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reduction in extrahepatic shunting, presumably because of vasoconstriction of the collateral blood vessels themselves (unpublished observations).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.*

References

- 1 Jenkins SA, Baxter JN, Corbett WA, Shields R. Effects of a somatostatin analogue on hepatic haemodynamics in the pig and on intravariceal pressure in man. *Br J Surg* 1985; **72**: 1009–12.
- 2 Jenkins SA, Baxter JN, Snowdon S, Whitehouse G, Shields R. The effects of somatostatin and SMS 201–995 on hepatic and systemic haemodynamics in patients with cirrhosis and portal hypertension [Abstract]. *Gut* 1988; **29**: A720.

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