Osteoporosis and liver disease: additional reasons for coeliac disease screening

We read with great interest the recently published guidelines on the management of osteoporosis associated with chronic liver disease (Gut 2002; 50(suppl I):11–9). However, we would like to add a few words of comment. Associations between coeliac disease (CD) and primary biliary cirrhosis in particular and other autoimmune liver diseases in general have been reported. In addition, it has been suggested that these individuals should be screened for CD. Thus we suggest that physicians caring for patients with the above mentioned liver diseases should screen them for CD in the presence of signs and symptoms suggestive of malabsorption such as osteoporosis. This seems a reasonable strategy as detection of CD will allow for a more rational therapeutic approach to the risks determined by this association. Complications due to the presence of CD, such as malnutrition, anaemia, and osteoporosis, may have a considerable impact on liver disease management and the need/success of transplantation.

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References

Caelerul induced pancreatitis

We have read with interest the article by Frossard et al (Gut 2002; 50:78–83) entitled “Both thermal and non-thermal stress protect against caerulein induced pancreatitis. In the present study Frossard et al showed that thermal and non-thermal stress induced by injection of the β agonist isoproterenol upregulated HSP70 in the pancreas which is associated with amelioration of subsequent induced caerulein pancreatitis. The authors hypothesise that the protective effects on pancreatitis severity caused by thermal and non-thermal stress may be mediated by HSP70. We believe however that both heat shock stress and non-thermal stress can stimulate several other anti-inflammatory pathways which were not discussed in this study, all of which could be alternative explanations for the observations that were made.

It is widely established that catecholamines, both endogenously released during heat stress shock or by injection of isoproterenol, can influence activation of inflammatory pathways during inflammation and infection (reviewed by van der Poll). Evidence exists that catecholamines exert anti-inflammatory effects on a number of host mediator systems such as the cytokine network and neutrophils, all of which are implicated in the pathogenesis of acute pancreatitis and the pancreatitis associated systemic inflammatory response syndrome. Catecholamines, either endogenously produced or exogenously administered, may act to dampen excessive pro-inflammatory pathways by mechanisms not related to enhanced production of heat shock proteins. Firstly, catecholamines exert anti-inflammatory effects on the cytokine network by inhibiting the production of proinflammatory cytokines such as tumour necrosis factor (TNF), interleukin (IL)-1β, IL-12, and interferon γ (IFN-γ), of which TNF and IL-1β have been implicated as mediators that play a proinflammatory role in acute pancreatitis. Secondly, in animal models of endotoxaemia, pretreatment with isoproterenol enhances the production of the anti-inflammatory cytokine IL-10 which has been shown to be protective in acute pancreatitis. Thirdly, in endotoxiaemia models, β adrenergic stimulation results in reduction of levels of CC chemokines. Fourthly, neutrophil migration to the pancreas, one of the hallmarks of acute pancreatitis, is inhibited by the administration of β agonists but also affects LPS induced neutrophil degranulation in vivo. Fifthly, with regard to the hypothesis that HSP70 prevents the activation of trypsinogen in the pancreas, it must be noted that recent evidence suggests that neutrophils and possibly cytokines can also influence trypsinogen activation. Therefore, the reduction in trypsinogen activation shown in their study might be unrelated to HSP expression and may be explained by the reduction of inflammation due to β adrenergic effect.

Therefore, we believe that the conclusion by Frossard et al that the protective effects of thermal and non-thermal stress might be mediated by HSP70 is only one possible explanation and that their observations might also be explained by the immunomodulatory effects of catecholamines.

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References

When we embarked on this project, we were also concerned that all the stresses that result in the induction of HSP70 may have other non-HSP related effects and did mention this in our discussion. At that point, we did not have the tools to show the crucial protective role played by HSP70.

To prove that a cause-effect relationship exists between thermal and non-thermal stress and protection against pancreatitis, we adopted the anti-inflammatory approach in another recently published experimental study to indicate unequivocally that the thermal stress induced protection of intrapancreatic trypsinogen activation and protection against caerulein induced pancreatitis are mediated by HSP70. Furthermore, our studies have shown that HSP70 induction that occurs during the evolution of pancreatitis in non-thermally stressed rats acts to limit the severity of pancreatitis.

