LETTERS TO THE EDITOR

When is a coeliac a coeliac?

EDITOR,—We read with interest the Science Alert comment by Mäki (*Gut* 1997;**41**:565–6) on Dieterich *et al*'s paper[1] identifying tissue glutamine (tTG) as the antigen for endomysial antibody (EMA). Unfortunately, Dr Mäki's comments were somewhat speculative and severely biased towards his own view that gliadin somehow (but how?!) reveals neoepitopes which, by inducing antibodies to connective tissue, apparently provide the key to the central pathogenic mechanism for gluten sensitivity. It is hardly useful to read that ". . . coeliac disease is indeed self-perpetuating and irreversible if the environmental trigger, gliadin, is not removed . . .": that information has been around since Dicke's era.

That there have been exciting findings from Sollid and colleagues from Oslo regarding the in vitro response of cloned (CD4+) mucosal T lymphocytes to gliadin and its derivative peptides with the production of interferon γ and other Th1-type cytokines,[2] seems to have escaped Dr Mäki's pen.

Moreover, it seems certain that, over the next few years, the Oslo group is set to define the qualitative T lymphocyte responses underlying mucosal damage in gluten sensitivity, and the gliadin peptides which evoke such changes. It is important to stress that these experiments underpin the drift of clinical research over the years which again has led to the inevitable conclusion that gluten sensitivity depends on T lymphocyte responses and not on B (humoral) immunology.[3 4] That gluten sensitivity with all its clinical and immunopathological findings can occur without demonstrable antibody[5] should amply inform Dr Mäki (and others) that a theory of pathogenesis for gluten sensitivity, based solely on antibodies, will not do[5]; that idea has already been dismissed by others.[6 7]

More importantly, at present there is no discussion in the literature about EMA negative patients. It is important to avoid a self-fulfilling prophecy—that is, taking biopsy samples only from EMA positive individuals. A recent editorial (*Lancet* 1991;**337**:590) notes the disparity between diagnosis and serology. In most studies, the sensitivity of serological markers has been evaluated in terms of severe (flat) mucosal lesions, or alternatively, a biopsy had only been performed when serological markers were positive.[8–10]

In contrast, we showed when using tTG that sensitivities and specificities for a subgroup of patients fulfilling the ESPGAN criteria with partial villous atrophy at presentation, initially tested by the Berlin group (Dieterich, Schuppan), gave disappointing values of 44% and 88% respectively.

Again, in two independent, prospectively studied groups of coeliac patients,[11 12] the overall sensitivity and specificity of EMA was 50%, and 90–95% respectively. Clearly, EMA is not exclusively positive in every gluten sensitised individual. However, when EMA positivity is related to the severity of the proximal mucosal biopsy, then sensitivity for EMA is about 90% for total villous atrophy, but only 30% for the milder infiltrative-hyperplastic lesions with partial villous atrophy.[13] Thus whether the EMA test is positive or not depends entirely on the presence of a severe lesion and possibly on the length of intestine involved. This point needs to be remembered in population studies, especially when a flat, severe lesion is taken as sole manifestation of coeliac disease.

Much more needs to be learned about effective screening for gluten sensitised individuals. Endomysial antibodies alone fail to predict all such cases and clearly, therefore, do not constitute the universal panacea for this disease as Dr Mäki wants us to believe. Gluten sensitivity is not due exclusively to endomysial antibody production.

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Gastric bacterial overgrowth is a cause of false positive diagnosis of *Helicobacter pylori* infection using ^{13}C urea breath test

EDITOR,—We read with interest the paper by Dominguez-Munos *et al* (*Gut* 1997;**40**:459–62) describing an optimal test drink in the ^{13}C-urea breath test (^{13}C UBT) for the diagnosis of *Helicobacter pylori* infection. In this study all *H pylori* negative subjects (adults with dyspeptic symptoms) had a negative result with the ^{13}C UBT (specificity 100%) after different meals. In other studies, using ^{13}C UBT to document *H pylori* infection both in adults and children, the sensitivity of the test ranged from 92 to 100% whereas specificity was usually above 92%.[1 2] However, no explanation has been given for the occurrence of false positive tests. Methodological bias and problems in defining the cut off value are possible reasons. However, there are no explanations for some false positive tests.[3 4] Here, we report two children with a positive ^{13}C UBT resulting from the presence of urease positive bacteria other than *H pylori* in the stomach.

