Small bowel biopsies in patients with iron deficiency anaemia

EDITOR,—We noted in the guidelines for the management of iron deficiency anaemia (Gut 2000;46(suppl IV):i1–i5) that it is recommended that small bowel biopsies should be undertaken in patients presenting with iron deficiency anaemia. This has been the subject of much discussion between the pathology department and ours recently.

Antendiomyal antibodies (EMAs) give a result close to 100% sensitivity and specificity 1 although other studies suggest around 95% sensitivity with much lower values for specificity depending on the exact criteria for gluten sensitive enteropathy adopted. 2 This has been discussed at both the recent British Society of Gastroenterology (BSG) meeting (Ciclitira PJ, State of the Art Lecture: Coeliac Disease—pathogenesis and prevention. BSG Annual Meeting, March 2000) and the American Gastroenterology Association (AGA) meeting (Schuppan D, Spryc: new insights into pathogenesis and management. AGA Postgraduate Course, May 2000).

A possible reason for these differences in sensitivity and specificity in the literature is that standards vary between different pathology departments in the techniques of anti-EMA assessment. 3 Most hospital biochemistry departments perform the tests using monkey oesophagus tissue bought from commercial suppliers; on the market at present there are 19 different suppliers of monkey oesophagus and 23 different suppliers of the enzyme immunoassay (EIA) (United Kingdom External Quality Assessment Services for Autoimmunity) (NEQAS). These tissue samples will be of varying quality, and the EIAs will vary in their accuracy. To compound the problems the immunofluorescent technique is essentially subjective, and results will vary between different observers. Our laboratory automatically measures total IgA levels, patients deficient in IgA will have negative IgA EMA, and laboratories need to have the resources to measure IgA EMA in these patients.

Our biochemistry laboratory is evaluating antitissue transglutaminase antibody (ATGAs) assays to replace the monkey oesophagus immunofluorescence system, which should be more automated and therefore more cost effective. Initial sensitivity and specificity for ATGAs assays are promising. 4 There is some evidence that endoscopic markers are useful in detecting villous abnormality in coeliac disease 5 and we await the development of high resolution endoscopy. 6 It seems that although duodenal biopsy is traditionally regarded as the gold standard, there is still the possibility that false negatives in patients with patchy villous changes will occur with these screening methods. A recent abstract from the AGA from Columbia University, New York, found that fewer than half of duodenal biopsy samples in their study were orientated sufficiently to allow evaluation of villous atrophy, and 39% of patients had patchy disease. 7

The implications in terms of resources for the histology department in handling multiple duodenal biopsies in all patients with iron deficiency anaemia are considerable and this is currently the subject of an internal audit at our hospital. Although for the reasons stated anti-EMA has a higher specificity than histology, and we have found that the prevalence of duodenal biopsy specimens reaching the histopathology departments even if duodenal biopsy were a second line test. We wonder if the guidelines for duodenal biopsy and antidiomyal antibodies in coeliac disease should be the subject of further discussion.

C B PEARCE H D DUNCAN D SINCLAIR D N POLLER
Department of Gastroenterology, Queen Alexandra Hospital, Portsmouth, UK
Correspondence to: C B Pearce, Department of Medicine, Queen Alexandra Hospital, Cosham, Portsmouth PO6 3LY, UK
callum@pearcey.screaming.net

Callum B Pearce is financially supported by Fresenius Ltd.

7 Miros M. High resolution upper endoscopy detects all patients with celiac disease macroscopically. Gastroenterology 2000;118(Suppl 2): abstract 3403.

Reply

EDITOR,—Pearce et al raise the possibility of using the endomysial antibody (EMA) test instead of histology of small bowel biopsies as a test for coeliac disease in the investigation of iron deficiency anaemia (IDA). They wish to consider this because of the considerable resource implications for histology departments.

The EMA test is excellent but its value is dependent on the prevalence of coeliac disease in the population being tested (the pre-test probability). Based on all peer reviewed published studies from 1985 to 1999, we have calculated specificity to be 98.4% (95% confidence interval (CI) 98.0–98.8) and sensitivity to be 93.8% (95% CI 92.7–94.9). 1 These give a likelihood ratio for a positive test of 59 and for a negative test of 0.06. We have found that the prevalence of coeliac disease in IDA is 4%. Thus using the Fagan nomogram, if the EMA test is positive, the post-test probability of coeliac disease is 75%—that is, one in four patients with IDA who have a positive EMA test will have normal histology on small bowel biopsy. If the EMA test is negative, the post-test probability is 0.2%, which effectively excludes coeliac disease.

