Correspondence

Somatostatin on intravariceal pressure

SIR,—We read with interest the article on the effects of somatostatin on intravariceal pressure in patients with cirrhosis and portal hypertension (Kleber G, *et al*, *Gut* 1988; **29**: 153–6). We were not surprised, however, that the investigators failed to observe any change in intravariceal pressure or systemic haemodynamics 2.5 to six minutes after administration of a bolus dose of somatostatin. In our experience, the reduction in intravariceal pressure,¹ portal pressure¹ or wedged hepatic venous pressure (WHVP)² in patients with cirrhosis and portal hypertension occurs within five to 10 seconds after bolus administration of somatostatin or SMS 201–995. The reduction in intravariceal pressure or WHVP after bolus administration of somatostatin or SMS 201–995 is followed approximately 20–30 seconds later by a small but significant increase in arterial blood pressure and a bradycardia, suggesting that the systemic changes are secondary to the effects on splanchnic haemodynamics. Furthermore, the effects of a bolus dose of somatostatin and its analogue on splanchnic and hepatic haemodynamics are transient, the values returning to normal within two minutes after completion of the administration. Consequently, Kleber and his colleagues, by measuring intravariceal pressure 2.5 to six minutes after bolus administration of somatostatin would have missed any response. We find it surprising that knowing that the biological half life of somatostatin is very short, and if Fig. 1 is correct, each measurement of intravariceal pressure took approximately two minutes to record, the investigators did not leave the needle in the varix to continuously record the pressure after a bolus administration of somatostatin. Such a protocol, in addition to providing more informative data, would also have circumvented any problems which bleeding must have caused after removal of the needle from the varix.

The increase in intravariceal pressure after an infusion of somatostatin is less easy to explain. We have recently observed, however, that an infusion of somatostatin to rats with cirrhosis and portal hypertension produces an initial fall in portal pressure followed by a very marked reduction in extrahepatic shunting, presumably because of vasoconstriction of the collateral blood vessels themselves (unpublished observations). Vasoconstriction of the collateral blood vessels by somatostatin may explain the increase in variceal pressure following infusion of the hormone. Clearly, however, further work is necessary to fully elucidate the mechanisms of

somatostatin and its analogue SMS 201–995 on splanchnic haemodynamics and collateral blood flow in portal hypertension.

S A JENKINS AND R SHIELDS

*Dept of Surgery,
University of Liverpool,
PO Box 147,
Liverpool L69 3BX.*

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Reply

SIR,—Drs Jenkins and Shields pointed out that because of its short half life, the effects of somatostatin are of a shortlived nature.^{1,2} In principle, we agree with this observation. It is difficult, however, to detect such shortlived effects of somatostatin on variceal pressure in patients. We recorded the pressures at each single measurement as long as possible to get valid base line values. In the conscious patient during the procedure the needle may be accidentally displaced and continuous measurement after bolus injection cannot be guaranteed in every patient. We therefore decided to administer the drug after displacement of the needle and to puncture the varix a second time thereafter. This study design allowed us to record the second pressure only 2.5 to six minutes after drug administration.³ Thus, haemodynamic effects of rapid onset and short duration^{1,3} would have been missed. Indeed, for detection of very early effects one should try to administer the drug while the needle is still in the varix. It is questionable, however, whether shortlived pressure reductions are important for haemostasis. We feel that pressure reductions caused by somatostatin should be present for a longer duration in order to be relevant in the setting of acute variceal bleeding. Therefore, we also studied a group of patients in whom a constant infusion of somatostatin preceded by a bolus injection, a dosage regimen previously suggested,⁴ was administered. Also under these conditions no reductions in variceal pressure were observed. This may be because of raised cardiopulmonary pressures^{5,6} or vasoconstriction of the collateral vessels.

G KLEBER, T SAUERBRUCH, G FISCHER,
AND G PAUMGARTNER

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Sigmoid motility recording

SIR,—Drs Trotman and Misiewicz (*Gut* 1988; **29**: 218-22) show an improved technique of sigmoid motility recording. The recording tips were placed by colonoscope 25, 35, 45, and 55 cm and confirmed radiographically, so that the entire sigmoid was recorded, an improvement over older studies where the tips were placed by rigid sigmoidoscope. Also, the subjects were examined 18 hours after cleansing and colonoscopy so the effects of these disturbances could be minimised. It never seemed logical to attach much credence to studies on the lower part of the freshly prepared sigmoid and extrapolate the results to the rest of the sigmoid or colon.

Laudable though these improvements may be, however, little can be concluded from the study comparing pre- and postprandial mean maximal amplitude, per cent duration of activity and motility index in diverticular disease, irritable bowel syndrome and controls. First, the subjects are unequal in numbers (6, 20, and 13), age and sex. More importantly, we are given too little information on the patients. The six diverticular disease patients were said to have 'recent symptoms related to diverticular disease'. Does this mean they had peridiverticulitis or abscess? If not, in the light of studies which show the minority of patients with uncomplicated diverticular disease have symptoms and that these are features of the irritable bowel syndrome, how are these results to be interpreted?^{1,2}

Of the 20 IBS patients, eight had constipation,

four diarrhoea, seven alternating constipation and diarrhoea, and one was normal. We need to know more. Would all agree that these subjects had the irritable bowel syndrome, even the one with normal bowel habit? If so many would expect that those with diarrhoea would have a colon motility pattern distinct from those with constipation. The statement 'correlation between the various symptoms of the IBS and the motility index were not apparent' is not sufficient.

The correlation of symptoms to events in the gut is rarely achieved, and even then one cannot be sure whether the event is primary or secondary. Thus, studies that seek to define the physiological correlates of IBS symptoms must rigorously define the patients they are studying, stratify them for specific symptoms, and attempt to attach temporal relationships between the events and the symptoms. If we are to improve our understanding of the pathogenesis of IBS symptoms and diverticular disease, advances in technology must be accompanied by more sophisticated definition of the subjects studied.

W G THOMPSON

*Digestive Disease Research Group,
Division of Gastroenterology,
Ottawa Civic Hospital,
University of Ottawa,
1053 Carling Ave,
Ottawa, Ontario, Canada, K1Y 4E9.*

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Reply

SIR,—We thank Dr Thompson for his interest in our recent paper. As he rightly points out, this is the first systematic study of intraluminal pressure (IP) in the human sigmoid colon. Previously published reports claiming to show IPs measured in the sigmoid have, we believe, mostly come from recordings made only in the rectum. Our paper sets out to describe a reliable method of placing pressure sensitive catheters in the sigmoid colon and the results presented should be viewed as preliminary findings. From the numbers studied it is clearly not possible to make definitive statements about sigmoid motility in either diverticular disease or the irritable bowel syndrome. None the less, the results are particularly interesting because of the very high amplitude pressures that were found, with values many times higher than appears anywhere in the published literature pertaining to