A 14 month old girl operated on just after birth for a congenital diaphragmatic hernia and presenting with severe gastro-oesophageal reflux associated with oesophageal dilatation and swallowing dysfunction was referred because of gastro-oesophageal haemorrhage. Endoscopy revealed oesophageal dilatation, severe oesophagitis and gastric stasis. The gastric and duodenal mucosa appeared normal. She was treated for two months with H_2 receptor antagonists. Antral and fundal biopsy samples (n=5) showed mild gastritis and were *H pylori* negative on histology (Giemsa staining). Direct examination and culture of gastric biopsy specimens were both negative for *H pylori*. Serum specific antibodies against *H pylori* (ELISA) were also negative. ^{13}C UBT was abnormal (5.63 $\delta\%_o$; normal values <3 $\delta\%_o$). Culture of gastric secretions revealed gastric bacterial overgrowth with colonic bacteria known to have urease activity (that is, *Proteus mirabilis*).

An 8 year old boy operated on just after birth for gastroschisis was referred because of a six month history of abdominal pain. Physical examination was normal. Endoscopy revealed moderate gastric stasis. Examination and culture of both antral and fundic biopsy specimens (n=5) were negative for *H pylori* as were serum specific antibodies against *H pylori* (ELISA). ^{13}C UBT was slightly abnormal (3.25 $\delta\%_o$, normal values <3 $\delta\%_o$). Culture of gastric secretions revealed gastric bacterial overgrowth with species, including micrococcus, with urease activity.

These two cases demonstrate that hydrolysis of urea as a result of bacterial metabolism can occur in the stomach of *H pylori* negative subjects, and that ^{13}C-urea can be hydrolysed in the presence of urease from bacterial species other than *H pylori*. Several bacteria—for example, *P mirabilis*, *Escherichia coli, Yersinia enterocolita*, *Klebsiella pneumoniae, Staphylococcus aureus*, have urease activity, but they do not usually colonise the stomach. Gastric bacterial overgrowth was probably favoured by prolonged antisecretory treatment in the first case and by gastric emptying abnormalities in the second (intestinal malrotation associated with gastroschisis). Urease activity associated with *H pylori* infection usually causes greater excretion of ^{13}C than that observed in our two patients (5.6 and 3.25 $\delta\%_o$ respectively). As the cut off value of 3.00 $\delta\%_o$ has been validated in both adults and children[2 3] and no technical bias occurred, false positive resuts can be ruled out in our patients.

In summary, the ^{13}C UBT is a sensitive and specific method for the non-invasive detection of *H pylori* infection, but gastric bacterial overgrowth may lead to a false positive

diagnosis. These patients may be wrongly considered to be *H pylori* positive if a single, non-invasive test is used. In some circumstances (long term use of antisecretory drugs or abnormalities of gastric motility) a low positive ^{13}C UBT without other evidence of *H pylori* infection (serology, bacteriology, histology) may be suggestive of gastric bacterial overgrowth.

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The Maastricht Consensus Report

Treating young dyspeptic patients

EDITOR,—The Maastricht Consensus Report (*Gut* 1997;**41**:8–13) is a welcome benchmark summarising current opinion and scientific evidence regarding the role of *Helicobacter pylori* in gastroduodenal disorders. Whereas the management of peptic ulcer disease is no longer controversial and is very evidence-based the same is not yet true for the syndrome of non-ulcer dyspepsia and the management of the uninvestigated dyspeptic patient. The recommendation of the Maastricht Report reflects this uncertainty. They recommend that at the specialist level, eradication therapy for *H pylori* infected non-ulcer dyspepsia is "advisable", based on supportive scientific evidence, but only after "full investigation" including endoscopy, ultrasound and other tests. However, in the management algorithm for the uninvestigated dyspeptic in primary care, non-invasive testing (with a breath test) and treatment is recommended for patients who are at a low risk of gastric carcinoma. Why such a difference? If it is recommended that a breath test is investigation enough of dyspepsia in primary care then an endoscopy and biopsy should be adequate in specialist practice if there are no other clinical indicators of another diagnosis (such as biliary colic) and the patient is at low risk of malignancy. The difficulty is that non-ulcer dyspepsia will remain a hard target and even several studies of symptom response after eradication therapy due to be reported shortly will not resolve the issues as there will be perennial debate about inclusion and exclusion criteria in such trials and these will have a great bearing on outcomes. Moreover, the ability to quantitate the lifetime risk reduction of peptic ulcer disease and perhaps even gastric carcinoma in patients who have eradication therapy will remain contentious. Medico-legal issues and patient preferences will also continue to be important factors influencing the decision to investigate and treat. At present the suggested test and treat strategy of uninvestigated patients seems reasonable for well-informed, low-risk patients with endoscopy the recourse if needed. Further investigation and the decision to test and treat for *H pylori* in uninvestigated dyspeptics and investigated dyspeptics who fit the criteria for non-ulcer dyspepsia will no doubt remain a decision that is assessed on a "case by case" basis as suggested in the recent report of the American Digestive Health Initiative.[1]

