LETTERS

To perform or not to perform liver biopsy: an alternative view

I would like to thank Joy and Scott for their comments in their letter in response to my review (Gut 2002;51:9–10). I entirely agree with their view that ultrasound is highly specific and sensitive for the diagnosis of fatty liver. However, I do not feel that the presence or absence of fatty liver is the issue here. It is established that approximately 30% of patients with fatty liver who have significant fibrosis will go on to develop chronic liver disease and cirrhosis, with all its complications, including hepatoma. The purpose of historical sampling is not to confirm the presence of fatty liver but to see whether fibrosis and other abnormalities are present, putting the patient at risk of developing chronic liver disease.

This issue was addressed in a recent article by Saadeh and colleagues who compared patients with non-alcoholic steatohepatitis (NASH) and those with steatosis (non-alcoholic fatty liver disease (NAFLD)) alone. The authors evaluated the role of various radiological modalities, including ultrasound, computed tomography, and magnetic resonance imaging, in the role of distinguishing NASH and the less aggressive forms of NAFLD. Their conclusion was that none of the radiological modalities detected the presence of hepatocyte ballooning, Mallory’s hyaline, or fibrosis, which are the important features in the diagnosis of NASH. The study showed that ultrasound had high sensitivity and specificity for the diagnosis of severe steatosis but confirmed that ultrasound had no predictive value in the diagnosis of fibrosis or cirrhosis.

On the basis of this article together with earlier studies, I can find no basis for the conclusion reached by Joy and Scott that ultrasound is a reasonable alternative to liver biopsy for patients who have abnormal liver function tests with no diagnostic serology.

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References


Non-medical treatment of GORD

The interesting comment of Heading (Gut 2002;50:592–3) on the work by Flibbe et al needs to be completed by considering the anatomical factor in the study of gastro-oesophageal reflux disease (GORD) before advocating any non-medical appropriate treatment (fundoplicator, Stratten procedure, Gastropexie, etc.). The multiple controversies arising from all non-medical proposed treatments, with contradiictory results, are due to the complete neglect of delineating the cardio-oesophageal junction (CEJ) and the shape of the angle of His, and the role of the anatomical factor in selecting the correct candidate for successful non-medical treatment.

The new generation of gastrointestinal specialists, who come after the endoscopy era, are not aware of the radiology of the gastro-intestinal tract, particularly when we need to have the anatomical configuration of the CEJ.

Gastric physiology and junction motility are the next step in evaluating any case of GORD. Ignoring the anatomical shape of the CEJ is behind the various conflicting results that we are hearing at medical meetings devoted to GORD.

Imposing the study of the anatomical feature of the junction, which is very variable from person to person, is the first step in evaluating any proposed treatment of GORD, medically or surgically.

Applying the devices (Plicator, Stratten procedure, etc) without studying the anatomy of the junction is behind the side effects of these proposed procedures.

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Association between K469E allele of intercellular adhesion molecule 1 gene and inflammatory bowel disease in different populations

We read with interest the article by Matsuzawa et al showing an association between the K469E allele of intercellular adhesion molecule (ICAM)-1 gene and inflammatory bowel disease (IBD) in a Japanese population (Gut 2003;52:75–8). The ICAM-1 gene lies on chromosome 19p13, previously implicated in determining susceptibility to IBD, and codifies for a surface glycoprotein that belongs to the immunoglobulin superfamily. ICAM-1 plays an important role in the trafficking and activation of leucocytes and is upregulated in the inflamed mucosa of IBD patients. Matsuzawa et al found that the allelic frequency of K469 was significantly higher in both Crohn’s disease (CD) and ulcerative colitis (UC) patients and healthy controls. The G241R polymorphism of the ICAM-1 gene was also investigated in these studies, and IBD patients were stratified by antineutrophil cytoplasmic antibody (ANCA) status. In particular, Yang et al found a significantly increased frequency of the G241R polymorphism both in ANCA negative UC and ANCA positive CD patients’ while Braun et al showed an association between R241 and UC, independently of ANCA status.1

