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 (Oster 2002; 50: suppl 1:1–9). 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.1–3 In addition, it has been suggested that these individuals should be considered as an at risk group for whom serological testing for CD is indicated. Patients with CD are at high risk of developing low bone mineral density and bone turnover impairment,2 and it has been shown that adherence to a gluten free diet has a significant positive impact on these parameters.4–5 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 its natural course.6

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References

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mental systems. Echolamines have been used in the past to induce HSP70 expression. In fact, cultured rat brown adipocytes is due to nitric oxide induced HSP70 expression. In fact, cat-
icosulted the targets of additional sampling. We considered the standardised protocol of biopsy sampling is a morphological question. Firstly, are there “special” requirements (that is, a protocol of gastric biopsy sampling) to be satisfied when carrying out the endoscopic procedure? Secondly, are there evidence-based criteria for selecting patients to be included in surveillance pro-
grame? The authors do not provide detailed information on the number of biopsy samples obtained per endoscopy. We believe that a standardised protocol of biopsy sampling is a leading part of any upper endoscopy procedure, and mucosal “abnormalities” should be con-
tidered the targets of additional sampling.1 Taking into account that 46% of the cancers referred to in Whiting’s study were discovered within 13 months from the second last procedure, we agree with the authors who con-
sidered these cancers endoscopically missed.

The second point of concern is the rationale for a surveillance protocol. Any cost effective strategy of secondary cancer prevention requires that the risk of cancer to be higher within the population undergoing surveillance than in the general population. In the Birmingham study, such a prerequisite does not seem to have been assumed in the study design. Endoscopy surveillance definitively included the whole spectrum of abnormalities described in gastric carcinogenesis,11 from regenerative changes (with a nearly null cancer risk) to non-invasive neoplasia (which carries, by definition, predis-
position for progression to invasion and metastasis). As a consequence of (i) cancers missed at endoscopy and (ii) shortness of the follow up period, the results shown in their table 3 may be misleading. The association of intestinal metaplasia (regardless of its histo-
chemical phenotype) with the highest risk of cancer evolution is not only biologically ques-
tionable but, and this is even worse, it may result in inappropriate patient management. While intestinal metaplasia represents the most common background of stomach cancer,11 “gastric intestinalisation” per se does not carry the phenotypic and genotypic altera-
tions pre-currying invasive neoplasia. Most importantly, the high prevalence of metaplas-
atic lesions within subjects who will never develop adenocarcinoma excludes (non-
extensive) intestinal metaplasia as the proper target of surveillance protocol design.

In the natural history of epithelial tumours, the term dysplasia identifies a lesion that car-
ries biological alterations comparable with those of full fledge cancer but lacking stromal invasion.1 Recently, the term “dyspla-
sia” has been replaced by “non-invasive neoplasia”, which more clearly identifies such a lesion as the most advanced alteration ante-
cedent to invasive adenocarcinoma.4 Since 1985, we have prospectively followed up a

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>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Follow up (months)</td>
<td>Follow up (months)</td>
</tr>
<tr>
<td>Low grade non-invasive neoplasia (99 cases)</td>
<td>1.5 (1–2)</td>
<td>1.5 (1–2)</td>
</tr>
<tr>
<td>High grade non-invasive neoplasia (25 cases)</td>
<td>2 (1–4)</td>
<td>2 (1–4)</td>
</tr>
<tr>
<td>Total</td>
<td>1.7 (1–4)</td>
<td>1.7 (1–4)</td>
</tr>
</tbody>
</table>

EGC, early gastric cancer (that is, UICC pathological stage I); 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. The Veneto Region has been supported by the Veneto Region. The prospective study on gastric non-invasive neoplasia has been supported by the Veneto Region. The prospective study on gastric non-invasive neoplasia has been supported by the Veneto Region. The prospective study on gastric non-invasive neoplasia has been supported by the Veneto Region. The prospective study on gastric non-invasive neoplasia has been supported by the Veneto Region. The prospective study on gastric non-invasive neoplasia has been supported by the Veneto Region. The prospective study on gastric non-invasive neoplasia has been supported by the Veneto Region. The prospective study on gastric non-invasive neoplasia has been supported by the Veneto Region.