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1 Anonymous. The report of the Digestive Health Initiative International Update Conference on Helicobacter pylori. *Gastroenterology* (in press).

Functional dyspepsia in the young

EDITOR,—I read with interest the Maastricht Consensus Report on the diagnosis and treatment of *Helicobacter pylori* infection (*Gut* 1997;**41**:8–13). Whereas the role of *H pylori* in peptic ulcer disease, gastric carcinoma and mucosa associated lymphoid tissue type lymphoma is established, its role in functional dyspepsia is still controversial. Recent data indicate that *H pylori* positive patients with functional dyspepsia benefit from eradication therapy.

In 1989, we published a treatment algorithm in which serological screening had a key part in the decision whether or not to endoscope patients presenting with dyspepsia.[1] We suggested that endoscopy was not essential and advocated anti-*H pylori* treatment in seropositive dyspeptic patients. In our original algorithm there were several unanswered questions regarding coincidental non-helicobacter related disorders. These questions would have to be answered before serological screening could be used in routine practice. At that time this algorithm was refuted.[2] Nevertheless since then several papers have been published in which serological screening was used. However no data were available on non-helicobacter related disorders of the upper gastrointestinal tract and also real screening was not done as selected patient populations were used.[3–5]

Much to my surprise the Maastricht Consensus Report advocates anti-*H pylori* therapy in seropositive dyspeptic patients under 45 years of age without the need for endoscopy. Although, from a clinical point of view I fully agree with this statement, it is based on common sense and not on scientific evidence. To the best of my knowledge, no prospective studies have been done in which seropositive patients did not undergo endoscopy. Selected patient populations were studied in all of the references quoted in the report. Endoscopy should be omitted, in retrospective analysis, on seronegative cases.

If serology is used and endoscopy is not performed in selected cases, whether *H pylori* positive or negative, it is inevitable that some cases of non-helicobacter related disease will be missed, reflux oesophagitis being the most important. It is essential that a non-selected patient population is assessed to determine how many cases of reflux oesophagitis would be missed if endoscopy was not done. This is especially true as the clinical presentation of reflux oesophagitis is far from specific. We showed in a recent paper that the majority of dyspeptic patients with reflux oesophagitis were *H pylori* negative,[6] and that, at least in theory, the best screening strategy seemed to be to omit endoscopy in seronegative patients.

The statement that serological screening is cost effective and leads to more efficient use of endoscopy facilities has yet to be proved in prospective randomised studies. The only study published to date is unsuitable as a selected patient population was used.[7]

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Dual publication

EDITOR,—I was astonished, as I am sure many were, to see publication of the The Maastricht Consensus Report (1997;**41**:8–13) in *Gut*. Not only was this surprising, but to see it appear as a leading article was even more amazing particularly in an issue which carried an editorial by yourself on research misconduct, quite rightly condemning similar practices.

Under the circumstances, it does not appear unreasonable to enquire whether you were aware at the time that a synopsis of this event had previously been published in the *European Journal of Gastroenterology and Hepatology* (1997;**9**:1–2)? If so, no acknowledgement appears to have been included in this parallel report. Had you been informed that the meeting from which this report had its origins was organised "with an educational grant from Astra-Hässle" with accompanying documentation inferring that travel and hotel expenses were paid for participants and discussions limited to those who were paid for? If so, why is this not acknowledged in the leading article and it registered as a possible "conflict of interest" as seems to be the philosophy of your parent publishing group, and acceptance of financial support within the stated policy of your own journal. Perhaps your readers should further be aware that this publication is the result of discussions by a self-appointed group who have no mandate to represent any official bodies or organisations.

