Omeprazole in H2 blocker non-responders

Sir,—The results of the study by Delchier et al. on the similar effectiveness of omeprazole 20 mg mane and ranitidine 150 mg twice daily in H2 receptor blocker non-responders are very interesting, but also the comments by Bate1 on this paper are important. We fully agree with Bate’s opinion that a six week trial of an omeprazole 20 mg should be judged sufficient to define resistance to H2 blockers, because ulcer healing rates further increase by continuing therapy with these drugs to eight weeks.2 It must also be emphasised that the adoption of standardised definitions of ulcer refractoriness continues to generate confusion in this field and prevents a useful comparability of findings pertaining to different studies.

Even though Delchier and colleagues adopted patient selection criteria which may have greatly influenced their final results, it is worth pointing out that the reduced efficacy of omeprazole in their trial is a relevant factor in determining the lack of significant difference between this drug and ranitidine in healing resistant ulcers. As the authors discussed in their paper, the well known variability of individual response to single daily doses of omeprazole 20 mg3 may be the most reasonable explanation for the low efficacy of this dosage regimen in their study compared with the impressive one obtained in other trials, which tested single daily doses of omeprazole 40 mg.4 Some of our recent data seem to sustain their supposition. We used 24 hour continuous pH-metry7 to study two patients with endoscopically proven duodenal ulcers on the fifth day of treatment with omeprazole 20 mg mane. As reported in the Figure, the circadian profile of gastric acidity of both patients resulted poorly influenced by the drug. These findings show that the antisecretory effect of omeprazole 20 mg is very low in some subjects and the variability in acid suppression with this dosage can be even higher than previously reported.5 The reasons for this are at present unclear, but a derangement in the pharmacokinetic pathways of the drug might be involved.6 As regards patients’ compliance, we could check daily drug intake because they were hospitalised.

On the basis of our data, it seems advisable to take into consideration the authors’ suggestions that omeprazole 40 mg is probably the optimal dosage for treating H2 blocker non-responders and that 24 hour pH monitoring could be valid for verifying whether the clinically recommended dose of omeprazole 20 mg in duodenal ulcer disease,7 is really appropriate in individual patients.

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Figure: 24 hour gastric acidity profiles of two duodenal ulcer patients on the fifth day of treatment with omeprazole 20 mg mane. (D= dinner, B= breakfast, L= lunch.)
n numerous calculi and showing fibrous thickening of their walls. The ducts were lined by normal and mildly to severely dysplastic biliary epithelium. Severe dysplasia was characterised by columnar, angulated, stratified pleomorphic nuclei with prominent nucleoli, and a papillary surface (Figure). No invasive carcinoma was identified.

In the discussion the author refers to our article ‘IgA class antibody against human jejunum in sera of children with dermatitis herpetiformis’ (J Invest Dermatol 1986; 87: 703–6), as follows: ‘...type of reticulin antibodies reacting with human liver and spleen has also been described previously and already Seah et al and Eterman et al showed that such antibodies can react with human jejunum, a finding recently confirmed also by Karpáti et al.’ Here we described for the first time IgA type antibodies binding to human jejunum and that they may be related to reticulin antibodies.

An IgG type reticulin antibody reacting with human small bowel was seen by Eterman et al. ‘...the IgG type of reticulin antibodies were reported of low frequencies (18–46%) and low specificity (75–85%) in coeliac disease. In contrast, IgG class antibodies seem to be more sensitive and specific.’ (From the introduction of Dr Hallström’s paper.)

Jejunal antibodies have distinctive characteristic signs compared with other IgA type reticulin antibodies: they bind to the small bowel, which is the damaged organ in gluten sensitive enteropathy and they bind at the site of gluten absorption which is the precursor of the disease. In addition, the binding site of IgA type jejunal antibody corresponds to or is very similar to the extracellular IgA deposition detected in the diseased jejenum of patients with gluten sensitive enteropathy. Because of the damaged structure of jejunal jejunum, this similarity can be ascertained by investigating the diseased small bowel of patients with almost normal villous structure: (a) in jejunal biopsy samples taken several hours to one to two days after gluten challenge in coeliac patients who have recovered on gluten free diet; (b) we found IgA deposits in the small bowel of dermatitis herpetiformis patients with almost normal jejunal structure.

In the present work Dr Hallstrom found both the endomysium, and IgA type reticulin antibodies to be very important in the diagnosis of gluten sensitive enteropathy, and by absorption studies the endomysium antibody (substratum of endomysium) was related to the human subtype of reticulin antibodies and was distinguished from that of rat subtype. We think that if antihuman antibodies are important in considering the pathogenesis of coeliac disease, they must be related to the IgA type antibodies reacting with human jejunum.

We conclude that one of the reticulin antibodies category mentioned does not correspond to the pathological concept of the IgA type jejunal antibodies supplied in our study. S KARPÁTI* I ROSNÁI E TOROK* *Heim Pál Hospital for Children, Budapest, Hungary 1*Medical-Laboratory Clinic of University-Municip. FRG (supported by the Humboldt-Foundation, Bonn, FRG) 2*Etyep Hospital for Children, Budapest, Hungary 3000 M.2 Fővám körút 9–11, FRG


**BOOK REVIEWS**


**Common problems in gastrointestinal surgery.** One of a series produced by Year Book Medical Publishers on a variety of surgical subjects. The contribution in question is edited by Joseph Fischer, chairman of surgery at the University of Cincinnati. The approach is refreshing and novel. Each chapter is introduced by a specific clinical problem: four to eight line case history, one or more consultant is then asked to comment. Most contributors are pithy and to the point. Their comment usually consists of a brief overview of the literature, some reference to pathophysiology followed by the contributors own view on management. The book is largely, I suspect, designed to assist the private practitioner in North America to provide optimum clinical management based upon the views of experienced clinicians. The layout, diagrams and artwork are pleasing. Only key references are provided. The contributors: 66 in all are household names in GI circles, a few have been retired from practice for a variable time but most are regarded as contemporary experts in their disciplines. Only three are not from the USA (two from the UK and one from Canada). The reader must therefore expect a US bias to the text. Surprisingly the section on oesophageal and thoracic problems does not include any contribution on oesophageal carcinoma which some will find surprising with the development of endoscopic endoluminal ultrasonography, the growing recognition of early oesophageal cancer and the impact of low morbidity bypass, intubation and laser therapy on palliative therapy. I find it curious to come across two breast problems in the thoracic section.

The gastrooduodenal section includes a single contribution on GI bleeding. The emphasis, as is prevalent throughout the book, is on surgical treatment without even reference to endoscopic assessment or the role of endoscopic therapy. The medical/surgical divide is a real one in North America and the concept of joint management is not one that flavours this book.

The hepatoiliary section is varied and interesting, but it is difficult to do justice to all that has occurred over the last decade in liver transplantation by reference to a single case report.

The endocrine section makes interesting reading, but the gastrointestinal component of many case reports is enigmatic.

There is some unfortunate duplication in the colorectal section particularly with reference to diverticular disease and regional enteritis. The important clinical problem of major colonic haemorrhage takes no account of rapid bowel preparation and therapeutic endoscopy or the impact of intraoperative panendoscopy on surgical strategy.

This is a bold and attractive approach to a surgical update. In gastroenterology it must include joint management with gastroenterologists. The experiment has been a good one.