References


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 −1.55 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 Standards1 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, in contrast with Alemzadeh et al who used 22 years. The population we serve is ethnically diverse and therefore we have confined this analysis to Caucasians, as per the Dutch study. There was no deficit in height of the parents (48 mothers and 46 fathers) of children with childhood onset Crohn’s disease compared with the general population. Furthermore, there was no height deficit in children examined by onset of their child’s Crohn’s disease: mean “prepubertal” parental SDS 0.00 (SD 1.11) and mean “postpubertal” parental SDS 0.08 (SD 1.12) (n=70 and n=24, respectively; p=0.34). In addition, we found no significant sex difference: mean paternal SDS −0.20 (SD 0.98) and mean maternal SDS 0.03 (SD 1.22) (n=46 and n=48, respectively; p=0.94). In 27 cases (18 males and nine females) children of these parents are now aged more than 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% (25/27); 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 symptoms before the age of 16 is reduced.1 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 due to 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 by some to be 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.

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 1: Final height of Caucasian Crohn’s patients with pre and postpubertal onset of symptoms

<table>
<thead>
<tr>
<th>Age at Height Measurement</th>
<th>Prepubertal</th>
<th>Postpubertal</th>
<th>Pre v post</th>
<th>n</th>
<th>Pre v post p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;16 years at last height</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deficit (cm)</td>
<td>−0.68</td>
<td>−1.17 to −0.19</td>
<td>−0.57</td>
<td>−1.16 to 0.02</td>
<td>−1.01</td>
</tr>
<tr>
<td>n</td>
<td>20</td>
<td>7</td>
<td>7</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>&gt;18 years at last height</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deficit (cm)</td>
<td>−0.73</td>
<td>−1.42 to −0.04</td>
<td>−0.65</td>
<td>−1.48 to 0.18</td>
<td>−1.07</td>
</tr>
<tr>
<td>n</td>
<td>18</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td></td>
</tr>
</tbody>
</table>

Data are mean (95% confidence interval).
References

Author’s reply
I think that this study is clear. We agree with the authors that the differences are possibly caused by the other “end point” of puberty and the small population in both studies. Another difference could be that the authors did not calculate the corrected height SDS (height SDS target height SDS); this may be lower for the prepubertal group compared with the postpubertal group. Furthermore, they used another formula for target height. This formula does not include a correction for secular trends which underestimate the deficit. Also, there is no information on the effect of corticosteroid use.

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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 carriers of the C282Y mutation 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% (transferrin detected 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 (82%) 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 Dub lin by Ryan et al. However, the proportion that will develop clinical “disease” related to hereditary haemochromatosis remains uncertain. Ryan et al proposed that underdiagnosis of hereditary haemochromatosis might be due to the non-specific nature of the symptoms early in the disease. They noted that fatigue, arthropathy, and male impotence were common complaints in these C282Y homozygotes identified by family screening. However, they provided no evidence that these symptoms were due to iron excess as they appear to be common in their biochemically non-expressing control group. It would be interesting to know whether any of these non-specific symptoms improved with phlebotomy.

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 that will eventually exhibit the clinical phenotype of hereditary haemochromatosis.

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References

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 unchecked surgical intervention. The Vienna classification has introduced new avenues to the understanding of the process of carcinogenesis in the gastrointestinal tract. For some Western pathologists in the Vienna group who also received histological training in Japan, the concept of intraepithelial carcinoma (that is, carcinoma in situ) and of intramucosal carcinoma appeared natural. Although during the initial phase of discussions some Western pathologists argued 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 dismembered 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 some maintain elusive whether carcinoma in situ is preceded by high grade dysplasia or develops without a prodro mic phase. By the same token we do not know whether carcinoma in situ antedates intramu cosal carcinoma. If those microscopic realities of colorectal carcinogenesis are being ignored, how are we going to learn in a correct sequential fashion the intricate molecular footsteps that telescope from dysplasia to submucosal carcinoma? As that Pandora box is being presented to pathologists we should treasure it by opening it little by little.

One criticism of the Vienna classification may be that although various categories of neoplasia were listed, the histopathological criteria for each one of the lesions were not verbalised, thus postponing the opportunity for its worldwide acceptance. Notwithstanding, some Western pathologists have started to herald the new “doctrine” by proposing histopathological descriptions (criteria) for each one of the various categories proposed in Vienna.