In the light of the above, it will perhaps come as no surprise that the conclusions recommend widespread testing of dyspeptics under the age of 45 years for *Helicobacter pylori* and subsequent treatment in primary care, supported by no evidence-base whatsoever. The other major conclusion, that a proton pump inhibitor based treatment regimen should usually be used is perhaps also understandable in the light of the conference's financial support. Indeed, the conclusions are not even supported by data quoted by these authors themselves, which includes a number of studies with various proton pump based triple therapies which, on an "intention to treat" basis have eradication rates less than the stated ideal of 80%.

It appears that you have either been seriously misled or made a grave error of judgement in publishing this paper. Its contents are confused and misleading, its conclusions restrictive and its appearance in print repetitive, and it does little to guide any of us in the management of *H pylori* infections in Europe, or indeed the world, today. Furthermore, its publication does little to enhance the reputation of *Gut* internationally.

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Reply

EDITOR,—We expected that controversy and criticism would follow our attempt to propose European guidelines for the management of a disease as complex as *Helicobacter pylori* infection. Dr Heatley's letter however goes beyond this and can be only regarded as an example of destructive and uninformed criticism. Dr Heatley incorrectly reports aspects of both the structure and the nature of the Maastricht meeting.

The allegation of dual publication, and to castigate both the authors and the editor, is unfair and misleading. An abstract was published in the *European Journal of Gastroenterology and Hepatology*, but the complete report with a detailed description of the meeting structure and outcome, including references supporting the various conclusions, was published in *Gut*. The Consensus Report is **not** original work but is of educational value and was intended to be disseminated widely

The group was not self appointed. The Maastricht Conference gathered together an expert faculty from various medical disciplines, the European *Helicobacter pylori* Study Group, and national representatives, who were nominated by their national gastroenterology societies from 19 European countries. Our aim was to produce management guidelines in this very complex field.[1 2]

The European *Helicobacter pylori* Study Group, since its foundation in 1987,[3] has been very active in the organisation of educational and scientific meetings both in and beyond Europe initially at a time when *H pylori* was not widely accepted as a cause of gastric disease and presently when there is increasing demand for guidance in treating the infection.

To blame the organisers for seeking support from industry, which was given in the form of an unrestricted educational grant, is unfair. Representatives from the major pharmaceutical companies with an interest in the treatment of peptic ulcer disease, such as Astra, Byk Gulden, Glaxo Wellcome, and Takeda, were invited and did in fact attend the meeting.

With regard to medical aspects of Dr Heatley's criticisms, we would like to remind him that treating *H pylori* positive dyspeptic patients under 45 years of age, without alarm symptoms, with eradication therapy in primary care is not uncommon in the UK.[4] In the absence of evidence, the best available knowledge and experience should be taken into account in guiding clinical practice. At the Maastricht conference we suggested that general practitioners, gastroenterologists and microbiologists should form an interactive network to implement and sustain our recommendations.

The firm recommendation for the PPI containing regimen at a standard dose given twice daily in combination with amoxycillin, clarithromycin or metronidazole was based on the clinical trials available at that time which showed that this treatment was the most suitable in terms of efficacy, compliance and side effects.

We never intended that the Consensus Report should be the final word on the management of *H pylori* infection but rather that is would be a starting point and would prompt discussion over the next few years. Certainly, it had enough impact to serve as a model and to inspire other important consensus conferences that have recently taken place in North America, Japan and in the Asia-Pacific region.

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From the editor

The authors of the Maastricht Consensus Report were entirely open about the fact that they intended to submit a brief synopsis of the report to the *European Journal of Gastroenterology and Hepatology* at the time that they submitted their manuscript for consideration for publication in *Gut*. I regarded this as an "abstract" of the main report and thus did not consider this dual publication. The authors did not disclose the meeting sponsor in the report but this oversight was put right in a subsequent issue of the journal.

