Caerulein induced pancreatitis (letter)
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Caerulein induced pancreatitis


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Osteoporosis and liver disease: additional reasons for coeliac disease screening

We read with great interest the recently published guidelines on the management of coeliac disease associated with chronic liver disease (Gut 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 2 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.1 Patients with CD are at high risk of developing low bone mineral density and bone turnover impairment,3 and osteoporosis, may have a considerable 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 the need/success of transplantation.6 7

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References

Caeerulin 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 caeuerulin induced pancreatitis and prevent trypsinogen activation in the pancreas”. We have a few comments with regard to the interpretation of the data that were obtained in this experiment.

Previous experimental work by Frossard and others have implicated HSP70 as playing a protective role in caeuerulin induced acute pancreatitis. In the present study Frossard et al showed that thermal and non-thermal stress induced by injection of the β agonist isopropenol upregulated HSP70 in the pancreas which is associated with amelioration of subseqently induced caeuerulin 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 shock stress or by injection of isoproterenol, can influence activation of inflammatory pathways during inflammation and infection7 (reviewed by van der Poll and others).8 Evidence exists that catecholamines exert anti-inflammatory effects on a number of host mediator systems, such as the cytokine network and neutrophils,9 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 proinflammatory 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 proinflamamatory 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 isopropenol enhances the production of the anti-inflammatory cytokine IL-10 which has been shown to be protective in acute pancreatitis.10 Thirdly, in endotoxemia models, β adrenergic stimulation results in reduced expression of TNF and IL-10 and IL-12.11 Exogenous and endogenous catecholamines induce the production of macrophage inflammatory protein (MIP) 1α via a beta adrenergic receptor mediated mechanism.12

References
Authors’ reply

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, cathelolamines 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 thermal protection against pancreatitis, we adopted the antisense oligonucleotide approach in another recently published experimental study (2) 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.

Using antisense oligonucleotides to HSP70, Nisoli and colleagues have also shown that the protective effects of noradrenaline against tumour necrosis factor alpha induced apoptosis in cultured rat brown adipocytes is due to nitric oxide induced HSP70 expression. In fact, cathelolamines have been used in the past to induce heat shock proteins in several experimental systems. 3,4

Westerloo et al have cited examples wherein exogenous or endogenous cathelolamines inhibit the production of inflammatory cytokines and enhance the production of interleukin 10 (IL-10), an anti-inflammatory cyto- kine that has been shown to limit the severity of pancreatitis. Unfortunately, in the studies cited expression of HSP70 was not monitored. It is entirely possible that prior thermal or non-thermal stress induce HSP70 which may in turn lead to the enhanced production of anti-inflammatory factors and attenuation of proinflammatory cytokines. Indeed this has been shown to be the case in many experimental systems, including animal models of sepsis (reviewed by Brummer-Smith and colleagues (5)). Moreover, mycobacterial HSP70 has been shown to prevent adjuvant arthritis and induce IL-10 producing T cells. 6

The mechanism(s) by which HSP70 might protect against caerulein induced pancreatitis is not yet known. Experiments examining the relationship between HSP70 and the inflammatory cascade induction during caerulein induced pancreatitis are currently underway.

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References


Pathology and cost effectiveness of endoscopic 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 pro- grammes?

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- sidered the targets of additional sampling. 7 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 the risk of cancer to be higher within the target group 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, 8,9 from regenerative changes (with a nearly null cancer risk) to non-invasive neoplasia (which carries, by definition, predisposition 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 question- able 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 altera- tions pre-curying invasive neoplasia. Most importantly, the high prevalence of metaplas- tic lesions within subjects who will never develop adenocarcinoma exclude (non-extensive) intestinal metaplasia as the proper target of surveillance (or at least at present). In the natural history of epithelial tumours, the term dysplasia identifies a lesion that car- ries biological alterations comparable with that of full fledged cancer but lacking stromal invasion.10 Recently, the term “dyspla- sia” has been replaced by “non-invasive neoplasia”, which more clearly identifies such a lesion as the most advanced alteration antecedent to invasive adenocarcinoma. 11 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 enrolment</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></td>
<td>EGC</td>
<td>AGC</td>
</tr>
<tr>
<td>Low grade non-invasive neoplasia (99 cases)</td>
<td>48 (38–80)</td>
<td>5</td>
</tr>
<tr>
<td>High grade non-invasive neoplasia (25 cases)</td>
<td>30 (11–72)</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>33 (13–80)</td>
<td>19</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 emphasising 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 Italian Office for Instruction and University Research (MTUR: Chiron project July 2000).