We therefore agree that the EMA test could be used instead of small bowel biopsy to exclude coeliac disease in patients with IDA. However, as most of the costs of obtaining a histological diagnosis may be in the endoscopic examination (during which small bowel biopsies are taken by most gastroenterologists since we validated the technique in 1981) and as most patients will have an endoscopy anyway, it seemed reasonable to us to apply the definitive test (that is, histology) in all those under 45 years of age. For those not undergoing endoscopy, such as menstruating women under 45 years, we recommend the EMA test in our guidelines.

We agree that patchiness of mucosal abnormality, which we formally documented in 1976, needs to be taken into consideration and we routinely take at least four endoscopic biopsies. We realise that this may increase the cost of histology. If after a proper cost benefit study histology is found to be excessively expensive, consideration could be given to immediate stereomicroscopic assessment of biopsies which was popular in the 1960s and early 1970s, 8 and which we still apply when time allows. This is a very simple technique which allows visualisation at a glance of the whole surface of biopsies as well as helping to unfold and correctly orientate the specimens before fixation if histology is still deemed necessary. However, both this and high resolution endoscopy need to be applied for correct interpretation and would also need good quality photography for a permanent record if fixed biopsies were not stored.

In conclusion, we agree that the EMA test is a reasonable alternative for the investigation of coeliac disease in IDA but until we have a proper cost benefit study indicating otherwise, we recommend histology of endoscopic small bowel biopsies when endoscopy is already being undertaken.

B B SCOTT
Lincoln County Hospital, Lincoln LN2 4BJ, UK
dbbscott@ail.com

Is coeliac disease a confounding factor in the diagnosis of NASH?

EDITORS.—We read with great interest the paper by Wigg et al (Gut 2001;48:206–11) concerning the role of small intestinal bacterial overgrowth in the pathogenesis of non-alcoholic steatohepatitis (NASH) but we would like to comment on the enrolment criteria used for their study. We agree with Farrell (Gut 2001;48:148–9) that the Adelaide group’s failure to characterise the variables of obesity and diabetes in their population might result in selection bias. Moreover, they made no attempt to exclude the possibility of coeliac disease (CD) which can also be associated with altered intestinal permeability even when the disease is subclinical.

In approximately 40% of all adults with this disease, increased serum transaminase levels are found at diagnosis,1 and such elevations may be the only abnormality in cases of “occult” CD. In fact, in a study by Bardella et al, 9.3% of cases of unexplained chronic hypertransaminasaemia were ultimately diagnosed as CD.2 From December 1997 to December 1999, we observed 30 subjects (22 males, eight females; mean age 40 (9.3) years; mean weight 71.6 (7.9) kg) with clinical and laboratory pictures fully compatible with a diagnosis of NASH—that is, AST 56.3 (13.6) IU/l; ALT 102 (36.8) IU/l (normal 7–45); histological findings of macrovesicular steatosis, inflammation, hepatic fibrosis, and Mallory’s bodies; no history of alcohol consumption; and no other significant liver disease. All 30 patients had serum assays of IgG and IgA antibodies against gliadin and endomysium antibody (EMA), and duodenal biopsies were collected from those who were EMA positive. Four of these patients (one male and three females; mean age 30.6 (5.5) years) were thus diagnosed as having occult CD. The only clinical abnormalities were elevations in serum transaminase and sonographic evidence of fatty infiltration of the liver. All four were placed on a gluten free diet and followed with clinical examination and blood chemistry studies every three months. After three months on the prescribed diet, all patients presented decreases in serum transaminase levels (AST 29.6 (9.7) IU/l and ALT 23.6 (2.5) IU/l), duodenal histology has improved considerably, and there is no sign of steatosis on sonography.

CD may cause increased intestinal permeability3 and its clinical, biochemical, and histological findings are similar to those of NASH.4 The fact that elevated transaminase levels and EMA positivity can be documented even in the subclinical stages of CD suggests that the inflammatory process in this disease may be triggered by the same opportunistic agent identified by Farrell as a cause of tissue damage in NASH. In a recent study, Lahat et al. showed that CD is also associated with increased expression of inflammatory cytokines, including tumour necrosis factor α (TNF-α).

