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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 osteoporosis associated with chronic liver disease (Gut 2002;50(suppl I):11–9). However, we would like to add a few words of comment. Associations between coeliac disease (CD) and primary biliary cirrhosis in particular and other autoimmune liver diseases in general have been reported.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.4 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.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.6–8

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References

Caeerulien induced pancreatitis

We have read with interest the article by Frossard et al (Gut 2002;50:78–83) entitled “Both thermal and non-thermal stress protect against caerulein induced pancreatitis. In the present study Frossard et al showed that thermal and non-thermal stress induced by injection of the β agonist isoproterenol upregulated HSP70 in the pancreas which is associated with amelioration of sub-sequently induced caerulein pancreatitis. The authors hypothesise that the protective effects on pancreatitis severity caused by thermal and non-thermal stress may be mediated by HSP70. We believe however that both heat shock stress and non-thermal stress can stimulate several other anti-inflammator 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).7 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. Second, in animal models of endotoxaemia, pretreatment with isoproterenol enhances the production of the anti-inflammatory cytokines IL-10 which has been shown to be protective in acute pancreatitis.8 Thirdly, in endotoxiaemia models, β adrenergic stimulation results in reduction of levels of CC chemokines.9 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 tryptophan in the pancreas, it must be noted that recent evidence suggests that neutrophils and possibly cytokines can also influence trypsinogen activation. Therefore, the reduction in trypsinogen activation shown in their study might be unrelated to HSP expression and may be explained by the reduction of inflammation due to β adrenergic effect.7–9

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
PostScript


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, 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 thermal and non-thermal 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 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 heat shock protein 70 expression. *J Pharmacol Exp Ther* 2001;301:1421–30.


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 all felled gland 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 antece- dent to invasive adenocarcinoma. 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>Low grade non-invasive neoplasia (99 cases)</td>
<td>48 (38–80)</td>
<td>1.5 (1–2)</td>
</tr>
<tr>
<td>High grade non-invasive neoplasia (25 cases)</td>
<td>30 (13–72)</td>
<td>2 (1–4)</td>
</tr>
<tr>
<td>Total</td>
<td>33 (13–60)</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 probably coexisting 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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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+1))/2 and for female patients by (maternal height+ (paternal height+1))/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”嵋16 years. In contrast with Alemzadeh et al that adult height of patients with onset of symptoms before the age of 16 is reduced12. Using age of onset of symptoms as a proxy for puberty, we found no significant difference in final height between those with pre- and postpubertal onset of symptoms. This is in contrast with the findings of Alemzadeh et al and may be because of differences in the upper age limit of “postpuberty” (16 versus 22 years).

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

<table>
<thead>
<tr>
<th></th>
<th>All</th>
<th>Prepubertal</th>
<th>Postpubertal</th>
<th>Pre vs post (p value)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n=16</td>
<td>n&gt;16 at last height</td>
<td>n&lt;16 at last height</td>
<td></td>
</tr>
<tr>
<td>Deficit (cm)</td>
<td></td>
<td>0.68</td>
<td>-1.17 to -0.19</td>
<td>0.057 (-1.6 to 0.02)</td>
</tr>
<tr>
<td></td>
<td>n</td>
<td>5.9</td>
<td>20</td>
<td>7</td>
</tr>
<tr>
<td>Deficit (cm)</td>
<td></td>
<td>-0.73</td>
<td>-1.42 to -0.04</td>
<td>0.065 (-1.48 to 0.18)</td>
</tr>
<tr>
<td></td>
<td>n</td>
<td>5.3</td>
<td>13</td>
<td>3</td>
</tr>
</tbody>
</table>

Data are mean (95% confidence interval).

www.gutjnl.com
In their paper, Ryan et al. of clinical not biochemical Underdiagnosis of hereditary deficit. Also, there is no information on the secular trend which will underestimate the This formula does not include a correction for with the postpubertal group. Furthermore, did not calculate the corrected height SDS the authors that the differences are possibly I think that this study is clear. We agree with Author's reply... References

Buckler J, Azooz O, El

Hildebrand H

Hubner C, Croft

Thorburn D, A J Morris, A J Stanley


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 can 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 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 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. 1 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 \( \geq 45\% \) (transferrin detected immunochromatically) or a serum ferritin of \( \geq 300 \mu g \). 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, of which 10 (45%) had 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 these 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 appeared to be as 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. 2 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 “Gastro- intestinal epithelial neoplasia: Vienna revisited” 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 terminol- ogy as it could lead to misinterpretation by surgeons and to unnecessary surgical inter- vention. The Vienna classification 1 has introduced new avenues to the understanding of the process of carcinogenesis in the gastrointestinal 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 intramuscular carcino- noma appeared natural. Although during the period of discussions of the Vienna classifica- tion and comparable with that reported from Dub- lin, the Vienna group appeared reluctant to accept such controversial notions, the discussion became less intense during the second day, and at the end a “compromise” was reached, gaining finally the pages of this journal. 2

The Vienna classification 1 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 dyspla- sia. On the other hand we remain 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- scular carcinoma. If those microscopic realities of colorectal carcinogenesis are being ignored, how are we going to learn in a correct sequen- tial 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 1 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 promoting histopathological descriptions (criteria) for each one of the various categories proposed in Vienna. 3

“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. 4 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 boundary, and scalloped nuclear membranes. Bridges of nucleoli associated chromatin reaching irregular chromatin deposits are seen in the nuclear membrane. Irregular chromatin are also seen connecting angular chromatin clumps. The nuclear polarity is dis- rupted, and marked cell pleomorphism and aberrant mitoses are present. Structural al- terations may occur such as budding or branching crypts or tubules, with epithelial septa and back to back glands, and cribriform septa of epithelial cells in clusters and sheets. Those structures are confined to the basement membrane of the epithelial layer. 2 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 3a–2 of the Vienna 1 and their correct identification for future molecular research.