When assessed in vitro, cathecolamines can exert anti-inflammatory effects independent of heat shock proteins. In our paper and their comments on the evolutionary conservation of intracellular heat shock protein 70 and delayed cardioprotection in the rat through alpha 1 adrenoceptors.

Gastroenterology 2002;122:974-84.

References
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Table 1 Invasive cancer detected during follow up of non-invasive gastric neoplasia

<table>
<thead>
<tr>
<th>Histology at enrollment</th>
<th>Gastric cancer detected after follow up longer than 12 months</th>
<th>Gastric cancer detected within 12 months from initial diagnosis</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Follow up (months)</td>
<td>Gastric cancer</td>
</tr>
<tr>
<td>Low grade non-invasive neoplasia (99 cases)</td>
<td>48 (38–80)</td>
<td>1</td>
</tr>
<tr>
<td>High grade non-invasive neoplasia (25 cases)</td>
<td>30 (13–72)</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>33 (13–80)</td>
<td>19</td>
</tr>
</tbody>
</table>

Note: AGC, early gastric cancer (that is, UICC pathological stage II); AGC, advanced gastric cancer (that is, UICC pathological stages II–III); GC-nos, gastric cancer of unknown pathological stage.

**Mean (range)**
series of patients with low and high grade gastric non-invasive neoplasia. The follow up schedule was differentiated a priori, depending on the cancer risk presumably associated with each grade of lesion. The number of patients enrolled in each diagnostic category, follow up time, and number of cancers detected are shown in Table 1. It is worth emphasizing the high prevalence of early gastric cancers (77%) among the 30 cases of cancer detected in our prospective follow up study. The 19 cases of cancer detected in the long term follow up support the premalignant significance of non-invasive neoplasia while the 11 cases detected within one year from the original endoscopy fully demonstrated that non-invasive neoplasia frequently coexists with advanced cancer. Both of these observations could represent valid foundations in drawing a surveillance programme aimed at secondary gastric cancer prevention.

Acknowledgements

The prospective study on gastric non-invasive neoplasia has been supported by the Veneto Region (project number 909/06-99) and granted by the V eneto Region. The prospective study on gastric non-invasive neoplasia.

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The prospective study on gastric non-invasive neoplasia has been supported by the Veneto Region (project number 909/06-99) and granted by the V eneto Region.

Adult height in patients with early onset of Crohn’s disease

Alemzadeh et al (Gut 2002;51:26–9) reported that adult height, compared with the general Dutch population, was reduced by a mean of −0.9 SDs (95% confidence interval −1.5 to −0.28) in 15 Crohn’s patients with prepubertal onset of symptoms. However, the calculated deviation from “target” height (based on parental height) did not reach statistical significance and the authors have speculated that familial short stature, and not Crohn’s disease, may be a factor in this group. Furthermore, no height deficit was found in those with postpubertal onset of symptoms. We are currently undertaking a review of those with childhood onset diseases attending our paediatric and adult IBD clinics. In the majority of cases parental height was measured by trained auxologists, although in some, details were not available in the case notes to discern the method of measurement and may therefore have included self reported parental heights. We calculated SDS scores from the revised British Longitudinal standards using the method described by Alemzadeh et al (mean British male adult height of 176.0 cm (SD 6.3) and female adult height of 163.6 cm (SD 5.7)). “Target height” was calculated for male patients by (paternal height−(maternal height+13))/2 and for female patients by (maternal height+paternal height−13))/2 (cm). “Prepubertal” children were defined as males and females with onset of symptoms at <13 and <11 years, respectively. We defined the upper limit of “postpuberty” as 16 years (mean age 19.3 (SD 2.6) years). An analysis of final height is presented in Table 1. A separate analysis was also carried out using the nomogram of the Child Growth Foundation which corrects height until the age of 22, but this did not alter our findings and the data are therefore not presented.