In the light of the findings reviewed here, we feel that all patients with unexplained hypertransaminasaemia should be screened for CD, and that CD must be excluded before the diagnosis of NASH is made.

G ARIEGO
L MIELE
G PIGNATARO
M POMPILI
G L RAPACCINI
G GASBARRINI
Institute of Internal Medicine, Policlínico Universitario A Gemelli,
Catholic University of Sacred Heart, Rome, Italy.

Correspondence to: Dr A Greico, Institute of Internal Medicine, Catholic University of Sacred Heart, Largo Gemelli 8—00168 Rome, Italy.
angreico@katamail.com


Induction of multiple autoantibodies to islet cell antigens during treatment with interferon alpha for chronic hepatitis C

EDITORS.—Induction or augmentation of autoimmunity during the treatment of chronic hepatitis C with interferon alpha is a well known phenomenon and a matter of great concern to physicians involved in the field of viral hepatitis. In recent years there have been a number of reports suggesting a link between the antiviral therapy and the development of antibodies to multiple autoantigens. In a recent issue of Gut, Wesche et al. (Gut 2001;48:378–83) described the appearance of antibodies to 21-hydroxylase, an autoantigen of the adrenal cortex, and autoantibodies to glutamate decarboxylase 65 (GAD65) and the tyrosine phosphatase IA2 (IA2), both important autoantigens with respect to the pathogenesis of autoimmune (type 1) diabetes. Autoantibodies to GAD65 and IA2 appeared during or after therapy with alpha interferon for chronic hepatitis C in 5/62 and 1/62 patients, respectively. However, none of these patients was positive for both antibodies.

Type 1 diabetes is regarded as a chronic autoimmune disease caused by selective destruction of the insulin producing β cells. The disease is mediated by T cells; autoantibodies are well established markers for an ongoing autoimmune process within the islets.1 As these autoantibodies usually appear prior to the clinical onset of the disease, they may be used to predict type 1 diabetes in predisposed individuals. In recent studies it has been shown that only those individuals in whom more than one diabetes related autoantibody could be determined are at considerable risk of developing type 1 diabetes.2 Overall, the risk of developing type 1 diabetes is directly correlated with the number of positive autoantibodies.3 Therefore, combined screening for diabetes related autoantibodies is suggested to increase the specificity and the positive predictive value of the autoantibody tests.

We studied 56 patients with chronic hepatitis C (defined by positive anti-HCV and positive HCV-RNA) for the appearance of diabetes related autoantibodies after interferon therapy. We first screened for islet cell antibodies by a nm-precipitation assay. A total of 11 autoantibodies (indirect immunofluorescence) and if positive additionally determined autoantibodies to GAD 65, IA2, and insulin (radioimmunoassay and ELISA, respectively). In case of positivity for any antibody we analysed a pretreatment serum sample to

www.gutjnl.com

Letters, Book reviews, Notices

Gut: first published as 10.1136/gut.49.4.596a on 1 October 2001.
Table 1 Characteristics of patients with diabetes related autoantibodies after interferon alpha therapy for chronic hepatitis C infection

<table>
<thead>
<tr>
<th>Patient No</th>
<th>Sex/age</th>
<th>Mode of infection/genotype of HCV</th>
<th>Date of interferon therapy</th>
<th>Diabetes related autoantibodies</th>
<th>Estimated risk for type 1 diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td>No 1</td>
<td>male/52 y</td>
<td>Unknown/genotype 1</td>
<td>8/98–9/99</td>
<td>IAA, GAD 65</td>
<td>Moderate</td>
</tr>
<tr>
<td>No 2</td>
<td>male/49 y</td>
<td>IV drug use/genotype 1</td>
<td>9/98–10/98</td>
<td>ICA, GAD, IA2</td>
<td>High</td>
</tr>
<tr>
<td>No 3</td>
<td>female/48 y</td>
<td>Blood transfusion/genotype 1</td>
<td>7/98–7/99</td>
<td>IAA</td>
<td>Low</td>
</tr>
<tr>
<td>No 4</td>
<td>female/41 y</td>
<td>Unknown/genotype 3</td>
<td>11/98–9/99</td>
<td>IAA</td>
<td>Low</td>
</tr>
</tbody>
</table>


Reinnervation following a diaphragm—a new paradigm for sensory bowel symptoms?