We also searched for the K469E mutation in 42 consecutive Italian IBD patients (31 males, mean age 36 (14) years), 17 with CD and 25 with UC, and 227 ethnically matched controls. Our preliminary results (see table 1), although obtained in a limited number of patients, are in contrast with the findings of Matsuzawa et al (Gut 2003;52:75–8) and confirm those obtained in Caucasians patients.2–4 The possible explanation is that the frequency in results is the influence of the different geographic distribution of the genetic mutation. Japanese patients with IBD have a genetic background that differs from Western patients, as also demonstrated recently for the NO2/CARD15 gene polymorphisms. Indeed, several studies have reported an association between CD and NO2/CARD15 mutations in Caucasians but not in Japanese cohorts.5–7 These data indicate that there may be significant genetic heterogeneity between different ethnic and racial IBD populations and environmental factors may play a leading role in the pathogenesis of IBD. Thus gene-environment interactions represent a crucial event in the pathogenesis of IBD and they cannot be considered as distinct entities.

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Table 1 Allicic frequencies of the E/K469 ICAM-1 polymorphism in Italian patients affected by IBD, and in controls

<table>
<thead>
<tr>
<th>Allelic frequency (%)</th>
<th>Controls (n=227)</th>
<th>IBD (n=42)</th>
<th>UC (n=25)</th>
<th>CD (n=17)</th>
</tr>
</thead>
<tbody>
<tr>
<td>E469</td>
<td>45</td>
<td>45</td>
<td>46</td>
<td>44</td>
</tr>
<tr>
<td>K469</td>
<td>55</td>
<td>55</td>
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<td>56</td>
</tr>
</tbody>
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IBD, inflammatory bowel disease; UC, ulcerative colitis; CD, Crohn’s disease; ICAM, intercellular adhesion molecule.

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References

Authors’ reply
We would like to address the possible cause of the inconsistency of the ICAM1 alleles, which associated with inflammatory bowel disease (IBD), between Japanese (K469) (in association with inflammatory bowel disease ICAM1) the inconsistency of the associations there may be different linkage disequilibrium in this region (chromosome 19p13).

The third explanation is that these inconsistencies may be due to sample size and/or inappropriate control populations. As the ICAM1 polymorphism consists of only two SNPs and these polymorphisms are less informative, a considerable volume of sample size and simple composition of haplotype in the ethnic group would be required to detect the increase in the predisposing haplotype.

In conclusion, this inconsistency of the associated ICAM1 allele is most likely due to the difference in linkage disequilibria of the ICAM1 allele to the DPM in 19p13 among ethnic groups. Haplotype-positive associations between the ICAM1 polymorphism and IBD in these ethnic groups highlights the potential importance of this region in the search for the predisposing gene(s) to IBD.

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References

Eosinophilic oesophagitis: treatment using Montelukast
I read with interest the paper by Atwood and colleagues (2003; 52: 181–5) on eosinophilic oesophagitis (EO). According to the authors, the distinct clinical syndrome of EO is not usually seen either as a component of gastro-oesophageal reflux disease or as a variant of eosinophilic gastroenteritis (EG).

The diagnostic hallmark of EO is odynophagia and the diagnosis is always histology dependent (>20 eosinophils/high power field) (Atwood 2003; 52: 181–5). In the paediatric setting, the condition is widely recognised but the adult EO may escape diagnosis due to general lack of awareness of the condition. In this respect, the paper by Atwood and colleagues (2003; 52: 181–5) is a valuable contribution towards understanding the complex pathophysiology of EO.

The pathophysiology of EG or EO may be similar to that of asthma. Asthmatic patients demonstrate increased production of cytokine leukotrienes during acute asthma attacks.5 CysteinyI leukotrienes have potent chemotactic and inflammatory properties for eosinophils and play an important role in the pathophysiology of asthma.