In the majority of patients (85% (23/27); p=0.008, χ² test) final height was less than “target height”, and in 22% (6/27) the final height deficit was more than 10 cm. For those aged over 18 years the values were 88% ((14/16); p=0.022, χ² test) and 25% (4/16), respectively.

In this sample of patients with childhood onset Crohn’s disease we found no evidence of a familial basis for short stature. Our data confirm the findings of others that mean adult height of patients with onset of symp- toms before the age of 16 is reduced.3,4 Using age of onset of symptoms as a proxy for puberty, we found no significant difference in final height between those with pre- and postpubertal onset of symptoms. This is in contrast with the findings of Alemzadeh et al and may be because of differences in the upper age limit of “postpuberty” (16 versus 22 years).

Growth failure remains a concern to our British Crohn’s patients and although the mean deficit of 5–6 cm from target height may be considered clinically inconsequential this includes a subset with much more significant growth impairment. A better understanding of the mechanisms underlying growth failure is required to determine whether there is an identifiable group of children that may benefit from early and more intensive immunosuppression and/or nutritional therapy.5,6 We agree with Alemzadeh et al that only larger (population based) studies will have the power to determine the effect of factors such as site of disease activity and therapeutic intervention.

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<table>
<thead>
<tr>
<th>Table 1 Final height of Caucasian Crohn’s patients with pre and postpubertal onset of symptoms</th>
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<tr>
<td><strong>All</strong></td>
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<tr>
<td>n</td>
</tr>
<tr>
<td>Deficit (cm)</td>
</tr>
<tr>
<td>&gt;16 years at last height</td>
</tr>
<tr>
<td>-0.68</td>
</tr>
<tr>
<td>-0.59</td>
</tr>
<tr>
<td>≤18 years at last height</td>
</tr>
<tr>
<td>-0.73</td>
</tr>
<tr>
<td>-0.53</td>
</tr>
</tbody>
</table>

Data are mean (95% confidence interval).

References


of clinical not biochemical deficit. Also, there is no information on the
This formula does not include a correction for
they used another formula for target height. Furthermore,
Another difference could be that the authors
caused by the other “end point” of puberty
I think that this study is clear. We agree with
Author’s reply
I think that this study is clear. We agree with
References

Underdiagnosis of hereditary haemochromatosis: reflects lack of clinical not biochemical penetration
In their paper, Ryan et al (Gut 2002; 51:108–12) reported that 78% of men (mean age 42 years) and 36% of women (mean age 39 years) who were identified as C282Y homozygotes by family screening had evidence of biochemical iron overload. They concluded that underdiagnosis of hereditary haemochromatosis may be the result of failure to diagnose the phenotype in patients with iron overload.
In Glasgow, the prevalence of the C282Y homozygous state is high at approximately 1 in 180 of the population, of whom only 3.1% had been diagnosed by August 2001. Of these known cases we identified 42 (20 males) C282Y homozygotes who had been diagnosed by predictive genetic testing of family members of affected probands. At diagnosis, all 20 males (mean age 46) had evidence of biochemical iron overload, defined as a transferrin saturation of >45%. Two patients had iron overload identified immunohchemically) or a serum ferritin of >300 µg. Both parameters were elevated in 15 (75%) individuals, with three having an isolated transferrin saturation and two an isolated elevated ferritin.
Of the 22 females (mean age 44) identified, 18 (81%) had evidence of biochemical iron overload, with 10 (45%) having raised transferrin saturation and ferritin, as defined above. A further seven patients had an isolated elevation in transferrin saturation and one had an elevated ferritin alone. Only four (9.5%) C282Y homozygotes identified by family testing had no evidence of biochemical iron overload. All of the individuals were female (age range 17–48 years). Unfortunately, due to the retrospective nature of the analysis, it was not possible to assess symptoms at diagnosis.