We decided to publish the Maastricht Consensus Report because of the importance of *Helicobacter pylori* in clinical practice. I am not surprised that the views expressed do not necessarily find universal approval but one should never shy away from controversy.

Efficacy of ranitidine bismuth citrate (RBC) dual and triple therapies for the eradication of *Helicobacter pylori*

EDITOR,—We write in response to the leading article "Current European concepts in the management of *Helicobacter pylori* infection. The Maastricht Consensus Report" recently published (*Gut* 1997;**41**:8–13).

The phrase "Additionally, no recommendation can be made regarding the role of RBC until more convincing data are available" is somewhat at variance with the conclusion agreed at the meeting in Maastricht in September 1996 which was sponsored by Astra-Hässle.

The data on RBC reviewed at the Maastricht meeting was based on conclusions drawn from limited information on clinical trials which did not have the eradication of *H pylori* as the primary endpoint. The actual conclusion at the end of the meeting was that "more data are needed to define the role of RBC". This was disseminated widely in the form of a document handed out at the 5th United European Gastroenterology Week in Paris in November 1996, and in the summary of the Maastricht conclusions printed in January 1997.[1]

In the year since this meeting took place, results from several clinical trials on RBC dual and triple[2 3] therapy have been published or presented at several international congresses. RBC has now been evaluated in clinical trials for the eradication of *H pylori* in over 6300 patients. Our conclusions based on all clinical trials to date are that dual therapy of RBC 400 mg twice daily with clarithromycin 500 mg twice daily for 14 days (nine treatment arms from nine clinical studies; six double blind) gave a pooled observed eradication in 946 of 1037 patients (91.2%), a pooled intention to treat eradication in 946 of 1153 patients (82.0%), and the eradication rates were comparable in patients with or without an extra 14 days of RBC 400 mg twice daily added to ensure duodenal ulcer healing. A clinical study evaluating RBC with clarithromycin dual therapy showing a per protocol eradication rate of 95.9% in a large, double blind, randomised study was published in February 1997.[4]

Results of the first two head-to-head, double blind, randomised clinical studies showed that therapy with RBC and clarithromycin gave *H pylori* eradication rates which were highly significantly superior, both clinically and statistically, to omeprazole plus either amoxycillin[5] or clarithromycin.[6]

For those wishing to use three drugs in place of two, triple therapy of RBC 400 mg twice daily with clarithromycin twice daily and a nitroimidazole for seven days (nine treatment arms from eight clinical studies; three double blind) gave a pooled intention to treat eradication in 659 of 751 patients (87.7%). Triple therapy of RBC 400 mg twive daily with clarithromycin 500 mg twice daily and amoxycillin 1000 mg twice daily for seven days (eight treatment arms from seven clinical studies; two double blind) gave a pooled intention to treat eradication in 348 of 417 patients (83.5%). Both these treatment regimens were as effective as a proton pump inhibitor with the same antibiotics.

The latest information available from clinical trials with RBC–antibiotic combinations show that either a 14 day dual therapy of RBC with clarithromycin or a seven day triple therapy of RBC with two antibiotics achieve >80% eradication of *H pylori* as assessed on an

intention to treat basis. Each of these regimens therefore meet the "Maastricht criteria" of simple and effective eradication therapies.

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Reply

EDITOR,—While it is true that Astra Hässle provided the grant to the European *Helicobacter pylori* Study Group (EHPSG), it is also true that Drs Duggan and Williamson from Glaxo Wellcome were invited to and took part in the Maastricht meeting.

It was the firm intention of the organisers to collect all available information from clinical trials on *H pylori* with the aim of producing comprehensive guidelines for the management of *H pylori* infection. At that time however (September 1996) there were not sufficient data on RBC based seven day treatment for it to be recommended in the Consensus Report. New data are now available. At the recent EHPSG meeting in Lisbon further data were presented on the efficacy of RBC based treatment and it now meets the criteria agreed at Maastricht.

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BOOK REVIEWS

Inflammatory Bowel Disease: Trigger Factors and Trends in Therapy. Caprilli R, ed. (Pp 218; illustrated; price not given.) Stuttgart: Schattauer, 1997. ISBN 3-7945-18808-X.