In EG, accumulated eosinophils cause severe tissue damage characteristic of EG. CysteinyI leukotrienes, along with cytokines interleukin 3 and 5, and growth factor-macrophage-stimulating factor play a role in the recruitment of eosinophils into the tissue causing the damage.

No controlled treatment trial for EG or EO exist. Limited results have been achieved with oral cromolyn, ketotifen, and other antihistamines. Oral corticosteroids are effective but long term use is complicated by side effects.

Montelukast is a leukotriene receptor antagonist (LTRA) which acts to bind and selectively blocks the leukotriene D4 (LTD4) receptor. Because LTD4 is both a pro-inflammatory and chemotactic factor for eosinophils, this may provide the rationale for treating a patient with EG or EO with a LTRA. The first reported case of successful Montelukast treatment for CSS in a young EG patient was published in 1999.7 Montelukast was originally licensed in the UK for use in asthma.8 There has been some concern regarding association between the use of LTRA and Churg-Strauss syndrome (CSS) in asthma.9 CSS is a rare form of eosinophilic vasculitis associated with asthma. This syndrome has previously been associated with the use of Zafirlukast.1 The Committee on Safety of Medicines has received 12 reports of CSS and pulmonary eosinophilia possibly associated with Montelukast.1 There are other reports of Montelukast induced CSS in asthma patients in the literature.

Atwood and colleagues (2003; 52: 181–5) observed nausea in four patients and myalgia in one in the Montelukast group but there was no mention of CSS. In the previous report of Montelukast therapy in EG, it was shown that Montelukast did not reduce peripheral eosinophilia (TE) or symptoms in a patient with severe EG complicated by oesophageal stricture. In another report, Montelukast reduced eosinophilic oesophagitis but there was no mention of whether TE was reduced.1 In Atwood et al’s paper (2003; 52: 181–5), treatment with Montelukast for a median


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Effect of a rapid access flexible sigmoidoscopy clinic on the yield of early stage rectal cancer

We read with interest the debate on population based endoscopic screening for colorectal cancer (Gut 2003;52:323–6). While we agree that the case for population screening is compelling, we believe that Macafee and Scholefield’s statement that “earlier diagnosis is pelling, we believe that Macafee and Scholefield’s statement that “earlier diagnosis is pelling, we believe that Macafee and Scholefield’s statement that “earlier diagnosis is pelling, we believe that Macafee and Scholefield’s statement that “earlier diagnosis is pelling, we believe that Macafee and Scholefield’s statement that “earlier diagnosis is pelling, we believe that Macafee and Scholefield’s statement that “earlier diagnosis is pelling, we believe that Macafee and Scholefield’s statement that “earlier diagnosis is pelling, we believe that Macafee and Scholefield’s statement that “earlier diagnosis is pelling, we believe that Macafee and Scholefield’s statement that “earlier diagnosis is pelling, we believe that Macafee and Scholefield’s statement that “earlier diagnosis is pelling, we believe that Macafee and Scholefield’s statement 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Collagenous colitis: constipation or diarrhoea?

As an axiom, collagenous colitis is characterised by diarrhoea, lymphocytic inflammation, and a thickened subepithelial collagen layer in the colorectal mucosa. Various case presentations in the literature have reported that frequent white needle pricks on the submucosal layer of collagenous colitis and non-collagenous inflammatory disease are non-specific and can be used to prove this hypothesis.

Our recent study investigated 32 patients with histologically identified collagenous colitis. In contrast with the current definition, all had chronic constipation and only 14 had the well-known diarrhoea. We also treated all of them with budesonide (Budesonid, Dr Falk Pharma) and patients receiving budesonide had a clinical response: stool frequency and stool weight were significantly reduced. A correlation between the grade of inflammation as well as collagen layer thickness and clinical picture of collagenous colitis and food allergy. The high frequency of specific antibodies to food antigens in patients with collagenous colitis might be related to food allergy. Patient sera were analysed for common food antigens. Our data support the hypothesis that patients with collagenous colitis have laboratory and clinical evidence of food allergy: the high frequency of specific antibodies to food antigens and the increased total IgE levels imply a possible connection between collagenous colitis and food allergy and suggest a possible reason for the paradox of diarrhoea constipation. Corticosteroids are the most effective drugs available for the treatment of allergic diseases and are very useful in treatment because they have potent anti-inflammatory effects.