The prevalence of biochemical iron overload in our predominantly Celtic population is high and comparable with that reported from Dubl

in a recent large population screening study from the USA, Beutler et al reported the prevalence of biochemical iron overload in C282Y homozygotes to be similar to that observed by the Dublin group and ourselves. However, they found no evidence of more frequent symptoms in C282Y homozygotes compared with controls, even if biochemical iron overload was present. It appears that these individuals have iron overload and a number of unrelated non-specific symptoms, similar to those seen in the general population.
Prospective longitudinal studies are required to establish the proportion of C282Y homozygotes who will eventually exhibit the clinical phenotype of hereditary haemochromatosis.

Gastrointestinal epithelial neoplasia
We read with interest the viewpoint “Gastrointestinal epithelial neoplasia: Vienna revisited” by Dixon (Gut 2002; 51:130–1).
For many years Western gastrointestinal pathologists have followed the recommendations of British gastrointestinal pathologists. We learned that terms such as carcinoma in situ should be banned from the diagnostic terminology as it could lead to misinterpretation by surgeons and unnecessary surgical intervention.
The Vienna classification has introduced new avenues to the understanding of the process of carcinogenesis in the gastro-intestinal tract. For some Western pathologists in the Vienna group who also received histopathological training in Japan, the concept of intraepithelial carcinoma (that is, carcinoma in situ) and of intramucosal carcinoma appeared natural. Although during the process of discussions British gastrointestinal pathologists appeared reluctant to accept such controversial notions, the discussion became less intense during the second day, and at the end a consensus was reached, gaining finally the pages of this journal.

The Vienna classification dismissed the concept of dysplasia from that of carcinoma in its earlier forms. After many years of studying adenomas we now know that low grade dysplasia may progress to high grade dysplasia. On the other hand, there remains elusive whether carcinoma in situ is preceded by high grade dysplasia or develops without a prodro-

morphic phase. By the same token we do not know whether carcinoma in situ antedates intramu-

Collagen and iron overload in the gastro-intestinal mucosa is characterised by spindle or gang shaped, elongated, pleomorphic, hyperchro-
matic nuclei, and regular nuclear membrane wherein carcinoma in situ displays large vesicular nuclei, irregular nuclear nucleoli, and scolloped nuclear membranes. Bridges of nucleolus associated chromatin reaching irregular chromatin deposits are seen in the nuclei of carcinoma in situ and chromatin are also seen connecting angular chromatin clumps. The nuclear polarity is disrupted, and marked cell pleomorphism and aberrant mitosis are present. Structural alterations may occur such as budding or branching crypts or tubules, with epithelial septa and back to back glands, and cribriform sheet of epithelial cells in clusters and sheets. Those structures are confined to the basement membrane of the epithelial layer. But surprisingly, despite those differences, high grade dysplasia and carcinoma in situ are still being regarded as synonyms in the Western literature.

The present discussion is beyond the usefulness of the Vienna classification as a tool for proper treatment; the discussion aims to point out our present lack of knowledge regarding the histogenesis of lesions repre-

sented by categories 2–4. The multi-centre nature of the Vienna’ and their correct identification for future molecular research.

The viewpoint of Dixon appears to be in conflict with the demand of gastrointestinal pathologists who are willing to embrace this new “doctrine” in order to acquire accurate information on the histological steps followed
by early neoplastic lesions of the gastrointestinal tract. Only then will we be able to translate such events into molecular terms. The normal and pathological anatomy of the different parts of the gastrointestinal tract and the abdominal organs, and the extraperitoneal spaces is described in detail. There are excellent descriptions of the intraperitoneal spread of infections and of making it recurrent. There is also a chapter on internal abdominal hernias. Perforative disorders and their mode of spread are described in detail, the diverse locations of pancreatic pseudocysts with intraperitoneal air are well illustrated.

CT is now used to show the many places where free intraperitoneal air can collect in the abdomen. In recent years, Cho and Baker have used this information to reassess the radiological appearances of free intraperitoneal air on the supine abdominal radiograph and have also described a number of new signs. They have brought this information together in their chapter and the result is an excellent and well illustrated contribution, providing information on an important subject that is not widely available elsewhere.