In preface to my review of Dr Caprilli's interesting book I must declare a bias, as I am the author of a volume of similar size and title, also published in 1997. I hope that this has not influenced my comments too much, as the style and objectives of the two books are quite different and potentially complementary. Although it has a strongly topical slant, my book is much more a general overview of inflammatory bowel disease and bears the strengths and weaknesses of single authorship. In contrast, Dr Caprilli has gathered together the material of the 46 contributors to a conference held in 1996. Many of the names are well known, representing major centres in Europe (including Israel) and North America, with a perhaps understandable leaning to the Italian (22 authors). It seemed at first a little odd that there were no British contributors, but rather than encouraging collective xenophobic paranoia this may just as well represent a reason for commending the dedication of my colleagues to their national society, given that the British Society of Gastroenterology was meeting simultaneously, and in Manchester compared with Capri at that!

The book is arranged into six main sections, and in addition to the themes implied by its subtitle, there are pieces on disease stratification, postoperative recurrence, pouchitis, and the place (or otherwise) of the ileorectal anastomosis as definitive therapy for ulcerative colitis. It is probably a reflection of the passage of time that, although there is a chapter on family studies, there is no focused section on genetics. It is less clear why the measles hypothesis is not included in the section on predisposing and trigger factors—perhaps because Professor Ekbom was recruited to write instead about cancer! Measles does not appear in the index, but there is in fact a reasoned analysis of its potential importance in a wide-ranging chapter on paediatric inflammatory bowel disease.

A FORBES

Hepatobiliary, Pancreatic and Splenic Disease in Children. Medical and Surgical Management. Balistreri W F, Ohi R, Todani T, Tsuchida Y, eds. (Pp 605; illustrated; 495.) Amsterdam: Elsevier Science, 1997. ISBN 0-444-82052-3.

This is an earnest and well meaning book written by mainly Japanese and American authors. Although entitled *Medical and Surgical Management of Hepatobiliary, Pancreatic and Splenic Disease in Children*, this book concentrates on surgical management of selected topics and cannot be considered a comprehensive guide to the medical management of paediatric liver disease.

Nevertheless the book has many strengths. There are five chapters on basic morphology and physiology of the liver which include an excellent summary of the embryology of the liver and bile ducts in this rapidly developing field, and details of the development of the pancreas, including the normal and variant anatomy, which would be helpful to both surgeons and endoscopists. This was followed by a fascinating chapter on the functional development of the liver which explains many of the difficulties experienced by neonates with liver disease. There are very detailed chapters on bilirubin and bile acid metabolism which explore the basis for neonatal jaundice and inborn errors of bile salt metabolism.

The next series of chapters are devoted to the investigation of the child with liver disease. I found the chapter on the role of liver biopsy disappointing as it contains a number of factual errors (for example, Wilson's disease does not present with neonatal cholestasis) and does not discuss the difficulties of making an early histological diagnosis of biliary atresia, which is an important practical problem in the 1990s.

The chapters on the role of imaging with ultrasound and hepatoscintigraphy are more detailed than would be required and although clinical applications are discussed, clear guidelines are not obvious, particularly as scintigraphy is now rarely used. In contrast the chapter on the use of magnetic resonance imaging and computed tomography scanning is both well written and relevant.

The chapters devoted to the use of percutaneous transhepatic cholangiography and endoscopic cholangiopancreatography are brief and many important indications for these techniques are not included (for example, sclerosing cholangitis, post-transplant complications).

In the clinical section of the book, the chapter on neonatal cholestasis provides a good practical guide to the investigation and management of these infants, but no other medical liver diseases are included with the exception of cystic fibrosis.

Given the importance of biliary atresia to the Japanese community, it is not surprising that this disease is over represented in this book. There is a long and detailed chapter concentrating on the surgical aspects of hepatic portoenterostomy which duplicates information in earlier chapters.

The surgical highlights include an up to date and relevant chapter on the medical and surgical aspects of portal hypertension by Professor Howard, and a detailed review of liver trauma which will be of considerable help to both paediatricians and surgeons caring for children with abdominal trauma.

No book on liver disease would be complete without a chapter or chapters on liver transplantation. Transplantation from both cadaveric and living donors is discussed with the emphasis very firmly on the surgical approach and management. There is little attempt to provide information on quality of life and survival.

