LETTERS

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The editors will decide as before whether to also publish it in a future paper issue.

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 1):i1–9). 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 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, and it has been shown that adherence to a gluten free diet has a significant positive impact on these parameters. 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


Caerulein induced pancreatitis

We have read with interest the article by Frossard et al (Gut 2002;50:78–83) entitled “Both thermal and nonthermal 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 subsequently 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 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 shock stress 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 endotoxemia 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, towards chemotactic stimuli such as C5a and lipopolysaccharide (LPS) is reduced by 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 effects.

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
We thank van Westerloo et al for their interest in our paper and their comments on the interpretation of our data. They are of the opinion that besides heat shock proteins both thermal and non-thermal stress can stimulate several other anti-inflammatory pathways that in turn could be responsible for the protective effects observed in the study. Secondly, catecholamines can exert anti-inflammatory effects independent of heat shock proteins.

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 catecholamine stress and protection against pancreatitis, we adopted the anti-sense oligonucleotide 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 caerulein-induced protection of intrapancreatic trypsinogen activation and acute pancreatitis in rats.


Pathology and cost effectiveness of endoscopy surveillance for premalignant gastric lesions

We read with great interest the article by Whiting et al (Gut 2002;50:378-81). The Birmingham experience shows how the prevalence of gastric cancers detected at an early stage is significantly higher in the endoscopically surveyed population than in non-surveyed patients. As a result, this study demon-strates that, in the secondary prevention of gastric malignancy, the “once in a lifetime” strategy (suggested for colorectal cancer) is not cost effective while repeated endoscopies (in selected patients) seem most appropriate. This conclusion however raises two cardinal questions. Firstly, are there “special” require-ments (that is, a protocol of gastric biopsy sampling) to be satisfied when carrying out the upper endoscopy procedure? Secondly, are there “evidence based criteria” for selecting patients to be included in surveillance programmes?

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 considered the targets of additional sampling. 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 considered 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 the risk of cancer to be higher within the subsets 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, 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 histochemical 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, “gastric intestinalisation” per se does not carry the phenotypic and genotypic alterations pre-curring invasive neoplasia. Most importantly, the high prevalence of metaplasia 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. In the natural history of epithelial tumours, the term dysplasia identifies a lesion that carries biological alterations comparable with those of full fledge cancer but lacking stromal invasion. Recently, the term “dysplasia” has been replaced by “non-invasive neoplasia”, which more clearly identifies such a lesion as the most advanced alteration ante-cedent to invasive adenocarcinoma. Since 1985, we have prospectively followed up a

| Table 1 | Invasive cancer detected during follow up of non-invasive gastric neoplasia |
|-----------------------------------------------|
| Gastric cancer detected after follow up longer than 12 months | Gastric cancer detected within 12 months from initial diagnosis |
| Histology at enrollment | Follow up (months) | EGC | AGC | GC-nos | Follow up (months) | EGC | AGC | GC-nos |
|-----------------------------------------------|
| Low grade non-invasive neoplasia (99 cases) | 48 (38-80) | 5 | 1 | 2 | 1.5 (1-2) | 1 | 1 | 0 |
| High grade non-invasive neoplasia (25 cases) | 30 (13-72) | 19 | 3 | 1 | 2 (1-4) | 6 | 3 | 0 |
| Total | 33 (13-80) | 19 | 3 | 1 | 1.7 (1-4) | 11 | 3 | 0 |

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).
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 potential of the 11 cases detected within one year from the endoscopy. Furthermore, no height deficit was found in those with postpubertal onset of symptoms.

We are currently undertaking a review of those with childhood onset of symptoms 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 1 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 (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 our data set is predominantly Caucasian and may be because of differences in the upper age limit of “postpuberty” (16 versus 22 years).

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 deficit in height when examined per the Dutch study. 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.

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

<table>
<thead>
<tr>
<th>All</th>
<th>Prepubertal</th>
<th>Postpubertal</th>
<th>Pre v post</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n=16</td>
<td>n=14</td>
<td>n=16</td>
</tr>
<tr>
<td>&gt;16 years at last height</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SDS</td>
<td>-0.68</td>
<td>-1.17 to -0.19</td>
<td>-0.57</td>
</tr>
<tr>
<td>Deficit (cm)</td>
<td>-5.9</td>
<td>-8.2 to -3.4</td>
<td>-5.5</td>
</tr>
<tr>
<td>&lt;18 years at last height</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SDS</td>
<td>-0.73</td>
<td>-1.42 to -0.04</td>
<td>-0.65</td>
</tr>
<tr>
<td>Deficit (cm)</td>
<td>-5.3</td>
<td>-8.6 to -2.0</td>
<td>-5.1</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 trend, which might underestimate the defect. 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 penetrance
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 due to 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 5.1% 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% (transferrin detected immunocytochemically) 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 the individuals were identified in a 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 symp- toms 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 appeared to be as common in their biochemi- cally non-expressing control group. It would be interesting to know whether any of these non-specific symptoms improved with phlebo- nomy.

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 fre- quent 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 homozyg- otes who will eventually exhibit the clinical phenotype of hereditary haemochromatosis.