This edition of Dynamic Radiology of the Abdomen lives up to the reputation established by previous editions. It should be of interest to all doctors who wish to learn more about the abdomen, particularly radiologists, oncologists, and surgeons. Radiologists who perform any type of abdominal imaging should find this latest edition invaluable and registrars training in radiology should become acquainted with it early in their career. In the preface to the first edition, Lloyd Nyhus commented on the innovative nature of this work and stated that it was an important reading source for surgeons; it likely that he would be equally enthusiastic about the latest edition.

The provision of nutritional support for patients in hospital and in the community has derived from a heightened awareness of the impact of malnutrition on patient outcome and quality of life. Attention has focused on the prevalence of disease related malnutrition, both in terms of weight loss and weight for height, but also in terms of micronutrient deficiencies. Drives to enhance awareness of undernutrition in the community have followed efforts to improve nutrition in hospitals through food improvement, supplement provision, and artificial feeding. The need for routine nutritional assessment in clinical practice, especially in chronic disease, and its careful documentation has become ever more appreciated, particularly among gastroenterologists.

It is widely acknowledged that the approach to nutritional care is best made via a multiprofessional team which combines the skill and knowledge of dieticians, nutrition nurse specialists, pharmacists, and doctors. Teams like this are the key to excellent patient centred nutritional care and lead to overall hospital strategies for nutritional support which seamlessly combine, in a cost effective way, the use of food and supplements with artificial feeding. The nutrients by the simplest safest route acceptable to the patient. Such teams need to develop more in the community too.

As the more enlightened hospitals form teams, a demand develops for a comprehensive textbook. This one is widely seen as one of the best and it now has the collaborative input of what goes on in the more Anglophile corners of ESPEN. As such it unsurprisingly contains some excellent chapters and others which are not. This book satisfies the reader to understand the complex anatomical arrangement of the abdominal organs and spaces and how they are modified by disease processes. The normal and pathological anatomy of the different parts of the gastrointestinal tract and the abdominal organs, and the extraperitoneal spaces is described in detail. There are excellent descriptions of the intraperitoneal spread of infections and of making it recurrent. There is also a chapter on internal abdominal hernias. Perforative disorders and their mode of spread are described in detail, the diverse locations of pancreatic pseudocysts with intraperitoneal air are well illustrated.

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Oesophageal disease brings together many disciplines within the field of gastroenterology and the book is aimed primarily at the specialist oesophageal surgeon. Reconstruction of the oesophagus following resection for benign or malignant disease is one of the most challenging surgical procedures currently performed, and the oesophagus being such an unforgiving organ increases that challenge. Rather surprisingly, this is the first truly authoritative and evidence based volume to be devoted completely to this topic.

This slim volume of 84 pages comprises 10 chapters also review one or two abstracts. The first chapter does have a section on health and practice by clinical governance—sensible answer is that clinical governance might ensure “uniform standards” across heterogenous NHS practices, reassure the public and, in any case, it seems here to stay—at least for the moment.

It still baffles me as to what differences there are between excellent clinical practice and practice by clinical governance—presumably the latter is not meant to be quite as good, but will do. There seems to be a clamour for “acceptable” standards and this anticipating being in charge of ensurin we do our jobs properly and services are commissioned effectively. There are one or two omissions—for example, the lack of mention of varices as a cause of acute bleeding—but each chapter does have a section on health economics for those hopefully traversing the quagmire of costs, effectiveness, care, and quality. As C Norton wrote in the book, “there is a fine school. He is therefore well qualified to write this specialist tome. The historical chapter regales the courage of the pioneers of oesophageal surgery in the first half of the twentieth century, in which great British oesophageal surgeons including grey Turner, Ivor Lewis, Allison, and Belsey are afforded due prominence. Following general sections on the philosophy of and indications for oesophageal replacement and the existence of the available of the oesophageal substitute and route to bridge the gap, a chapter is then devoted to each of the principal reconstructive techniques using stomach, colon, and jejunum, as well as the use of prosthetic tubes. Each of the chapters goes into considerable detail about relevant surgical anatomy, physiology, operative technique, and complications and their management.