References

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References

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Hepatology: a Textbook of Liver Disease, 4th edition


The fourth edition of Zakim and Boyer’s Hepatology: a Textbook of Liver Disease is published this year and significantly updates the previous edition published in 1996. The work once again comes in two volumes and is written by 130 contributing authors, the majority of whom are from the USA. The book is arranged into four sections. Sections 1–4 are in volume 1 and cover cell biology, biochemistry, and physiology (section 1); the systemic effects of liver disease (section 2); and laboratory methods for evaluating liver disease (section 3). The whole of volume 2 is taken up by section IV covering aetiology, clinical features, diagnosis, and treatment of specific liver diseases subdivided into toxic injury, infection, chronic liver disease, tumours, childhood liver disease, diseases of the biliary tree, and special topics.

The approach works well and all the relevant areas are comprehensively covered. There is inevitably some duplication between sections but this is kept to a minimum. Placing most of the basic science in the first volume allows readers who want to concentrate on specific diseases to do so easily and then to refer to the first volume if they need further background. There are some inconsistencies in this approach, for instance clinical aetiology is included in section IV “Diagnosis and management of chronic forms of liver disease”. Given the major physiological role of the liver as an immune organ and
the fact that many acute liver diseases have an immunological basis, it might have been appropriate to highlight immunology in volume 1 where it would fit very well with the excellent chapters on hepatic regeneration and fibrosis. Section 3 contains an informative chapter on laparoscopy but only a four page section on hepatic imaging. Imaging is subsequently covered in the individual chapters in section IV but given the major advances in interventional radiology and imaging it would have been appropriate to give a chapter of its own. For example, there are only two brief references to positive emission tomography in the whole book.

Individual chapters are extremely well referenced although it might help to highlight the most significant references or those that provide an in depth review. One minor criticism is the quality of some of the figures. The chapters are richly illustrated but there is an irritating variation in the quality and style of the line drawings. It would have improved the overall appearance of the book if figures had been redrawn in a uniform style, and for some of the figures this would also have improved their clarity. The reluctance to use colour presumably based on cost considerations. However, the recently published Comprehensive Clinical Hepatology edited by O’Grady, Lake, and Howdell (Mosby), provides an example of how the use of modern technology can provide outstanding illustrations that enhance the readability of the book.

How does Zakim and Boyer compare with other similar volumes? The two main rivals are the Oxford Textbook of Clinical Hepatology and Schiff’s Diseases of the Liver, both of which were last revised in 1999. All three works are excellent. There are some differences in emphasis and presentation between them but all three are highly readable and cover the field comprehensively. I have greatly enjoyed having access to Zakim and Boyer over the last few weeks and would recommend the fourth edition unreservedly to anyone with an interest in liver disease, whether research scientists, specialist hepatologists, or gastroenterologists. The editors are to be congratulated for managing to improve an already outstanding reference work.