“For see or not to see” is not the question, as all lesions are there. As an example, dysplasia can be differentiated from carcinoma in situ. Dysplasia in the glandular gastrointestinal mucosa is characterised by spindle or cigar shaped, elongated, pleomorphic, hyperchromatic nuclei, and regular nuclear membrane whereas carcinoma in situ displays large vesicular nuclei, irregularly shaped, elongated, pleomorphic, hyperchromatic nuclei, and scalloped nuclear membranes. Bridges of nucleolus associated chromatin reaching irregular chromatin deposits are seen in the nuclear membrane whereas carcinoma in situ displays large nucleolus associated chromatin are also seen connecting angular chromatin clumps. The nuclear polarity is disrupted, and marked cell pleomorphism and aberrant mitoses are present. Structural alterations may occur such as budding or branching crypts or tubules, with epithelial septa and back to back glands, and cribriform growth 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 Vienna classification and their correct identification for future molecular research.

The viewpoint of Dixon appears to be in concert with the desire of many Western 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 gastro-intestinal tract. Only then will we be able to translate such events into molecular terms. Intestinal tract. Only then will we be able to translate such events into molecular terms.

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. Driven 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 to feed the nutrients by the simplest safest route acceptable to the patient. Such teams need to develop more in the community too.

Such teams need to develop more in the community too.
and thoughtfulness of a pharmaceutical company and I trust that someone will come forward to do this. I do not plan to part with my copy, despite continuing references to prokinetic drugs which are no longer, or were never, available in the UK.

P Hungin

Reconstructive Surgery of the Esophagus

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.

The omission was trained at the University of Chicago under doyens of oesophageal surgery including Skinner, Belsey, and DeMeester, and has continued the tradition of that 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 equipment 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 Esophagus is clearly well 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 describe 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.

A Watson

The Pelvic Floor: Its Function and Disorders

In my clinical practice, I have felt for a long time that the knowledge acquired in coloproctology 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 specialties 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 covering both 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 incontinence and an urge to defecate, or a patient complaining of urgency incontinence and a posterior pelvic floor dysfunction inducing straining at stools. The book contains a number of excellent chapters 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 showed 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 account. T o 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 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 the 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 multi-disciplinary 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 “perineology”, 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 their effort to gather together the knowledge of all the specialties involved in the pelvic floor.

P Denis

Gastroenterology Highlights 2001–02
Edited by E Quigley. Health press, 2002, £15.00, colour, pp 84. ISBN 1-903734-12-6

Gastroenterology Highlights 2001–2 is attractively presented in good quality four colour format. This slim volume of 84 pages comprises 10 chapters written by a panel of international experts. Topics covered range from diseases of the oesophagus, liver, gall bladder, to complications of liver disease, endoscopy, and colorectal cancer prevention and screening. The aim is to discuss key knowledge and to put them into context. In most chapters, about 20 papers are reviewed but the actual numbers range from 16 to 30. Most chapters also review one or two abstracts. The Can external control drive clinical standards? In the meantime we have clinical governance. What this actually means, how it is meant to operate, and whether governance guidelines will become yardsticks for judging performance also remain open. But at least it sounds like a good thing, and one that hopefully sensible answer is that clinical governance might ensure “uniform standards” across heterogeneous 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 ensuring 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. Word has it that this book is selling well; the authors have got that aspect of health economics correct and timely.

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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 from colorectal cancer. 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 are 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 Stretta procedure. Both Freedman’s study on the association between choledochectomy and oesophageal adenocarcinoma as well as Schnell’s report on nonsurgical 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 space in this book is clearly at a premium. I was surprised to read that “rectal examination as the only test for colorectal cancer” was “out”. This statement was not 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.

J Y Kang

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: Nancy Beuarez, 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: beuarez@ulb.ac.be

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: +61 9262 2277; fax: +61 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 Veronica Revicka. Tel: +420 241 445 759; 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 Beuarez, 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: beuarez@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 ACGBI 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: wwww.acgbi.org.uk

www.gutjnl.com
Pathology and cost effectiveness of endoscopy surveillance for premalignant gastric lesions
M Rugge, M Cassaro, G Pennelli, V M Russo, F Di Mario and F Farinati

Gut 2003 52: 453-454
doi: 10.1136/gut.52.3.453

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