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References

Gastrointestinal epithelial neoplasia
We read with interest the viewpoint “Gastro- intestinal epithelial neoplasia: Vienna revis- ited” by Dixon (Gut 2002; 51:130–1). For many years Western gastrointestinal pathologists have followed the recommenda- tions of British gastrointestinal pathologists. We learned that terms such as carcinoma in situ should be banned from the diagnostic termin- ology as it could lead to misinterpretation by surgeons and to unnecessary surgical inter- vention.

The Vienna classification has introduced new avenues to the understanding of the process of carcinogenesis in the gastro- intestinal tract. For some Western patholo- gists in the Vienna group who also received histopathological training in Japan, the con- cept of intraepithelial carcinoma (that is, car- cinoma in situ) and of intramucosal carcino- maca appeared natural. Although during the period of discussions and arguments, patholo- gists appeared reluctant to accept such contro- versial notions, the discussion became less intense during the second day, and at the end of the meeting, 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, there remains 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 sequen- cially fashion the intricate molecular footsteps that telescope from dysplasia to submucosal carcinoma? As that Pandora box is being pre- sented 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. Notwith- standing, 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.

“...to 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, hyperchro- matic nuclei, and regular nuclear membrane whereas carcinoma in situ displays large vesicular nuclei, irregular nuclear nucleoli, and scalloped nuclear membranes. Bridges of nucleous associated chromatin reaching irregular chromatin deposits are seen along the nuclear membrane and lesions of chromatin are also seen connecting angular chromatin clumps. The nuclear polarity is dis- rupted, and marked cell pleomorphism and aberrant mitosis are present. Structural alter- ations may occur such as budding or branching crypts or tubules, with epithelial septa and back to back glands, and cribiform septa 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 1–5 and by categories 6–12 of the Vienna and their correct identification for future molecular research.

The viewpoint of Dixon appears to be in concert with the Western 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 work of Johan Strauss has succeeded in orchestrating not only fiddlers but also workers engaged in the microscopic diagnosis of gastrointestinal epithelial neoplasia.

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References

BOOK REVIEWS

Dynamic Radiology of the Abdomen: Normal and Pathologic Anatomy, 5th edn


This is the fifth edition of Dynamic Radiology of the Abdomen: Normal and Pathologic Anatomy, a book that has become essential reading for all those aiming to be expert at abdominal imaging. Previous editions have been published in Italian, Japanese, Portuguese, and Spanish. The continued aim of the author is to present a systematic application of anatomical and dynamic principles to aid our understanding of the characteristic appearances and modes of spread of intra-abdominal disorders. Dissections and cross sectional views of cadavers are used in conjunction with a full range of imaging modalities, including plain radiographs, contrast studies, computed tomography (CT), ultrasound, magnetic resonance imaging (MRI), and endoscopic, laparoscopic, and intraoperative ultrasound.

This edition has been extensively updated with six new chapters, 180 additional pages, and more than 520 new illustrations. Subjects that are included for the first time include clinical embryology in relation to disorders that become clinically apparent in the adult, TNM staging of gastrointestinal cancers, and the manifestations of free intraperitoneal air. There are now 11 other contributing authors, but Morton Meyers is solely responsible for about three quarters of the book and many of the of the cases illustrated are reproduced from his own numerous publications.

This book is well written, superbly illustrated, and comprehensively referenced. The illustrations, particularly the extensive use of cross sectional spiral CT images, make it easier for 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 that of the other abdominal organs, and the extraperitoneal spaces is described in detail. There are excellent descriptions of the intraperitoneal spread of infections and of the peritoneal cavity. There is in a chapter on internal abdominal hernias. Pancreatic disorders and their mode of spread are described in detail, the diverse locations of pancreatic pseudocysts 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 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.

D Nolan

Artificial Nutrition Support in Clinical Practice, 2nd edn


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 skills 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 provide the nutrients by the simplest safest route acceptable to the patient.

Such teams need to develop more in the community. 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 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.

D Nolan

Dyspepsia


We all have a word of advice and the general practitioner is a handy person to ask. Getting it packaged right can be a challenge, not least when the front cover states that it is an “Indispensable guide to clinical practice”. In this unusual translation from German, two distinguished gastroenterologists have made a worthy effort to reach out and have successfully condensed most gastroenterological scenarios faced by the jobbing clinician into an attractive and accessible package. This little book packs a lot—while seeming to be a general and the theoretical, the general and the more specialist. I would recommend it as a sound basis and as a useful reference for further reading.

J Powell-Tuck
The Pelvic Floor: Its Function and Pathology

Reconstructive Surgery of the Esophagus

Clinical Governance in Gastroenterology

The Pelvic Floor: Its Function and Disorders

Gastroenterology Highlights 2001–02
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 Strepta procedure. Both Freedman's study on the association between cholecystectomy and oesophagal 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

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, Leineweberstr. 5, 79041 Freiburg/B, Germany. Tel: +49 761 15 140; fax: +49 761 13 14 339; email: sympsia@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: +420 241 445 759; 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 Beauprez, 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: beauprez@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: www.acgbi.org.uk

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