LETTERS TO 
THE EDITOR

Postprandial mesenteric blood flow

Sir,—We read with interest the study by Sieber et al. 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 8, secretin, gastrin 17, and glucagon. We would like to raise several points.

(1) The authors used a 3 MHz Doppler flowmeter. Wavelength considerations with this frequency probe dictate that the best AP spatial resolution of the diameter measuring cursors is ± 1 mm (a best ‘guess’ of 0.5 mm can sometimes be made). The authors, however, have chosen to use the mesenteric artery diameter readings to two decimal points and claim to be able to show increases in the diameter of between 5 and 26% after food intake. Furthermore, it is not clear whether an AP and lateral cross section diameter reading was used or a longitudinal AP diameter reading was taken (in which case the superior mesenteric artery was assumed to be circular). It would have been better if the authors had quoted the mean and the range of the diameter reading.

(2) The quoted increase in superior mesenteric arterial blood flow in response to the oral test meal (180–360% above basal) is far greater than previous reports. It is not clear from the data whether the same subjects were studied on all occasions and compared—a total of nine subjects was enrolled in the study but data are only shown for six. If the same six subjects were not examined on each occasion, direct comparisons of all subjects’ data would not be reliable. This is further compounded by the clear differences in basal mean (SEM) flow rates between the experiments (data not all shown) of 443 (38) ml/minute before the oral test meal and 1178 (130) ml/minute before the intraduodenal test meal. What is the day to day coefficient of variation for the technique in their hands? Were all observations performed by one operator?

(3) The authors state that they infused glucagon, cholecystokinin octapeptide 8, secretin, and gastrin in doses designed to simulate postprandial circulating concentrations. No data, however, are presented to support this, and the authors cannot therefore be sure that physiological concentrations of these hormones were achieved. Furthermore, the dose of glucagon used (500 ng/kg/hour or 8-33 ng/kg/minute) is a supraphysiological dose and would result in plasma concentration much greater than those normally circulating postprandially. A dose of less than 3 ng/kg/minute would have been more appropriate. We cannot therefore agree with the authors’ contention that the hormones ‘are unlikely to be involved as blood borne hormones’ in mediating splanchnic vasodilation on the data presented but agree with the hypothesis.

We have recently shown (unpublished data) that fasting superior mesenteric arterial blood flow in six subjects measured by Doppler ultrasound decreased during physiological infusions of glucagon. A 1 ng/kg/minute infusion produced no detectable rise in basal glucagon concentrations (mean (SEM) basal glucagon values 144 (15) ng/l after 30 minutes 1 ng/kg/minute glucagon infusion resulted in a value of 129 (80) ng/l and a 3 ng/kg/minute glucagon infusion raised values to 214 (37) ng/l. Mean (SEM) fasting blood flow fell from 678 (97) ml/minute to 549 (88) ml/minute after 30 minutes of 1 ng/kg/minute glucagon infusion and to 453 (63) ml/minute after 20 minutes of a 3 ng/kg/minute infusion. There were no associated changes in cardiac output, stroke volume, blood pressure, pulse, or peripheral vascular resistance. This suggests that glucagon is a selective splanchnic vasodilator at physiologic concentrations and, indeed, is not involved in mediating the postprandial hyperaemia observed in previous studies. It is noteworthy, however, that Lee et al. using indocyanine dye clearance methods 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 anesthetic subjects. Arterial blood flow, however (and hence superior mesenteric blood flow), rose significantly in those without well compensated cirrhosis, suggesting that at supraphysiological doses (similar to those used by Sieber et al.) glucagon may be a splanchnic vasodilator.

(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 pulse and blood pressure response to meals varies with both the age and the subject and meal composition. Therefore these data should have been included or discussed in the current study.

(5) Atropine was shown to attenuate the postprandial hyperemic 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 adrenergic and peptidergic mechanisms. Neuropeptide Y, an intramural polyptide, and calcitonin gene related peptide are known to be powerful splanchnic vasodilators and may also be involved in postprandial splanchnic vasodilation.

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Reply

Sir,—Dr Wells raises important points about our recent publication, especially about the methodology used to assess 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 ultrasound 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 elliptoid 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: ‘Our values have been reported by others’ with comparable meals and caloric loads. On the other hand, the reports cited by Dr Wells are hardly comparable.

In one study, atropine technique was used to subtract SMABF (dye dilution). In a second, a lower caloric load was tested and in the study of Qamar, 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 participated. For the experiments involving intraduodenal application, three additional subjects were