D H Adams

Genetic Disorders of the Exocrine Pancreas


This multiauthor work, derived from a symposium held in April 2001, summarises our current knowledge of the genetics of exocrine pancreatic disease. As is usual with such publications, the individual chapters have been written as free standing presentations which results in a degree of repetition. The editors have organised the chapters into sections; a “consensus conference” dealing with ethical issues and with guidelines for prevention, screening, and treatment is followed by sections on hereditary pancreatitis (HP), pancreatic cancer, and cystic fibrosis. Finally, there is a conference report and a monograph celebrating the work of Henry Lynch of the Iowa Pancreatic Tumor Registry. The first section will be of the most practical interest to general gastroenterologists. Gastrointestinal physicians and surgeons should already be aware of the ethics of screening from more common conditions such as colon cancer. A brief overview of these issues is followed by advice on whom to test, appropriate pre-test information, post-test information, and acting on the results. The algorithm looks forbidding but is straightforward in concept and logical. Both service and research testing are covered. Screening for cancer in HP is a more difficult issue. The advice that patients over 40 years should have annual screening at a specialist centre however may build a knowledge base for future patients but will not necessarily help our current population. The HP section suffers from its symposium origins. In the six years since David Whitcombe and his colleagues identified a mutation of the PRSS1 (cation trypsinogen) gene in a kindred with HP a new understanding of the relationship between the three entities of acute and chronic pancreatitis and pancreatic cancer has developed. It is a pity that the format of this book has not allowed this to be made explicit. It starts well with a two page overview neatly logging the known PRSS1 and SPINK mutations. Unfortunately, the chapters dealing with the mechanisms by which these mutations might translate into clinical disease, which logically should follow, are instead towards the end of the section (although an earlier chapter on gene mutations in children does cover some of this ground). The general reader might well find the intervening chapters on national experiences superficial. The review of inborn errors of metabolism is useful but surely belongs at the end of the section rather than in the middle.

The pancreatic cancer section is better organised and provides a comprehensive review of the contribution of genes to disease. In contrast with HP, there are no defined single gene mutations identified for pancreatic cancer. Families with two or more affected members have been reported: whether this simply represents random clustering is discussed. The risk of pancreatic cancer in accepted cancer syndromes such as FAMMM, Lynch syndrome II, BRCA2 mutation, and Peutz-Jehgers syndrome is also considered. The chapters on surveillance and molecular diagnosis will be of particular interest as they offer the first glimpse of hope for early detection and treatment. Non-pancreatologists may not be aware that premalignant ductal lesions termed PanINs have now been described and classified. Ways in which this discovery and molecular markers such as K-ras mutations may be exploited in screening strategies are reviewed, as are the practical difficulties of assessing the pancreas even with the benefit of EUS and CT. Ultimately however, all strategies being considered or tried will lead to a prophylactic pancreactectomy: the practicalities of the timing and extent of pancreactectomy are covered in the final chapter.

The final section covering cystic fibrosis and the Shwachman-Diamond syndrome is limited in its scope and appears to reflect one centre’s interests and research. Currently, pancreatic disease is either self limiting or incurable. Effective treatments for pancreatitis and pancreatic cancer will come from molecular and genetic research of the type described here. The presentation of this book is not perfect but the information it contains should be available to anyone dealing with pancreatic disease. Ask your library to buy it.

D Fine

CORRECTIONS

In the BSG Abstracts supplement, there was an error in abstract 179 by Li et al (Gut 2003;52 Suppl I:A44). In the revision of this abstract the sentence after the table should read “1 year survival for all patients with and without pre-existing Barrett’s was 51.5% and 31% respectively, and for those undergoing potential curative resection, was 72.6% and 52.7% respectively”. The authors apologise for the error.

In the author index of the BSG Abstracts supplement, J E Crabtree should have been listed as an author on abstract 126 by Jeremy et al (Gut 2003;52 Suppl I:A24). This was due to a technical error for which the journal apologises.

NOTICES

British Society of Gastroenterology Sir Francis Avery Jones Research Award 2004

Applications are invited by the Education Committee of the British Society of Gastroenterology who will recommend to Council the recipient of the 2004 Award. Applications (TWENTY COPIES) should include:

• A manuscript (2 A4 pages ONLY) describing the work conducted
• A bibliography of relevant personal publications
• An outline of the proposed content of the lecture, including title
• A written statement confirming that all or a substantial part of the work has been personally conducted in the UK or Eire.

