breath test. Sera were used only from patients in whom all three tests were positive (H pylori infection present) or all three tests were negative (infection absent). These latter patients were also questioned about treatment with antibiotics within the past six months and included in the present analysis only if the response was negative. Sera from this population were tested for anti-Hp antibodies with our own enzyme linked immunosorbent assay ELISA and two commercially available, first generation serological tests (anti-Hp IgG EIA Roche, Hoffman-La Roche, Basel, Switzerland, and a secondgeneration test, only 7% have anti-Hp without active infection. The cumulative percentage of patients reacting with either one of the three first generation tests amounted to 29%, resulting in a specificity of only 71%. These findings support the hypothesis that a single test, serum antibodies in an important fraction of people, anti-Hp antibodies may be due to non-specific binding to the antigen in the test kit rather to a specific response to H pylori infection in the past. While it is still possible that healthy people eventually eliminate H pylori spontaneously, this conclusion may not be drawn from our results based on the first generation serological test that was used. Similar caution, however, should be used in the interpretation of virtually all studies that reported H pylori prevalence data based on first generation serological tests. Epidemiological studies designed to gather information on the prevalence of H pylori should preferentially use direct proof of infection rather than serology.

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Medical treatment of bleeding peptic ulcer: old drugs, new regimens

Sir,—Haemorrhage from peptic ulcer is due to the erosion of artery at the ulcer base by the continuous digestive action of pepsin and hydrochloric acid. Platelet plug and clot formation (both factors being pH sensitive) seal the bleeding artery. Dissolution of the clot is the most important factor for peptic ulcer bleeding. Intragastric acidity prolongs the duration of bleeding as the gastric juice contains fibrinolytic substances and a pH <7 results in inhibition of platelet aggregation and dissolution of the clot. Understandably then, attempts made with the acid " vs pepsin" or to inhibit fibrinolysis should result in stabilisation of the clot and prevention of reflooding. Yet to date the efficacy of none of the above mentioned drugs

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Sensitivity 100 93 94 97
Specificity 80 85 85 85 (96)* 93

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["H pylori test results were included that reacted slightly or strongly (specificity within parentheses).""]
Reply

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