Visceral hypersensitivity has been identified as a significant feature in a proportion of patients with irritable bowel syndrome.

Reinnervation following a difficult intra-partum episode may be an important contributory factor. Many benign pelvic symptoms may be interpreted as pain or discomfort in response to touch (allodyniae or hyperalgesiae), including chronic pelvic pain, deep dyspareunia, urinary urgency, tampon discomfort, dysmenorrhoea, etc. Premature and prolonged maternal voluntary efforts in the second stage of labour may contribute to primary visceral denervation. Reinnervation has been demonstrated in the uterus, though an interval of five to ten years precedes the onset of sensory pelvic symptoms. Similar patterns of reinnervation have been demonstrated in the vulva and may occur in other pelvic viscera.

Acute or chronic pelvic pain is a common feature of gynaecological disease, but there is little understanding of the aetiologies, pathophysiologies, and effective treatments. The best management is the elimination of causative conditions. However, patients may continue to experience pain and discomfort and may find it difficult to identify the correct aetiology or the correct management.

MARTIN QUINN

Department of Obstetrics & Gynaecology, Hinchinbrooke Hospital, Huntingdon, PE29 6NT, UK
martin.quinn@hhb-tr.nhs.uk

Letters, Book Reviews, Notices

ICA, islet cell antibodies; GAD65, autoantibodies to glutamate decarboxylase 65; IA2, autoantibodies to tyrosine phosphatase IA2; IAA, insulin autoantibodies.

exclude the existence of any of these autoantibodies prior to interferon therapy.

We identified four patients with diabetes related autoantibodies after cessation of therapy with interferon alpha (table 1). None of the patients had any of these antibodies prior to antiviral therapy nor had they a positive family history for autoimmune diabetes. Patient Nos 3 and 4 only developed insulin autoantibodies at a low titre. Induction of antibodies to insulin is a known phenomenon during therapy with interferon alpha and is described in a frequent number of cases. All these patients have not developed an abnormal glucose tolerance test, this patient has a considerable risk of developing clinical overt diabetes. Therefore, if one autoantibody is present, those with more than one autoantibody are at a considerable risk of developing diabetes.

We have now followed the patient for 16 months after interferon therapy and he has not developed an abnormal fasting glucose or an abnormal glucose tolerance test. The predictive value of three positive autoantibodies was found in patient No 2. He developed three major autoantibodies during interferon therapy (GAD65, IA-2 and ICA). Based on the predictive value of three positive autoantibody tests, this patient has a considerable risk of developing clinical overt diabetes over the next years. To date (follow up for 12 months) he has not developed an abnormal fasting glucose or an abnormal glucose tolerance test. The situation in this patient is further complicated as he did not respond to the antiviral therapy and another course of interferon might further increase his risk of developing autoimmune diabetes.

In summary, different diabetes related autoantibodies can be induced during interferon therapy for chronic HCV infection. We propose that only those patients with more than one autoantibody are at a considerable risk of progressing to clinically overt disease. Therefore, if one autoantibody appears during antiviral therapy, follow up should include screening for all other diabetes related autoantibodies. Furthermore, the question of whether patients with multiple autoantibodies should be retreated with interferon remains unsolved. Here the physician has to weigh possible progression of liver disease against the possibility of inducing autoimmune diabetes.

**BOOK REVIEWS**


If asked to supply a subtitle for this excellent eminently readable book, I would suggest “All you ever wanted to know about the basics of colorectal cancer but were afraid to ask a non-surgeon” (then assumed that people would expect you to know already). The chapters are written by experts renowned in their field and each topic is dealt with comprehensively and clearly with the reader being led from one line of argument to the next in a satisfyingly logical way. Although aimed at the clinician rather than the scientist, where necessary the scientific evidence is given in sufficient detail without overwhelming the non-expert with technical jargon. Similarly, in this era of evidence based medicine, the authors are to be congratulated on choosing judiciously the most relevant clinical trials which help explain the development of currently accepted clinical practice. Areas of controversy (such as the use of total mesorectal excision (TME) in rectal cancer) are presented in a fair and (largely) unbiased way.

Perhaps a few more figures or diagrams might have been useful, although the easily readable text makes this less of a problem than it might have been (I assume the labeling of the first two figures in the chapter on adjuvant chemotherapy which does not correspond with the text will have been corrected by the time the book is published).