The viewpoint of Dixon appears to be in contrast with the viewpoint of British 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 gastro-intestinal tract. Only then will we be able to translate such events into molecular terms. At present, John Johann Struass 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

Dyspepsia

We all have a word of advice and the general practitioner is a handy reference point to. 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 transatlantic collaboration, 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 handy reference it is in fact a repository of facts and information and I confess to dipping into it often to confirm matters or to cull material for a presentation. For example, a map indicating the worldwide prevalence of Helicobacter pylori and tracings illustrating lower oesophageal pressures during swallowing enliven concepts glossed over in other publications.

None the less, the pedigree of the authors does tell on them in some of the sections. Hardly has the invisible ink (from the primary care practitioner’s viewpoint) dried on the Rome II definitions before we are exhorted in the chapter on “Functional dyspepsia” to differentiate, on clinical grounds, ulcer-like and dysmotility-like dyspepsia. This is accompanied by advice on tailored regimens based on acid suppression or dysmotility agents. In real life, successful management, one fears, is more likely to rely on serendipity than acumen but there can be no harm in thinking constructively. The “Functional dyspepsia” chapter did rather throw me: the first line defines it as discomfort or pain centred in the epigastrium; luckily I kept at it and further down the page was informed that this only applies where “common or uncommon structural, biochemical or infectious agents have been excluded”. Actually, this angst, and that of further subdividing functional dyspepsia, applies only to those who have heard of Rome II. Most primary care practitioners can thus relax. So can our gastroenterology colleagues who might otherwise be requested to confirm an exact diagnosis of functional dyspepsia in younger patients. Avoiding endoscopy here also avoids opprobrium; alas, the diagnosis of functional dyspepsia must remain in the mind rather than in the investigation suite. As the old saying goes, if the book which one needs to receive gratis although I do recommend its purchase if necessary. It is eminently suited to distribution through the good will
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 book 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 choice and availability of the oesophageal substitute and route to bridge the gap, a chapter is then devoted to each of the principal reconstructive techniques using stomach, colon, and jejunum, as well as the use of prosthetic tubes. Each of the chapters goes into considerable detail about relevant surgical anatomy, physiology, operative technique, and complications and their management.

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

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 concerning one of the clinical problems of the pelvic floor, associating the anterior and posterior components, including physiology, anatomy, diagnostic imaging, surgery, nursing, and psychology.

The previously published book by these authors was entitled Coloproctology and the Pelvic Floor—coloproctology has disappeared from the title of their new book, indicating that it is no longer possible to approach the posterior pelvic floor disorders without studying the pelvic floor as a whole. In this way, this new title by itself is a very strong message. However, the reader might be a little disappointed if he looks for how to treat, for example, a patient suffering from both anal and urinary incontinence, or a patient complaining of urinary stress incontinence and posterior pelvic floor dysfunction inducing straining at stools. The book contains a number of excellent summaries 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 physiopathology 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 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 awaiting for these future multidisciplinary clinics of “perieneology”, 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 trying to put together the knowledge of all the specialties involved in the pelvic floor.

P Denis

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 anticipates 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 correct and timely. The Publishers have put it together well, the authors have got that aspect of health economics correct and timely.

P Hungin

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, pancreas, and small bowel, to complications of liver disease, endoscopy, and colorectal cancer prevention and screening. The aim is to discuss key latest developments in the latest research and 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
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 cholecystectomy and oesophageal adenocarcinoma as well as Schnell’s report on non-surgical management of Barrett’s oesophagus with high grade dysplasia, were reviewed in the oesophagus chapter and again in the chapter on gastrointestinal cancer. Tighter editing could have avoided this duplication as the only test for colorectal cancer” was “in” or “out”. This statement was not referenced. The style is easy to read. 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 non-surgical management of Barrett’s oesophagus with high grade dysplasia, were reviewed in the oesophagus chapter and again in the chapter on gastrointestinal cancer. Tighter editing could have avoided this duplication as the only test for colorectal cancer” was “in” or “out”. This statement was not referenced.

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, Leinenwebstr. 5, 79041 Freiburg/Breisgau, Germany. Tel: +49 761 15 140; fax: +49 761 13 14 339; email: symposia@falkfoundation.de; website: www.falkfoundation.de

International Symposium on Viral Hepatitis and Liver Disease
This conference will take place on 6–10 April 2003 in Sydney, Australia. Further information: ISVHLD 2003 Congress Managers, GPO Box 128, Sydney NSW 2001, Australia. Tel: +612 9262 2277; fax: +612 9262 3135; email: isvhld@tourhosts.com.au; website: www.tourhosts.com.au/isvhld

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: acgb@asgb.org.uk; website: www.acgb.org.uk

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