LETTERS TO THE EDITOR

Autoimmune hepatitis in the elderly

EDITOR,—We were interested to read Professor James' comments on autoimmune hepatitis (AIH) in his review of parenchymal liver disease in the elderly (*Gut* 1997;41:430-2). He described an unpublished series of patients with AIH, 20% of whom were over 65 years of age¹ at presentation. We recently published an 11-year follow up of patients with type I AIH from an unselected population in South Wales.² Forty one patients were identified according to the AIH group criteria. The median age was 62 years and 23 (56%) patients were over 60 years of age at presentation; the usual presentation was an acute icteric hepatitis. Response to immunosuppression in the over-60's was gratifying and prompt and the five year survival rate compared favourably with that of our series as a whole. Our experience is similar to that of the Newcastle group, and it now seems clear that AIH should no longer be regarded primarily as a disease of the young. Indeed, our unselected series suggests that AIH is primarily a disease of the elderly. The diagnosis should be considered in any patient presenting with acute icteric hepatitis whatever his or her age.

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Consideration of histopathological subtypes and biopsy techniques in Barrett's oesophagus surveillance programmes

EDITOR,—In their recent study Macdonald *et al* (*Gut* 1997;41:303-7) concluded that as only 1 in 143 patients was identified as having carcinoma of the oesophagus as a result of surveillance, the policy should be reconsidered. This conclusion raises several questions: Which patients should undergo surveillance and does the histopathological subtype matter? In this study patients eligible for the endoscopic surveillance programme had to be under the age of 70, without other serious coexisting disease. The endoscopic Barrett's segment was >3 cm in all cases and patients with all histological subtypes (gastric 78% and specialised intestinal metaplasia 18%) were included. The definition of Barrett's oesophagus has a long and controversial history.¹ Specialised intestinal metaplasia is increasingly recognised as being the most common histological subtype,^{2,3} and patients with this epithelial type are at high risk of developing cancer.^{4,5} On the basis of this, many

investigators now use the term Barrett's oesophagus to indicate the presence of specialised intestinal metaplasia.⁶ Furthermore, a recently proposed re-classification of Barrett's oesophagus suggests that patients with specialised intestinal metaplasia extending beyond the gastro-oesophageal junction are the only group in which endoscopic surveillance is currently warranted.¹ *How many biopsy specimens should be taken?* In the Leicester study specimens were typically taken from all four quadrants at the mid-point of the involved mucosa. However, given the heterogeneous nature of the Barrett's epithelium,⁶⁻⁹ it may be necessary to take samples in every quadrant every 2 cm along the length of the Barrett's epithelium in order to detect areas of high grade dysplasia and carcinoma in situ. This is the regimen currently recommended by the Working Party Report to the World Congress of Gastroenterology.¹⁰

Perhaps if Barrett's surveillance programmes concentrated on patients with specialised intestinal metaplasia, and involved the collection of multiple biopsy samples, this would improve the yield and cost effectiveness of such programmes? Hopefully in the future, our collective experiences of Barrett's surveillance and a clearer understanding of the pathophysiology of Barrett's oesophagus, might enable a consensus to be reached.

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Reply

EDITOR,—We are grateful to Drs Fitzgerald and Burnham for their comments on our paper. It is important to remember that our report is a retrospective study encompassing our clinical practice over the past 10 years. As discussed in the paper, the definition of what constitutes Barrett's oesophagus has continued to alter over this period, the inclusion of short segment Barrett's being an additional complicating factor. We agree that many workers in the area now consider that the

presence of intestinal metaplasia is an absolute requirement for a diagnosis of Barrett's oesophagus to be made and that intestinal metaplasia constitutes a risk factor for the development of carcinoma.

In our paper, we quoted the criteria suggested by the World Health Organisation for screening for any disease.¹ As there is little evidence that these criteria have been met for screening of Barrett's oesophagus, particularly with regard to altering the outcome of patients,² caution must be shown in interpreting the guidelines produced at the World Congress of Gastroenterology³ as a "gold standard". We agree that performing four-quadrant biopsies every 2 cm is likely to pick up additional areas of mucosal abnormality. It must be remembered, however, that in our study the average length of the Barrett's mucosa was 7 cm and would therefore require an additional eight biopsy samples per patient. This has major implications for providing a screening service as it will increase the time taken to perform the procedure, thereby reducing the number of procedures that can be performed on an endoscopy list. It is also possible that this would result in a reduction in the patient's willingness to undergo further screening. In addition, the histological costs would be increased by about £60 per patient per endoscopy.

