adult human liver, and have since investigated expression in a number of other fetal tissues. We found evidence of expression in tongue, brain, intestine, mandible, eye, sternum, pancreas, spleen, and in placenta and umbilical cord, but not in adrenals, thymus, skin, or lung. High expression, however, was only observed in liver, intestine, and placenta. This pattern makes its unlikely that expression of the 6 Kbp HGF mRNA merely reflects haemopoiesis.

Finally, for completeness there are now two mRNA species for HGF, the most recently described being an alternatively spliced 1.5 Kbp transcript with an identical 5 prime cDNA sequence for the first 856 nucleotides downstream of the initiation codon but completely divergent at the 3 prime end. In summary, although the concept that only one growth factor is entirely responsible for liver regeneration is outmoded, the insights and interrelations of the HGF story seem likely to have an impact on our understanding of benign and perhaps malignant liver cell growth for many years to come.


8 Higuchi O, Nakamura T. Identification and change in the receptor for hepatocyte growth factor in rat liver after partial hepatectomy or induced hepatitis. Biochem Biophys Res Commun 1991; 176: 599-607.


reduced antral pressure waves and increased pyloric pressure waves observed in diabetic patients with symptomatic gastroparesis, but we agree that this hypothesis requires confirmation.

The mechanisms by which hyperglycaemia influences gastric motility are uncertain and may be indirect and multifactorial. A recent study performed by our group suggests that hyperinsulinaemia may not influence antpyloric motility.1


Prosp ective clinical and manometric study comparing pneumatic dilation and sublingual nifedipine in the treatment of oesophageal achalasia

Sir,—Dr Coccia and colleagues (Gut 1991; 32: 604-6) conclude that sublingual nifedipine is as good a treatment as pneumatic dilation of the gastrooesophageal sphincter in patients with stage I or II oesophageal achalasia. This conclusion is based on manometric investigations as well as clinical evaluation. Regarding the manometric study it is difficult to see whether the manometric technique is sufficient as the diameter of the pressure probe and their normal values are not stated. Further, it might be assumed that the tube is in a fixed position during the nifedipine treatment. If that is the case how did the authors make sure that it was maximal sphincter pressure they measured?

There is no mention of the relaxation of the sphincter in relation to swallowing and as achalasia means lack of relaxation that important parameter is missing. Also there was no information about the peristaltic response of the oesophageal body.

From a clinical point of view there is obviously no difference in the two treatment groups, but the possibility of a type II error is not calculated and these patients were only followed for a short period of time. It is important to know whether the stage I or II of achalasia progresses under the treatment with nifedipine or after dilation.

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Reply

Sir,—As are the patients their own controls, the diameter of the probe should be irrelevant. The probe diameter is 4/7 mm, however, and the values recorded in normal subjects are lower: oesophageal sphincter pressure is 19 (4-5) mm Hg (×SD) and pressure waves amplitude is 80 (15) mm Hg. The probe position was controlled periodically during the examination to make certain that it measured the maximal lower oesophageal sphincter pressure.

The question about lower oesophageal sphincter relaxation and return of peristalsis is very interesting. In fact, in some cases peristalsis returned and the postdeglutitive relaxation of the lower oesophageal sphincter was improved. This is however, the subject of other research which is in progress.

The absence of differences between the two treatment groups is not limited to a clinical point of view, but is also based on radiologic and manometric criteria (see Methods and Results sections). The duration of the follow up period is between that of the Traube (Gastroenterology 1986; 90: 1670) and to Gelfond (Gastroenterology 1982; 83: 963) and the improvement observed at the final control is against a possible progression of the disease.

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M BORTOLOTTI I Medical Clinic, University of Bologna, Bologna, Italy

European Workshop on Therapeutic Digestive Endoscopy

The Xth European workshop on therapeutic digestive endoscopy will take place at Erasme Hospital, ULB, Brussels, Belgium from June 16 to 18 1992. For further information please contact André Van Gossum, MD, Gastroenterology Department, Erasme Hospital, Route de Lennik 808, B-1070, Brussels (Phone: 32 2 555 37 12; fax: 32 2 555 46 97).

European Pancreatic Club

The XXIV Meeting will be held in Vienna, 25–29 August 1992. For information contact the Secretary of EASL, Dr P L M Jansen, Academic Medical Center, Meibergdreef 9, 1105 AZ Amsterdam, The Netherlands (Fax: 31 20 691 7033).

The European Association for the Study of the Liver (EASL)

The annual meeting will be held in Vienna, 25–29 August 1992. For information contact the Secretary of EASL, Dr P L M Jansen, Academic Medical Center, Meibergdreef 9, 1105 AZ Amsterdam, The Netherlands (Fax: 31 20 691 7033).

Study Day on Management of Pancreatic Cancer

This will be held on 24 March 1992 at the University of Southampton. Further details from Mrs J Daniels, University Surgical Unit,