Pancreatic disease in childhood is rare and one the strengths of this book is to explore the management of pancreatitis, pancreatic trauma and of pancreatic tumours.

A whole section is devoted to diseases of the spleen—for example, trauma and multisystem involvement, which is unusual but it was fascinating to discover the entity of the wandering spleen. Although the technique of splenectomy is discussed, the indications for this usually unnecessary procedure are not included.

In summary, this is a rather patchy book with strengths in basic physiology and morphology and sound on surgical technique and detail. I am sure that it will provide a useful reference book for young surgeons but will disappoint paediatricians looking for comprehensive medical management of hepatobiliary disease.

D A KELLY

Surgery of the Colon and Rectum. Nicholls J, Dozios R R, eds. (Pp 1300; £185.00.) Edinburgh: Churchill Livingstone, 1997. ISBN 0-443-05565-3.

This latest volume on colorectal disease represents an attempt to bridge the Atlantic gap

by bringing together the expertise of authors from both sides of the water, emphasising this by appointing distinguished joint editors from the UK and the USA. The publishers thereby have attempted to widen their potential audience. The place of such a textbook needs to be defined with the knowledge that within the American and the UK markets there are already two respected volumes covering the same subject. The publishers obviously still feel that there is a continuing market for such a text book in a world where the potential purchaser is becoming increasingly computer literate.

Some of the problems with multi-author textbooks include presentation and repetition. I think there is a contrast between the UK and the American style in writing and the American flavour in clinical practice is particularly evident in the chapter on the consulting room set-up and the range of procedures done, with suggestions that the expensive flexible sigmoidoscope will replace the much cheaper and more easily available rigid sigmoidoscope. Repetition may not be such a problem if any approach is from a different angle but—for example, in chapters 5 and 10 there is much repetition and in the chapter on haemorrhoids, perianal haematoma appears on both pages 214 and 230 with much overlapping content. There is also a repeat discussion on pages 352 and 371 on the adenoma-carcinoma sequence. Better editing is required for the next edition.

The authors have attempted a very wide coverage of their subject while at the same time publishing in one volume. This has produced a heavy volume to hold while reading and at the same time in order to accommodate all the data the print type is small and not easy to read for any period of time.

Having initially perhaps been rather negative this is a very comprehensive book with the included chapters having been written by a number of eminent contributors and I have no doubt that it will stand healthily alongside its competitors. I would suggest that this book will find its role as an excellent reference book rather than perhaps being a volume that students of coloproctology, be they undergraduates, potential postgraduates or consultants, will read as their initial introduction to the subject.

R GRACE

NOTES

6th Southeast European Congress of Paediatric Surgery: Short Bowel Syndrome

The 6th Southeast European Congress of Paediatric Surgery: Short Bowel Syndrome will be held in Graz, Austria, on 22–23 May 1998. Further information from: Dr Günther Schimpl, Department of Paediatric Surgery, Auenbruggerplatz 34, A-8036 LKH-Graz, Austria. Tel: +43 316 385 3762; Fax: +43 316 385 3775.

9th British Association of Day Surgery Annual Scientific Meeting

The 9th British Association of Day Surgery Annual Scientific Meeting and Exhibition will be held at the Harrogate International Centre, Harrogate, UK, on 4–6 June 1998. Further information from: Kite Communications, The Silk Mill House, 196 Huddersfield Road, Meltham, West Yorkshire HD7 3AP, UK. Tel: 01484 854575; Fax: 01484 854 576; email: info@kitecomms.co.uk.

9th International Symposium on Cells of the Hepatic Sinusoid

The 9th International Symposium on Cells of the Hepatic Sinusoid will be held in Christchurch, New Zealand, from 27 September to 1 October 1998. Further information from: Professor Robin Fraser, I.S.C.H.S., Christchurch School of Medicine, PO Box 4345, Christchurch 8001, New Zealand. Tel: +64 3 3640 587; Fax: +64 3 3640 593; email: grogers@chmeds.ac.nz.