Entrants must be 40 years old or less on 31 December 2004 but need not be a member of the Society. The recipient will be required to deliver a 30 minute lecture at the Annual meeting of the Society in Glasgow in March 2004. Applications (TWENTY COPIES) should be made to the Honorary Secretary, British Society of Gastroenterology, 3 St Andrews Place, London NW1 4LB by 1 December 2003.

British Society of Gastroenterology Hopkins Endoscopy Prize 2004

Applications are invited by the Endoscopy Committee of the British Society of Gastroenterology who will recommend to the Council the recipient of the 2004 Award. Applications (TEN COPIES) should include:

• A manuscript (2 A4 pages ONLY) describing the work conducted
• A bibliography of relevant personal publications
• An outline of the proposed content of the lecture, including title
• A written statement confirming that all or a substantial part of the work has been personally conducted in the UK or Eire.

An applicant need not be a member of the Society. The recipient will be required to deliver a 20 minute lecture at the Annual meeting of the Society in Glasgow in March 2004. Applications (TEN COPIES) should be made to the Honorary Secretary, British Society of Gastroenterology, 3 St Andrews Place, London NW1 4LB by 1 December 2003.
European Helicobacter Study Group (EHSG)

This meeting, on Helicobacter infections and gastroduodenal pathology, will be held on 3–6 September 2003 in Stockholm, Sweden. Further details: Professor Torkel Wadstrom, President-EHSG, Lund University, Department of Infectious Diseases & Medical Microbiology, Division of Bacteriology, Solvegatan 23, SE-223 62 Lund, Sweden. Tel: +46 46 173 241; fax: +46 46 152 564; email: Torkel.Wadstrom@mmb.lu.se; website: www.helicobacter.org

Falk Symposium

135—Immunological Diseases of Liver and Gut

This symposium will be held on 12–13 September 2003 in Prague, Czech Republic. Further details: Falk Foundation e.V., Congress Division, PO Box 6529, Leinenweberstr. 5, 79041 Freiburg/Br, Germany. Tel: +49 761 15 140; fax: +49 761 15 14 359; email: symposia@falkfoundation.de; website: www.falkfoundation.de

The European Society of Parenteral and Enteral Nutrition (ESPEN)

ESPEN will celebrate its silver anniversary at the time of the annual congress, which is to be held on 20–23 September 2003 in Cannes, France. Further details: www.espen.org

XII Falk Liver Week

The XII Falk Liver Week, in honour of Hans Popper's 100th birthday, will be held on 15–22 October 2003 in Freiburg, Germany. Further details - see Falk Symposia above.

European Course on Laparoscopic Endoscopy

This course will be held on 18–21 November 2003 in Brussels, Belgium. Further details: Secretary to Professor Cadière, Service de Chirurgie Digestive, Rue Haute 322, Brussels 1000, Belgium. Tel: +32 (0)2 648 07 60; fax: +32 (0)2 647 86 94; email: straeb.asmb@proximedia.be; website: www.straeb-asmb.com

4th Nutrition and Health Conference

A multidisciplinary event will be held on 21–22 November 2003 in London, UK. This year's topics include cancer, obesity, exercise on prescription, menopause, ageing, motivation skills, and coronary heart disease. Further details: Tanya Carr, 16 Brownlow Court, Lyttelton Road, London N2 0EA. Tel/fax: +44 (0)208 455 2126 or 6570; website: www.nutritionandhealth.co.uk

Hong Kong-Shanghai International Liver Congress 2004

This conference will be held on 14–17 February 2004 in Hong Kong. The topic of the conference is “Liver Diseases in the Post-Genomic Era”. Further details: Ms Kristie Leung, Room 102–105 School of General Nursing, Queen Mary Hospital, 102 Pokfulam Road, Hong Kong. Tel: +852 2818 4300/8101 2442; fax: +852 2818 4030; email: kristieleung@hepa2004.org; website: www.hepa2004.org

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To perform or not to perform liver biopsy: an alternative view

R W Chapman

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