There are a few minor quibbles concerning details of individual chapters. The statement that in rectal cancer the use of TME to reduce local recurrence rates to less than 10% precludes the need for adjuvant therapy—adjuvant chemotherapy is still indicated in these patients. The chapter on surgery for rectal cancer in metastatic disease deals mainly with local recurrence: it would have been interesting to have greater discussion of the role of surgery for hepatic and pulmonary metastases. A short historical introduction to the Dukes’ staging system (although well known) and its modifications (perhaps not so well known) would have been appreciated in the chapter on pathology. Some readers may find a little too much technical detail in the otherwise excellent chapter on radiotherapy.

The authors of the chapter on adjuvant chemotherapy may have changed their emphasis on radiotherapy as the “prime adjuvant weapon” in rectal cancer if they had known the results of the recently published NSABP-R02 trial which showed that radiotherapy seems to be more useful at reducing local recurrence rates (which is probably more of a problem in the subgroup of patients with positive circumferential margins or who undergo suboptimal surgery) rather than improving overall survival: the clinical choice for adjuvant treatment in these patients is probably between chemotherapy alone or combined chemoradiation.

The chapter on future directions misses the opportunity to discuss in greater detail the potential of the new drugs irinotecan, oxaliplatin, and the oral fluoropyrimidines. A separate chapter on imaging techniques might be considered for the next edition, in the context of the increasing interest in virtual colonoscopy and the value of MRI in helping to determine the resectability of rectal tumours.

I am somewhat loath to mention these points as they are probably a reflection of individual opinion and certainly should not detract from this valuable book, which would be an excellent introduction for a registrar or SHO plunged into the field and expected to be a knowledgeable expert in the outer. Likewise, as the relevance of a multidisciplinary approach to colorectal cancer becomes increasingly important, it is essential that the specialist in one discipline keeps up to date with the current thinking in related specialties: a function which this books serves admirably.

M ALLEN


This is a monumental effort and the editors, who have written much of this textbook themselves, are to be congratulated on a terrific job. They have taken on the task of reworking the iconic textbook first started by the late and great John Goligher. His was a very personal style, with repeated references to his own practice and results, and then a weighing of the evidence and a firm opinion, an approach which was much loved. With the growing emphasis on a larger body of evidence, the editors have widened their search in the world’s literature and updated it for the now established specialty of colorectalology which tends to be surgical but spans the many disciplines looking after patients with diseases of the lower gastrointestinal tract. And here, despite the view that electronic publishing will consign many textbook to history, is a second edition, tribute to its success and what Vie Fazio in his foreword calls the perspective “thing”. There must still be a place for one or two prominent and authoritative textbooks in any specialty provided they are kept up to date, and I have no doubt this will continue to be just such a tome.

There is much here which will be of interest to gastroenterologists as well as surgeons. If you want to argue the point with your local surgeon, give a talk which includes some surgical outcomes, write a medicolegal report, or find a reference, it is all there. The style of the previous edition has been retained, with clear text, excellent line drawings, and helpful tables. The bibliography arranged at the end of each section is exhaustive and comprehensive. New sections have been added on medi- cical management of anal fissure, never treat- ments for incontinence, and the combi- nined management of pelvic floor disorders.

In inflammatory bowel disease, the place of newer therapies has been discussed, and pouch salvage procedures are reviewed. Cancer has been updated with additions to the whole area of biology including hereditary and polyposis syndromes, as well as adjuvant therapy. The chapter on laparoscopic surgery in colorectal cancer which includes length of stay and cost issues. The recent pronouncement of the NICE in concluding that open surgery still has room for improvement, and until specialisation and quality improve these results and there are clear advantages, laparoscopic will remain in abeyance outside clinical trials. A good examination of the place of local excision includes transanal endoscopic microsurgery but with so many surgical staff carrying out colonscopy with specialisation, the technique was rather short and the newer approaches to polypectomy, including endoscopic mucosal resection, and placement of clips and ligatures, and tattooing was disappointing.

Inevitably there are some gaps. Although the place of nitrites is reviewed for the treatment of fissure, the more recent introduction of calcium channel blockers and a growing disinterest in TTS with GYN specialists and the management of Crohn’s fistulae, anti-tumour necrosis factor may have a profound impact on surgical practice but this too did not reach the cut. And the controversial new stapling procedure for haemorrhoids arrived too recently for inclusion.