Reconstructive Surgery of the Oesophagus is clearly written. While it draws heavily on the author’s considerable experience, one of the attractions of this book is that it is clearly evidence based, and as well as being liberally referenced, the key references and conclusions are highlighted in tabular form in each chapter. Another strong point for its predominantly surgical audience is the wealth of line drawings, which clearly depict surgical anatomy and technique. Overall, this is an excellent book, which takes its place well between existing tomes on oesophageal disease. I can recommend it wholeheartedly as an essential reference volume for both trainees and consultants in oesophageal surgery and indeed gastroenterologists might usefully dip into it occasionally so as to appreciate the many challenges facing their oesophageal surgeons in this fascinating branch of gastrointestinal surgery.

The Pelvic Floor: Its Function and Disorders


In my clinical practice, I have felt for a long time that the knowledge acquired in colorectalology should be more frequently shared with specialists in obstetrics, gynaecology, urology, neurology, etc. Therefore, I enjoyed receiving this book where the contributors range between at least 10 specialities which concern pelvic floor disorders. Each chapter is written by a leading figure or a group expert in this field. The book is a valuable starting point for gastroenterologists who wish to become up to date with data concerning the clinical problems of the pelvic floor, associating the anterior and posterior components, including physiology, anatomy, diagnostic imaging, surgery, nursing, and psychology.

The previously published book by these authors was entitled Coloproctology and the Pelvic Floor—coloproctology has disappeared from the title of their new book, indicating that it is no longer possible to approach the posterior pelvic floor disorders without studying the pelvic floor as a whole. In this way, this new title by itself is a very strong message. However, the reader might be a little disappointed if he looks for how to treat, for example, a patient suffering from both anal and urinary incontinence, or a patient complaining of urinary stress incontinence and posterior pelvic floor dysfunction inducing straining at stools. The book contains a number of excellent algorithms concerning pathogenesis, investigations, and treatment of the pelvic floor disorders. However, these algorithms have been constructed to treat either the anterior pelvic floor or the posterior pelvic floor, but not to treat a patient who complains simultaneously of the two parts of the pelvic floor. It was perhaps because the book was initially so promising and the subsequent chapters so interesting that I was hoping for a little more specific detail from the authors!

Nevertheless, the psychological characteristics of the pelvic floor disorders are very well described, suggesting that the impact of social factors, such as sexual abuse for example and psychological distress, on the expression of pelvic floor symptoms should be taken into account. To date it has not been very easy to suggest guidelines indicating how to achieve a balance between identifying the pathophysiology of pelvic floor disorders and understanding psychological factors. There is no doubt that the algorithms given at the beginning of the book will be very useful for the reader. However, they would have been even more useful if the experts had suggested at which step(s) of their algorithms they felt they need to investigate the psychological profile of their patients.

As C Norton wrote in the book, “there is a small but growing movement to create multidisciplinary pelvic floor clinics, where uro-gynaecologists, colorectal surgeons, specialist nurses, physiotherapists, neurologists, psychiatrists . . . work together to improve the management of pelvic floor disorders”. While we are waiting for these future multidisciplinary clinics of “perineurology”, it was probably not the time to furnish algorithms in this particular edition of The Pelvic Floor concerning investigations and management of associated symptoms of the anterior and posterior pelvic floor, integrating the psychological profiles of the patients. JH Pemberton, M Swash, and MM Henry must be acknowledged and congratulated for making together the knowledge of all the specialties involved in the pelvic floor.
vast majority of papers covered were published in 2001 or 2000. The Fast Fact Highlights series aims to “keep its readers abreast of the latest innovations” in each specialty. The flyer states that the information is presented “in an accessible style, comprehensively illustrated and fully indexed”. Have these aims been met? Certainly the style is easy to read. However, there are only three figures in the whole book. Two of these are world maps showing geographical variations in colorectal incidence and mortality, while a third figure is a rather pointless flow chart of “preventive steps” for patient groups at average, moderate, and high risk. The steps are identical for the first two groups: change in lifestyle, chemoprevention and screening, and early diagnosis. These steps are again repeated for the high risk group, with preventive surgery added. There is no subject index.