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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>
<th>[95% CI]</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18 years at last height</td>
<td>0.68</td>
<td>-1.17 to -0.19</td>
<td>-0.57</td>
<td>-1.16 to 0.02</td>
</tr>
<tr>
<td>Deficit (cm)</td>
<td>-5.9</td>
<td>-8.2 to -3.4</td>
<td>-5.5</td>
<td>-8.6 to -2.4</td>
</tr>
<tr>
<td>n</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>16 years at last height</td>
<td>0.73</td>
<td>-1.42 to -0.04</td>
<td>-0.65</td>
<td>-1.48 to 0.18</td>
</tr>
<tr>
<td>Deficit (cm)</td>
<td>-5.3</td>
<td>-8.6 to -2.0</td>
<td>-5.1</td>
<td>-8.9 to -1.38</td>
</tr>
</tbody>
</table>

Data are mean (95% confidence interval).

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 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 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 “postpubertal” as 16 years, in contrast with Alemzadeh et al who used 22 years. The population we serve is ethnically diverse and therefore we and the data are therefore not presented. 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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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 56% 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 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 transferin saturation of >300 µg % ferritin of >300 µg. Both parameters were elevated in 15 (75%) individuals, with three having an isolated elevated transferrin saturation and two an isolated elevated ferritin. Of the 22 females (mean age 44) identified, 18 (81%) had evidence of biochemical iron overload, of whom 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 Dublin 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 appeared 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 who 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 to unnecessary 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 histopathological training in Japan, the concept of intraepithelial carcinoma (that is, carcinoma in situ) and of intramucosal carcinoma appeared natural. Although during the 1980s a number of discussions in the Western pathologists appeared reluctant to accept such controversial notions, the discussion became less intense during the second day, and at the end of the conference it 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, remains elusive whether carcinoma in situ is preceded by high grade dysplasia or develops without a prodro- 

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, hyperchromatic nuclei, and regular nuclear membrane whereas carcinoma in situ displays large vesicular nuclei, irregular chromatin, and scalloped nuclear membranes. Bridges of nucleated associated chromatin reaching irregular chromatin deposits are seen in the nucleus of the former. Nuclear 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 cribiform 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 represented by the categories 4.2, 4.3, and 5.1 of the Vienna1 and their correct identification for future molecular research.

The viewpoint of Dixon appears to be in conflict with the developing view that some gastrointestinal pathologists who are willing to embrace this new “doctrine” in order to acquire accurate information on the histological steps followed.
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), ultrason, 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 other abdominal organs, and the extraperitoneal spaces is described in detail. There are excellent descriptions of the intraperitoneal spread of infections and of tumors. There is also 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.

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 must now be the colliagion's criterion edition. It is an edited collation in which British authors dominate but it also includes contributions from key players from continental teams, a demand which seamlessly combine, in a cost effective and practical manner. It is therefore comprehensible, with a sensible mixture of the practical and the theoretical, the general and the more specialist. I would recommend it as a sound basis and as a useful resource for further reading.

D Nolan
Clinical Governance in Gastroenterology


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 glub but 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 or two omissions—for example, the lack of evidence that the authors have their ear to the ground, shifting though it may be. It would be deluding to assume that all we know about gastroenterology, and that matters, to patient care can be compressed into 94 pages, but there is sufficient to keep governance types well busy. The information is well accessible but this is, of course, not a textbook. The chapter on standards, for example, is a disappointment 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 strain incontinence. The book contains 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 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 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 putting together the knowledge of all the specialties involved in the pelvic floor.

P Denis

Gastroenterology Highlights 2001–02

Edited by E Quiagley. 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 disease, small bowel, to complications of liver disease, endoscopy, and colorectal cancer prevention and screening. The aim is to discuss key papers and put them into context. How many chapters, about 20 papers are reviewed but the actual numbers range from 16 to 30. Most chapters also review one or two abstracts. The
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 for colorectal cancer. The steps are identical for the first two groups: change in lifestyle, diagnostic. 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 cholecystectomy 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 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, 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 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