There is general agreement that large multicentre studies are required for a conclusive assessment of the value of screening patients with Barrett's mucosa to be reached. Focusing on subgroups, such as those with co-existing strictures, may prove to be the only cost effective approach.

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Electrical spinal cord stimulation for painful peripheral neuropathy secondary to coeliac disease

EDITOR,—Spinal cord stimulation has been used for pain relief in patients with painful diabetic neuropathies.¹ Coeliac disease is associated with varied neurological complications (5-8%) including ataxia, peripheral neuropathy, myelopathy, myopathy, and dementia.² We report the novel use of electrical spinal cord stimulation for the management of chronic painful peripheral neuropathy secondary to coeliac disease.

A 47 year old man presented to the pain clinic with an 11 year history of generalised aches and pains in the lower limbs. However, they were now becoming more symptomatic and shooting in character. This generalised body pain resulted in his inability to work as a bank official. During the previous year he suffered from nausea, vomiting and weight loss, and a subsequent jejunal biopsy specimen yielded a diagnosis of coeliac disease. An

initial impression was that of peripheral neuropathy secondary to coeliac disease. Nerve conduction studies showed no evidence of large fibre peripheral neuropathy. Bone biochemistry confirmed vitamin D deficiency, secondary hyperparathyroidism and a notable increase in bone turnover. After a year on a gluten-free diet these indexes returned to normal; however, the patient still suffered from severe neuropathic pain in the lower extremities and was unable to resume full employment.

Initial pharmacological therapy for this painful neuropathy included trials of intravenous lignocaine and oral gabapentin which were without therapeutic benefit. In view of his ongoing symptoms and decreasing physical function, a spinal cord stimulator (Medtronic I TREL III system) was implanted and neuropathic pain relief was assessed by visual analogue scale. Pain was reduced by about 60–70%, and within two months the patient was able to return to full employment. This pain relief is sustained to date.

Although painful diabetic neuropathies have been treated with spinal cord stimulation,¹ to our knowledge this is the first case of a painful peripheral neuropathy secondary to coeliac disease being treated in this way. The aetiology of neurological dysfunction in coeliac disease is poorly understood and research has largely concentrated on vitamin deficiencies (B₁₂, E, D, folic acid, and pyridoxine) as a result of malabsorption. However, vitamin replacement rarely improves the neurological deficit or neuropathic symptoms.² We suggest that implantation of a spinal cord stimulator may result in improved physical function and decreased pain in patients with painful peripheral neuropathies secondary to malabsorption syndromes. Further studies to elucidate these mechanisms might allow spinal cord stimulation to be exploited more effectively in painful disorders that are currently refractory to conventional treatment modalities.

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Research misconduct and redundant publication

EDITOR.—The editorial on research misconduct (*Gut* 1997;**41**:1–2) is timely. Duplicate reports, redundant publications and “salami slicing” are products of today’s environment where academics are often judged by the length of their curriculum vitae and number of publications,^{1–3} rather than the quality of work and whether it has any impact on current medical practice. This necessity to publish may be due to institutional pressures, personal ambition, vanity, direct financial

gain, or even psychiatric illness.¹ The prevalence of fraud is estimated to be around 0.1–0.4% of research studies and over 70 cases have been documented.^{4–5} About 5% of drug trials are thought to involve misconduct of some sort.⁶ Peer review offers little or no protection against fraud.⁷

A few years ago, as assistant editor of a psychiatry journal, a case of duplicate publication was brought to my notice, several months after the original article had been published. The authors had sufficiently disguised the second article and it was published in a journal from another specialty. There was some debate about what constituted acceptable overlap; the paper concerned fell in the grey area and it was felt that the extent of duplication was not sufficient to prompt a retraction. Apart from informing the editor of the second journal, no further action was taken to my knowledge. However, I believe that if we were made aware of the duplicate version earlier, the paper in question would not have been accepted. Combining both manuscripts would certainly have led to a more comprehensive paper. The threshold for retracting a published article is probably much higher than the threshold for rejection before publication.

