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: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.3 Patients with CD are at high risk of developing low bone mineral density and bone turnover impairment,4 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 for successful transplantation.6

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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 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 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 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 Poll8). 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.9 Thirdly, in endotoxemia mice models, β adrenergic stimulation results in reduction of levels of CC chemokines.10

Participants were recruited at the Departments of General Internal Medicine, and Internal Medicine and AIDS, Academic Medical Centre, University of Amsterdam, the Netherlands.

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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 with the possibility that (in the induction of HSP70) noradrenaline could have exerted a protective role played by HSP70. To prove that a cause-effect relationship exists between these two events, 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 is not limited to those of full fledged cancer but lacking those of full fledged cancer but lacking important, the high prevalence of metaplasia (regardless of its histological spectrum) with the highest risk of cancer evolution is not only biologically questionable but, and this is even worse, it may result in inappropriate patient management. While intestinal metaplasia represents the most common background of stomach cancer, the term “intestinalisation” per se does not carry the phenotypic and genotypic alterations pre-carrying its risk of cancer evolution.

The natural history of epithelial tumours, the term dysplasia identifies a lesion that carries biological alterations comparable with those of full fledged 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 antecedent to invasive adenocarcinoma. Since 1985, we have prospectively followed up a

References


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-endoscopically surveyed patients. As a result, this study demonstrates 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” requirements (that is, a protocol of gastric biopsy sampling) to be satisfied when carrying out the upper endoscopy procedure? Second, 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 population undergoing surveillance than in the general population. In the Birmingham study, such a prerequisite does not seem to have been assumed in the study design. Endoscopy surveillance definitively included the whole spectrum of abnormalities described in gastric carcinogenesis, 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 histochenal phenotype) with the highest risk of cancer evolution is not only biologically questionable 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-carrying its risk of cancer evolution.

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Table 1

<table>
<thead>
<tr>
<th>Gastric cancer detected after follow up longer than 12 months</th>
<th>Gastric cancer detected within 12 months from initial diagnosis</th>
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<tbody>
<tr>
<td><strong>Histology at enrollment</strong></td>
<td>Follow up (months)</td>
</tr>
<tr>
<td>Low grade non-invasive neoplasia (99 cases)</td>
<td>GC-nos</td>
</tr>
<tr>
<td>48 (38–80)</td>
<td>1.5 [1–2]</td>
</tr>
<tr>
<td>High grade non-invasive neoplasia (25 cases)</td>
<td>3</td>
</tr>
<tr>
<td>30 (13–72)</td>
<td>2 [1–4]</td>
</tr>
<tr>
<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 at 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 the long term follow up support the premalignant significance of 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 9098-96) and granted by the Italian Office for Instruction and University Research (MTUR: Clinion 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 standards using the method described by Alemzadeh et al (mean British male adult height of 176.0 cm (SD 6.3) and female adult height of 163.6 (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 have confined this analysis to Caucasians, as such as site of disease activity and therapeutic intervention.

Table 1

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Prepubertal</th>
<th>Postpubertal</th>
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<tbody>
<tr>
<td>&gt;16 years at last height</td>
<td>0.68</td>
<td>0.57</td>
</tr>
<tr>
<td>Deficit (cm)</td>
<td>5.9</td>
<td>5.5</td>
</tr>
<tr>
<td>&lt;18 years at last height</td>
<td>0.73</td>
<td>0.65</td>
</tr>
<tr>
<td>Deficit (cm)</td>
<td>5.3</td>
<td>5.1</td>
</tr>
</tbody>
</table>

Data are mean (95% confidence interval).

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.

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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 pubertal group compared with the postpubertal group. Furthermore, they used another formula for target height. Furthermore, it appears that these individuals have iron overload. This formula does not include a correction for secular trends which underestimate the deficit. Also, there is no information on the effect of corticosteroid use.

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Underdiagnosis of hereditary haemochromatosis: reflects lack of clinical not biochemical 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 to be C282Y homozygotes by genetic testing were identified as C282Y homozygous state is high at approximately 1%, which is dependent on the population tested. However, they found 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. 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 initial period of discussions, some Western pathologists appeared reluctant to accept such controversial notions, the discussion became less intense during the second day, and at the end of the meeting 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 adenosomas 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- mic phase. By the same token we do not know whether carcinoma in situ antedates intramus- cosal carcinoma. If those microscopic realities of colorectal carcinogenesis are being ignored, how are we going to learn in a correct sequential fashion the intricate molecular footsteps that telescope from dysplasia to submucosal carcinoma? As that Pandora box is being presented to pathologists we should treasure it by opening it little by little.