I like the table in each chapter stating what are “in”, what are “out”, what are contentious, and what is still needed. However, it is irritating that many of the items mentioned as “in” or “out” have neither been discussed in the text nor referenced.

In the discussion on endoscopic treatment of gastro-oesophageal reflux, the EndoCinch and implantation of microspheres were discussed, but not the Stritta procedure. Both Freedman’s study on the association between cholecystectomy and oesophageal adenocarcinoma as well as Schnell’s report on non-surgical management of Barrett’s oesophagus with high grade dysplasia, were reviewed in the oesophagus chapter and again in the chapter on gastrointestinal cancer. Tighter editing could have avoided this duplication as “in” or “out” have neither been discussed in the text nor referenced. These brief reviews cannot by their nature be comprehensive. While this volume covers more ground than the short literature review booklets sponsored and distributed free by pharmaceutical companies, only about 20 papers are reviewed per topic. This can only represent a small selection of the many advances over a one to two year period, and falls far short of the excellent reviews in the Current Opinions in Gastroenterology series. It is probably unsuitable for a library collection, and is not a book I would myself keep for reference. I am uncertain who may wish to purchase this volume, even though it is modestly priced at £15. While it is an easy read, I suspect that few consultant gastroenterologists would want to buy this book. I doubt if many trainees would either.

**CORRECTION**

In the paper by Higham et al (Gut 2002;50:460–4) the heading for table 4 should read “Number of items (thousands) prescribed in England from 1990 to 1999. Prescription Cost Analysis system (Department of Health)”.

**NOTICES**

**38th EASL Annual Meeting**

The European Association for the Study of the Liver will be holding its 38th annual meeting on 29 March–1 April 2003 in Istanbul, Turkey. Further information can be found on the website www.easl.ch/easl2003.

**Falk Workshop—Inflammatory Bowel Disease: Turning New Advances into Practice**

This will be held on 3 April 2003 in Berne, Switzerland. Further details: Falk Foundation e.V., Congress Division, PO Box 6529, Leinenweberstr. 5, 79041 Freiburg/Br, Germany. Tel: +49 761 15 14 00; fax: +49 761 15 14 339; email: symposia@falkfoundation.de; website: www.falkfoundation.de

**International Symposium on Viral Hepatitis and Liver Disease**

This conference will take place on 6–10 April 2003 in Sydney, Australia. Further information: ISVHLD 2003 Congress Managers, GPO Box 128, Sydney NSW 2001, Australia. Tel: +612 9262 2277; fax: +612 9262 3155; email: isvhld@tourhosts.com.au; website: www.tourhosts.com.au/isvhld

**Prague Hepatology Meeting**

To be held on 5–7 June 2003 in Prague, Czech Republic. Leading speakers from Europe and the USA will present new ideas and suggestions on pathophysiology, diagnostics, and therapy of liver diseases in ten programmes blocks. Further details: Ms Veronika Revicka. Tel: +420 241 445 799; fax: +420 241 445 806; email: veronika@congressprague.cz

**Falk Symposia—New Findings on Pathogenesis and Progress in Management of IBD**

Two symposia and a workshop will be held on 10–14 June 2003 in Berlin, Germany. Further details - see Falk Workshop details above.

**Gastroenterology and Endotherapy: XXIst European Workshop**

This will be held on 16–18 June 2003 in Brussels, Belgium. Further details: Nancy Beaufreuz, Administrative Secretariat of the Workshop, Gastroenterology Department, Erasme Hospital, Route de Lennik 808, B-1070 Brussels, Belgium. Tel: +32 (0)2 555 49 00; fax: +32 (0)2 555 49 01; email: beaufrez@ulb.ac.be

**The Association of Coloproctology of Great Britain & Ireland**

This annual meeting will be held on 7–10 July 2003 in Edinburgh, UK. Further details: Conference Secretariat, The ACGB at the Royal College of Surgeons of England, 35–43 Lincoln’s Inn Fields, London WC2A 3PE. Tel: +44 (0)20 7973 0307; fax: +44 (0)20 7430 9235; email: acgbi@asgbi.org.uk; website: www.acgbi.org.uk