These are minor criticisms however. Although it comes in two hefty volumes and at a similarly weighty price, it is clear this is not a book to be read from cover to cover. It is in my view the finest reference textbook on the subject on both sides of the Atlantic, and will be taken frequently and enjoyably from the bookshelves in offices and studies of those who really care deeply about the management, medical and surgical, of these embarrassing, distressing, and challenging conditions. The owners will just have to make sure the books are returned.

N MORTENSEN


The choice of title for a book can make or break its sales. Despite any caution or maturity of judgement we may claim, it seems we are all at risk of being seduced by books with titles such as “Improve your golf”, or “Make the stock market work for you”. Read the book and find the success/wealth/glamorous lifestyle or whatever that has eluded you up to now. Practical management of oesophageal disease is in this mould—the title hints not at a glamorous lifestyle admittedly, but at delivering the easy answers gastroenterologists would like to find for all oesophageal problems. And of course it does not deliver them. However, if expectations are tempered to the real world rather than fantasy, this is a useful book, written by people who know their subject, describing what they do and why.

In 12 chapters, the 20 authors give their own perspectives on the major topics in oesophageal disease. There is a surgical bias (for example, medical management of gastro-oesophageal reflux disease is covered in two

www.gutjnl.com
Celiac Disease: Methods and Protocols

Michael Marsh’s latest book on celiac disease started out at a distinct disadvantage as I took it with a pile of novels to read while on a summer holiday in Provence. However, he need not have worried; this is a distinctive book and, to someone interested in celiac disease, it came as a refreshing approach to the subject.

Marsh was charged with editing a volume in a new series on “Methods in Molecular Medicine”. He was a good choice as he has spent virtually the whole of his professional life working on celiac disease and, in particular, on the morphology of the intestinal mucosa. He has dedicated the volume to the late Anne Ferguson, who did much to further our understanding of mucosal immunity, particularly in respect of celiac disease.

The book starts with a good introductory chapter that clearly states the modern understanding of gluten sensitivity and clinical features of the disease. There are three chapters outlining genetic methods, ranging from DNA extraction through positional cloning to complex family studies. These describe the techniques, as used in Richard Houlston’s laboratory, and are comprehensive with informative notes. However, they are not for the uninitiated in this area.

Tatham and colleagues have produced a good and easily accessible description of the modern classification of cereal proteins, as well as a detailed account of extraction, separation, and purification processes. Koning’s group has written three excellent complementary chapters on characterising gluten peptides using mass spectrometry, on producing synthetic peptides for T cell recognition, and on identifying specific peptide binding regions on HLA-DQ molecules. Ludvig Sollid’s group follows this with another excellent chapter on the establishment and use of gladin specific T cell lines and clones. This is written by obvious leaders in this field and the accompanying notes reveal a wealth of useful technical detail for the budding researcher.

Marsh’s group is responsible for two chapters on morphometry. The first is probably the best account yet that they have produced on their methods of morphometric analysis of small intestinal mucosa. The second chapter describes their methods for detecting changes in gluten sensitive rectal mucosa. These chapters are didactic and reflect the many years of careful observations made by Marsh and his coworkers. One wonders whether there is anything further to be measured in the small intestinal mucosa, but it is useful to have such a comprehensive account of these methods.

Riccardo Troncone and his colleagues describe the use of in vitro rectal gluten challenge as a means of studying immunological phenomena in celiac disease. These are interesting observations but of limited usefulness. It is disappointing that the immune morphometrical methods of Marsh are not used in these studies. I was sorry not to see a description of organ culture of small intestinal mucosa and the possibilities arising from it.

Paul Ciciolina’s laboratory has a long-standing interest in in situ hybridisation techniques and there is a good account of these from workers with first hand knowledge of the problems and pitfalls, as well as the usefulness. Per Brandtzæg is the acknowledged guru of gastrointestinal mucosal immunohistochemistry. His group provide two chapters, one on quantitative polymerase chain reaction for the assessment of cytokine mRNA expression, and a comprehensive one on immunohistochemistry. These chapters are extensive and detailed, with useful practical notes from an expert. Dietrich (on an ELISA for tissue transglutaminase antibodies) and Unsworth (on routine serological tests) provide two practically useful chapters for more routine diagnostic laboratories. Finally, the Edinburgh group, founded by Anne Ferguson, describes the technique and uses of whole gut lavage fluid analysis.