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

“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 chromatin, and scalloped nuclear membranes. Bridges of nucleoli associated chromatin reaching irregular chromatin deposits are seen along the nuclear membrane. The nuclei of carcinomatous chromatin are also seen connecting angular clumps. The nuclear polarity is disrupted, and marked cell pleomorphism and aberrant mitosis are present. Structural alterations may occur such as budding or branching crypts or tubules, with epithelial septa and back to back glands, and cribriform growth of epithelial cells in clusters and sheets. Those structures are confined to the basement membrane of the epithelial layer. But surprisingly, despite those differences, high grade dysplasia and carcinoma in situ are still being regarded as synonyms in the Western literature.

The present discussion is beyond the usefulness of the Vienna classification as a tool for proper treatment; the discussion aims to point out our present lack of knowledge regarding the histogenesis of lesions repre- sented by categories 4.2, 4.3 and 5.1 of the Vienna classification and their correct identification for future molecular research.

The viewpoint of Dixon appears to be in concert with the decision of some Western pathologists who are willing to embrace this new “doctrine” in order to acquire accurate information on the histological steps followed.
by early neoplastic lesions of the gastrointestinal tract. Only then will we be able to translate such events into molecular terms. Only then will we be able to translate such events into molecular terms. Only then will we be able to translate such events into molecular terms. Only then will we be able to translate such events into molecular terms. Only then will we be able to translate such events into molecular terms.

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 other abdominal organs, and the extraperitoneal spaces is described in detail. There are excellent descriptions of the intraperitoneal spread of infections and of making it easier for the radiologist to interpret the radiological appearances of free intraperitoneal air on the supine abdominal radiograph and have also described a number of new views. 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.

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 skill and knowledge of dieticians, nutrition nurse specialists, pharmacists, and doctors. Teams like this are the key to excellent patient centred nutritional care and lead to overall hospital strategies for nutritional support which seamlessly combine, in a cost effective way, the use of food and supplements with artificial feeding. The choice of the nutrients by the simplest safest route acceptable to the patient. Such teams need to develop more in the community too. We all have a word of advice and the general practitioner is a handy person to work with. 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. Hardy has the invisible ink (from the primary care practitioner's viewpoint) dried on the Rome II definition here and has been exhorted in the chapter on "Functional dyspepsia" to differentiate, on clinical grounds, ulcer-like and dysmotility-like dyspepsia. It is a common complaint which has been the subject of some excellent studies which have also described a number of new views. 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.

The "Functional dyspepsia" chapter did rather throw me: the first line defines it as distressful and distressful. Avoiding endoscopy here also avoids the need to receive gratis although I do recommend its purchase if necessary. It is eminently suited to distribution through the good will of the more enlightened hospitals form teams, a demand develops for a comprehensive textbook. This one is widely seen as one of the best and it now that the collaborations has been comprehensively topped, 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

Dyspepsia

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J Powell-Tuck
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 that hopefully sensible answer is that clinical governance might ensure “uniform standards” across heterogeneous NHS practices, reassure the public and, in any case, it seems here to stay—at least for the moment.

It still baffles me as to what differences there are between excellent clinical practice and practice by clinical governance—presumably the latter is not meant to be quite as good, but will do. There seems to be a clamour for “acceptable” standards and this anticipating being in charge of ensuring we do our jobs properly and services are commissioned effectively. There are one or two omissions—for example, the lack of mention of varices as a cause of acute bleeding—but each chapter does have a section on health economics correct and timely. The information is well accessible but this is, of course, not a textbook. The chapter on standards describes “A stoma is an artificial opening of the bowel on to the external skin”, sheer gold dust to the doctor who forgot to attend the surgery lectures or to the non-clinical manager now taking the governance types well busy. The information makes significant contributions to the external skin”, sheer gold dust to the doctor who forgot to attend the surgery lectures or to the non-clinical manager now taking the governance types well busy. The information makes significant contributions to the

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

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

Gastroenterology Highlights 2001–02

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

Gastroenterology Highlights 2001–02 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, and gall bladder, to complications of liver disease, endoscopy, and colorectal cancer prevention and screening. The aim is to discuss key issues and to present evidence to practitioners in the relevant fields. The book is divided into nine chapters, each containing 10 papers and printed in two sections. In each chapter, 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 micropheres were discussed, but not the Stretta procedure. Both Freedman’s study on the association betweencholecystectomy 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 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, Leinenweberstr. 5, 79041 Freiburg/B, 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: ISVHL 2003 Congress Managers, GPO Box 128, Sydney NSW 2001, Australia. Tel: +612 9262 2277; fax: +612 9262 3135; email: isvhl@tourhosts.com.au; website: www.tourhosts.com.au/isvhl

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: +420 241 445 739; fax: +420 241 445 806; email: veronika@congressprague.cz

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

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

Gastroenterology and Endotherapy: XXIst European Workshop

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

www.gutjnl.com

www.falkfoundation.de