In response to a similar situation, the editors of *Annals of the Rheumatic Diseases* have introduced the following statement in their information for authors: “Authors must declare, and submit copies of, any manuscripts in preparation or submitted elsewhere that are closely related to the manuscript to be considered.” I believe that this may not be sufficient as it provides a loophole as to what constitutes closely related, an area which authors have exploited. Often what editors consider “closely related” is disputed by the authors.⁹

Therefore, in addition to copies of closely related manuscripts, it may be useful to ask authors to submit a list of all their publications (related and otherwise), including those currently in the pipeline. Random audits can be done to ensure that there are no other papers which editors consider to be similar, which authors may not otherwise declare. These audits may be restricted to those authors who have a large number of publications (say, over 25 or 50) as they are more likely to have made a career out of such practice over several years. I emphasise that the vast majority of researchers with numerous publications would not have indulged in unethical research practice, but by definition, it is in this group that one would have a higher chance of finding expert salami slicers and those with overworked photocopyers. This method is certainly not fool proof and a clever cheat is difficult to expose⁷—but this at least increases the level of deception necessary for duplicate publications and may deter all except those with antisocial personality traits, nurtured in an environment that looks for quantity, not quality.

At least one journal black lists authors involved in dual publication for a period of five years.⁸ However, this does not prevent recalcitrant authors from perpetrating the same fraud on another journal, considering that retraction notices are not always obvious or easily accessible through a computerised database such as Medline.⁹ Editors have already agreed on uniform requirements for manuscripts. It may now be time for editors to maintain a common register of authors who have been black listed by one journal (similar to the Food and Drug Administra-

tion’s black list of clinical investigators),⁶ so that others are aware of the same. As members of the scientific community, the only way to avoid the threat of a “science police” is to encourage science to police itself.¹⁰

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BOOK REVIEW

Clinicians’ Guide to Inflammatory Bowel Disease. Forbes A. (Pp 257; illustrated; £39.50.) London: Chapman and Hall, 1997. ISBN 0-412-788500-0.

An “intellectual feat by one person achieves a global view of the topic with continuity and balance,” writes John Lennard-Jones in the foreword, and one has to agree. This is a monograph at its best which challenges the Goliath of multi-authored texts. Current literature is thoughtfully interpreted and coherently presented in a way that edited textbooks rarely achieve, forming the foundation for good advice on management.

It is aimed at gastrointestinal trainees and surgeons, as well as non-clinical research workers and specialist workers in the field, but will also be useful to experienced gastroenterologists. The overview of pathogenesis is masterly in its lucid analysis of the different avenues of research. So too are the sections on nutritional therapy, alternatives to steroids, short bowel syndrome, enterocutaneous fistulae, extraintestinal manifestations, and the controversial topic of cancer surveillance. For a book of this size (250 pages) it is extraordinarily well referenced (>700); indeed, the references are so up to date that it is mildly frustrating that some landmark papers before 1990 are not mentioned.

Given the style and detail with which complex issues are explained, I found the sections on standard clinical manifestations and treatment relatively superficial. I was not left with a clear impression of a treatment strategy that non-specialists could follow for uncomplicated cases. It is possible that this reflects the tertiary referral practice at St Mark’s, but in a book that excels in presented reasoned data, it would have been helpful to have tales evaluating the trials on steroid and salicylate therapy.

More discussion of the common clinical dilemmas of diarrhoea or abdominal pain unrelated to disease activity would also be helpful, as would expansion of the sections on functional aspects, social impact and prognosis. It is a pity that the index and radiograph reproduction are so poor, but this is the responsibility of the publishers who should sort this out for other books in this series.

These are minor criticisms and are measured against outstanding sections of the book, rather than other texts. It must be recommended to all gastroenterologists, trainees, and surgeons. Library copies will walk.

S TRAVIS

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 Yamadaoka, Suita, Osaka 565, Japan. Tel: +81 6 879 3753; Fax: +81 6 879 3759; email: gut@pedsurg.med.osaka-u.ac.jp.

Falk Symposia and Workshops

The Symposium on Induction and Modulation of Gastrointestinal Inflammation will be held in Saarbrücken, Germany, on 5–7 March 1998.

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.

CORRECTIONS

In the title to the paper by Andus *et al* (*Gut* 1997;41:651–7), agonist should read antagonist.

In table 3 in the paper by Iseki *et al* (*Gut* 1997;42:20–3), the data in the Undifferentiated and Total lines are incorrect. These should read, from left to right, Undifferentiated: 6 (40), 2 (13), 7 (47), and 15 (100); Total: 50 (77), 3 (5), 12 (18), and 65 (100).