Growth Factors and Nutrients in Intestinal Health and Disease

An International Symposium on Growth Factors and Nutrients in Intestinal Health and Disease will be held at the Rihga Royal Hotel, Osaka, Japan, from 31 October to 3 November 1998. Further information from: Kinya Sando, MD, Department of Pediatric Surgery, Osaka University Medical School, 2-2 Yamadoaka, Suita, Osaka 565, Japan. Tel: +81 6 879 3753; Fax: +81 6 879 3759; email: gut@pedsurg.med.osaka-u.ac.jp.

XXXth Annual Meeting of the European Pancreatic Club

The XXXth Annual Meeting of the European Pancreatic Club will be held in Thessaloniki, Greece, from 10 to 13 June 1998. Further information from: Diastasi, Congress Secretariat, 30 Katsimidou Street, Thessaloniki 54639, Greece. Tel: 30 31 938 203 or 905 110; Fax: 30 31 909 269.

XXIIIth International Update on Liver Disease

The XXIIIth International Update on Liver Disease will be held at the Royal Free Hospital School of Medicine, London, UK, from 16 to 19 July 1998. Further information from: Professor Neil McIntyre, University Department of Medicine, Royal Free Hospital, Pond Street, London NW3 2QG, UK. Tel: 0171 794 0500 ext 3969; Fax: 0171 830 2321.

Hong Kong Academy of Medicine—First International Congress

The Hong Kong Academy of Medicine will be hosting its First International Congress from 26 to 29 November 1998 in commemoration of the grand opening on its new building. Further information from: Ms Colour Lee, Conference Manager or Miss Phoebe Wong, Administrative Assistant, Hong Kong Academy of Medicine, 9/F, Multicentre Block A, Pamela Youde Nethersole Eastern Hospital, 3 Lok Man Road, Chai Wan, Hong Kong. Tel: 852 2515 5755; Fax: 852 2505 3149; Email: hkam@hkam.org.hk.

Hepatic and Splanchnic Circulation in Health and Disease

An International Conference on Hepatic and Splanchnic Circulation in Health and Disease will be held in Inverness, Scotland, from 20 to 23 June 1999. The conference is designed to provide an international forum of discussion among those interested in the circulatory control of the liver and splanchnic region. Free communication (including keynote lectures) and poster sessions will cover: physiological control, endothelial function, innervation, ischaemia-reperfusion injury, inflammation, portal hypertension, transplantation. **Abstract deadline: 1 November 1998**. Further information from: Dr Robert T Mathie, Department of Gastrointestinal Surgery, Imperial College School of Medicine, Hammersmith Hospital, London, UK. Tel/Fax: 0181 383 2267; Email: rmathie@rpms.ac.uk; Internet: http://www.otago.ac.nz/inverness.

Falk Symposia and Workshops

The Symposium on Innovative Concepts in Inflammatory Bowel Diseases will be held in Rostock, Germany, from 30 April to 2 May 1998.

The Symposium on New Aspects in Hepatology and Gastroenterology will be held in Tbilisi, Georgia, on 29 and 30 May 1998.

The Symposium on Advances in Inflammatory Bowel Diseases will be held in Brussels, Belgium, on 18–20 June 1998.

The Symposium on Diseases of the Liver and the Bile Ducts—New Aspects and Clinical Implications will be held in Prague, Czech Republic, on 12 and 13 June 1998.

The XV International Bile Acid Meeting: Bile Acids in Cholestasis will be held in Titisee, Germany, on 12 and 13 October 1998.

The Symposium on Colorectal Cancer: Molecular Mechanisms, Premalignant State and its Preventions will be held in Titisee, Germany, on 14 and 15 October 1998.

The Symposium on Intestinal Mucosa and its Diseases—Pathophysiology and Clinics will be held in Titisee, Germany, on 16 and 17 October 1998.

For further information on any of these symposia, please contact: Falk Foundation e.V.—Congress Division, Leinenweberstr. 5, PO Box 6529, D-79041 Freiburg, Germany. Tel: +49 761 130 340; Fax: +49 761 130 3459.

Clinical Training and Research Opportunities in Gastroenterology in Italy

The Società Italiana di Gastroenterologia (SIGE) have produced a booklet on clinical training and research opportunities in gastroenterology in Italy. Further information and a copy of the booklet are available from: SIGE Società Italiana di Gastroenterologia, Via Salvatore di Giacomo 66, 00142 Rome, Italy. Email: roma99sige@uni.net.