

reduced antral pressure waves¹² and increased pyloric pressure waves¹³ 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 does not influence antropyloric motility.¹⁴

R FRASER
M HOROWITZ
J DENT
Gastroenterology Unit and University
Department of Medicine,
Royal Adelaide Hospital,
Adelaide, South Australia, 5000

- Phillips WT, Schwartz JG, McMahan CA. Rapid gastric emptying in patients with early non-insulin-dependent diabetes mellitus. *N Engl J Med* 1991; 324: 130-1.
- Hedde R, Collins PJ, Dent J, Horowitz M, Read NW, Chatterton BE, et al. Gastric and oesophageal emptying in insulin-dependent diabetes mellitus. *J Gastroenterol Hepatol* 1986; 1: 97-113.
- Horowitz M, Maddox AF, Wishart J, Harding PE, Chatterton BE. Relationships between oesophageal transit and solid and liquid gastric emptying in diabetes mellitus. *Eur J Nucl Med* 1991; 18: 229-34.
- Kesharvarian A, Iber FL, Vaeth J. Gastric emptying in patients with insulin requiring diabetes mellitus. *Am J Gastroenterol* 1987; 82: 29-35.
- Wegener M, Borsch G, Schaffstein J, Luerweg C, Leverkus F. Gastrointestinal transit in patients with insulin-treated diabetes mellitus. *Dig Dis* 1990; 8: 23-36.
- Oliveira RB, Troncon LEA, Meneghelli UG, Dantas RO, Godoy RA. Gastric accommodation to distension and gastric emptying in diabetics with neuropathy. *Braz J Med Biol Res* 1984; 17: 49-55.
- Fraser R, Horowitz M, Maddox A, Harding P, Chatterton B, Dent J. Hyperglycaemia slows gastric emptying in type I diabetes mellitus. *Diabetologia* 1990; 33: 675-80.
- MacGregor I, Gueller R, Watts H, Meyer J. The effects of hyperglycaemia on gastric emptying in man. *Gastroenterology* 1976; 70: 190-6.
- Morgan LM, Tredger JA, Hampton SM, French AP, Peake JC, Marks V. The effect of dietary modification and hyperglycaemia on gastric emptying and gastric inhibitory polypeptide (GIP) secretion. *Br J Nutr* 1988; 60: 29-37.
- Fraser R, Horowitz M, Dent J. Hyperglycaemia stimulates pyloric motility in normal subjects. *Gut* 1991; 32: 475-8.
- Hedde R, Collins PJ, Dent J, Horowitz M, Read NW, Chatterton BE, Houghton LA. Motor mechanisms associated with slowing of the gastric emptying of a solid meal by an intraduodenal lipid infusion. *J Gastroenterol Hepatol* 1989; 4: 437-47.
- Camilleri M, Malagelada JR. Abnormal intestinal motility in diabetics with the gastroparesis syndrome. *Eur J Clin Invest* 1984; 13: 420-7.
- Mearin F, Camilleri M, Malagelada JR. Pyloric dysfunction in diabetics with recurrent nausea and vomiting. *Gastroenterology* 1986; 90: 1919-25.
- Fraser R, Fuller J, Horowitz M, Dent J. Effect of hypoglycaemia on pyloric motility. *Clin Sci* 1991; 81: 281-5.

Prospective clinical and manometric study comparing pneumatic dilatation 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 dilatation 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 dilatation.

L WALLIN
S BOESBY
Dept of Surgery and Gastroenterology D,
KAS Glostrup,
DK - Glostrup,
Denmark

Reply

SIR,—As the patients are 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=19 (4.5) mm Hg (×SD) and pressure waves amplitude=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 our follow up period is between that of Traube (*Gastroenterology* 1986; 90: 1670) and of Gelfond (*Gastroenterology* 1982; 83: 963) and the improvement observed at the final control is against a possible progression of the disease.

G COCCIA
Gastroenterology Dept,
Osp. Galliera, Genova, Italy
M BORTOLOTTI
I Medical Clinic,
University of Bologna,
Bologna, Italy

NOTES

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,

F Level Centre Block, Southampton General Hospital, Tremona Road, Southampton SO9 4XY (Tel: 0703 796144; fax: 0703 794020).

International Association for the Study of the Liver - Biennial Scientific Meeting

The Biennial Scientific Meeting of the International Association for the Study of the Liver will be held in Brighton from 3-6 June 1992. For further information please contact the IASL Conference Secretariat, 145 Islingword Road, Brighton, Sussex BN2 2SH, UK (Tel: (0) 273 623123; fax: (0) 273 622944).

C A Ewald Prize

The German Gastroenterology Society (Deutsche Gesellschaft für Verdauungs- und Stoffwechselkrankheiten) announces the C A Ewald Prize for outstanding scientific work on the topic 'Pathogenesis of peptic ulcer'. The applicant's work may be unpublished or published in 1990 to 1991 and should be written in German or English. The applicant is asked to submit five copies of his application as follows: (1) Scientific work, (2) Curriculum vitae, (3) List of all previous publications.

The C A Ewald Prize is sponsored by the Cascan Company, Wiesbaden, and amounts to 10 000 DM. The prize can be awarded only to applicants not older than 40 years. Applications should be sent not later than 30 April 1992 to: Professor Dr M Manns, Med. Hochschule Hannover, Abt Gastroenterologie, Konstanty-Gutschow-Str 8, 3000 Hannover 61.

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 from 11-14 October 1992 at Ulm, Germany. For further information please contact the EPC Scientific Secretariat, Mrs M Wild, Department of General Surgery, University of Ulm, Steinhövelstrasse 9, 7900 Ulm, Germany (Phone +731/1 79-2200, 2201; fax +731/179-2466).

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).