Marsh has assembled a group of international authorities on the various aspects he has chosen. The book achieves the aim of providing good coverage for many of the research techniques currently used in celiac disease. One wonders about the readership of such a volume. I suspect it will be quite specialised for those who, as Marsh says (page 8) want to “plunge into this complex pool of intrigue, this book should provide good introductory exposure”. Ah well! From the wizardry of Marsh and colleagues, I return to the witchcraft of Harry Potter.

P D HOWDLE
EASL Single Topic Conference
The EASL Single Topic Conference “Liver fibrosis: from basic science to clinical targets” will be held on 12–13 October 2001 in Florence, Italy. Organisers: Massimo Pinzani (University of Florence) and Detlef Schuppan (University of Erlangen-Nuernberg). The aim of the conference is to provide the latest information on this key area of hepatology and to translate the current knowledge into clinical terms. It is directed at both the expert in the field and the general hepatologist. Further information: Massimo Pinzani, Dipartimento di Medicina Interna, Università degli Studi di Firenze, Viale GB Morgagni, 85, I-50134 Firenze, Italy. Tel: +39 055 4277845; fax: +39 39 055 417123; email: m.pinzani@dfc.unifi.it

Lecture Course in Coloproctology
This course will be held on 15–17 October 2001 in Harrow, UK. Professor Russell Stitz from Australia will be the Sir Alan Parks Visiting Professor and, for the first time, there will be a Sir Francis Avery Jones Visiting Professor, which will be Professor Paul Rutgeerts from Belgium. Further information: The Administrator, St Mark’s Academic Institute, St Mark’s Hospital, Northwick Park, Harrow, Middx, HA1 3UJ, UK. Tel: +44 (0)20 8235 4046/8; fax: +44 (0)20 8235 4039; email: stmarks@ic.ac.uk; website: www.stmarkshospital.org.uk

International Symposium on Hyperammonemia, Liver Failure and Encephalopathy
This symposium will be held on 20–22 October 2001 in Valencia, Spain. Further information: Cátedra Santiago Grisolía, Fundación Museu de les Ciències Príncipe Felipe, Ciutat de les Arts i les Ciències, Avda. Instituto Obrero, s/n, 46013 Valencia, Spain. Tel: +34 96 197 44 66; fax: +34 96 197 44 70; email: catedrasg@cac.es. Deadline for submission of abstracts is 31 May 2001.

ICGH-2: The Second Iranian Congress of Gastroenterology and Hepatology
The main Iranian meeting of gastroenterologists and researchers in this field will be held on 27 October to 1 November 2001 in Tehran, Iran. Further information: Dr Shahin Merat, Digestive Diseases Research Center, Shariati Hospital, N. Kargar Street, Tehran 14114, Iran. Tel: +98 911 717 3966; fax: +98 21 225 3635; email: merat@ams.ac.ir; website: www.ams.ac.ir/icgh. Deadline for submission of abstracts is 31 May 2001.

Falk Symposium No 127: Autoimmune Diseases in Pediatric Gastroenterology
This Falk Symposium will be held on 8–9 November 2001 in Basel, Switzerland. Further information: see Falk Symposium No 126 above.

42nd Annual Conference of the Indian Society of Gastroenterology
This conference will be held on 23–29 November 2001 in Lucknow, India. The programme includes two pre-conference symposia (on gastrointestinal motility and scientific communication, on 23 November), a one day postgraduate course or CME (24 November), and an endoscopy workshop (28–29 November). Further information: Dr S R Naik, Department of Gastroenterology, SGPGI, Lucknow 226014, India. Tel: +91 522 440700 or 440800, ext 2400; fax: +91 522 440078 or 440017; website: www.sgpgi.ac.in/conf/ig2001.html

41st St Andrew’s Day Festival Symposium on Therapeutics
This will be held on 6–7 December 2001 in Edinburgh, UK. Further information: Ms Eileen Strawn, Symposium Co-ordinator. Tel: +44 (0)131 225 7324; fax: +44 (0)131 220 4393; email: e.strawn@rcpe.ac.uk; website: www.rcpe.ac.uk

14th Intensive European Course of Digestive Endoscopy
This course will be held on 17–18 December 2001 in Strasbourg, France. Further information: Michele Centonze Conseil, 6 bis rue des Cordiers, 75020 Paris, France. Tel: +33 1 44 62 68 80; fax: +33 1 43 49 66 58; email: mail@m-centonze-conseil.com