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):1–11). 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 In addition, it has been suggested that these individuals should be considered as an at risk group for whom serological testing for CD is indicated. Patients with CD are at high risk of developing low bone mineral density and bone turnover impairment,2 and it has been shown that adherence to a gluten free diet has a significant positive impact on these parameters.3 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.4

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References

Caezuline induced pancreatitis

We have read with interest the article by Fossard et al (Gut 2002;50:78–83) entitled “Both thermal and non-thermal stress protect against caerulein induced pancreatitis. In the present study Fossard 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-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 caeulemines, 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 et al.1). Evidence exists that caeruleamines 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. Caeulemines, 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, caeruleamines exert anti-inflammatory effects on the cytokine network by inducing the production of pro-inflammatory 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 pro-inflammatory 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.3 Thirdly, in endotoxaemia models, β adrenergic stimulation results in reduction of levels of CC chemokines.4 Fourthly, neutrophil migration to the pancreas, one of the hallmark signs of acute pancreatitis, towards chemotactic stimuli such as C5α 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 tryptophan activation. Therefore, the reduction in tryptophan activation shown in their study might be unrelated to HSP expression and may be explained by the reduction of inflammation due to β adrenergic effects.5

Therefore, we believe that the conclusion by Fossard 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 caeruleamines.

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

When we embarked on this project, we were also concerned that all the stresses that result in the induction of HSP70 may have other non-HSP related effects and did mention this in our discussion. At that point we did not have the tools to show the crucial protective role played by HSP70.

To prove that a cause-effect relationship exists between noradrenaline and protection against pancreatitis, we adopted the antisense oligonucleotide approach in another recently published experimental study6 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.


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 full fledged cancer but lacking stromal invasion.2 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.4 Since 1985, we have prospectively followed up a

Table 1 Invasive cancer detected during follow up of non-invasive gastric neoplasia

<table>
<thead>
<tr>
<th>Histology at enrollment</th>
<th>Gastric cancer detected after follow up longer than 12 months</th>
<th>Gastric cancer detected within 12 months from initial diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Follow up [months]</td>
<td>EGC</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 (13–72)</td>
<td>19</td>
</tr>
<tr>
<td>Total</td>
<td>33 (13–80)</td>
<td></td>
</tr>
</tbody>
</table>

EGC, early gastric cancer (that is, UICC pathological stage II); AGC, advanced gastric cancer (that is, UICC pathological stage II-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 preclinical significance of non-invasive neoplasia in patients 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 SD 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 6.5) and female adult height of 163.6 (SD 5.7)). “Target height” was calculated for male patients by (paternal height+ (maternal height+13))/2 and for female patients by (maternal height+ (paternal height+13))/2 cm. “Prepubertal” children were defined as males and females with onset of symptoms at <13 and <11 years, respectively. We defined the upper limit of “postpuberty” as age of 16 years. However, the calculated adult height of patients with onset of symptoms before the age of 16 is reduced. 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).

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 inconsequential this includes a subset with much more significant growth impairment. A better understanding of the mechanisms underlying growth failure is required to determine whether there is an identifiable group of children that may benefit from early and more intensive immunosuppression and/or nutritional therapy. We agree with Alemzadeh et al that only larger (population based) studies will have the power to determine the effect of factors such as site of disease activity and therapeutic intervention.

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Table 1

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

<table>
<thead>
<tr>
<th></th>
<th>All</th>
<th>Prepubertal</th>
<th>Postpubertal</th>
<th>Pre v post</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;16 years at last height</td>
<td>&gt;0.68 (−1.17 to −0.19)</td>
<td>−0.57 (−1.16 to 0.02)</td>
<td>−1.01 (−1.86 to −0.16)</td>
<td>p=0.41</td>
<td></td>
</tr>
<tr>
<td>Deficit (cm)</td>
<td>&lt;5.9 (−8.2 to −3.4)</td>
<td>−5.5 (−8.6 to −2.4)</td>
<td>−7.0 (−8.8 to −5.3)</td>
<td>p=0.62</td>
<td></td>
</tr>
<tr>
<td>n</td>
<td>20</td>
<td>7</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;18 years at last height</td>
<td>&gt;0.73 (−1.42 to −0.04)</td>
<td>−0.65 (−1.48 to 0.18)</td>
<td>−1.07 (−2.12 to −0.02)</td>
<td>p=0.58</td>
<td></td>
</tr>
<tr>
<td>Deficit (cm)</td>
<td>&lt;5.3 (−8.6 to −2.0)</td>
<td>−5.1 (−8.9 to −1.38)</td>
<td>−6.2 (−11.08 to −2.38)</td>
<td>p=0.84</td>
<td></td>
</tr>
<tr>
<td>n</td>
<td>16</td>
<td>13</td>
<td>3</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Data are mean (95% confidence interval).

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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 prepubertal group compared with the non-prepubertal group. Furthermore, they used another formula for target height. This formula does not include a correction for secular trends which will underestimate the secular trend which will underestimate 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 underestimation of biochemical iron overload 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. Thus, 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. They observed 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 determine 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 first day of discussions other Western pathologists appeared reluctant to accept such controversial notions, the discussion became less intense during the second day, and at the end a consensus was reached, gaining finally the pages of this journal.1

The Vienna classification1 dismissed the concept of dysplasia from that of carcinoma in its earlier forms. After many years of studying adenomas we now know that low grade dysplasia may progress to high grade dysplasia. On the other hand, there remains elusive whether carcinoma in situ is preceded by high grade dysplasia or develops without a prodromic phase. By the same token we do not know whether carcinoma in situ antedates intramucosal carcinoma. If those microscopic realities of colorectal carcinogenesis are being ignored, how are we going to learn in a correct sequential fashion the intricate molecular footsteps that telescope from dysplasia to submucosal carcinoma? As that Pandora box is being presented to pathologists we should treasure it by opening it little by little.

One criticism of the Vienna classification may be that although various categories of neoplasia were listed, the histopathological criteria for each one of the lesions were not verbalised, thus postponing the opportunity for its worldwide acceptance. Notwithstanding, some Western pathologists have started to herald the new “doctrine” by proposing histopathological descriptions (criteria) for each one of the various categories proposed in Vienna.2

“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.1 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 nucleoli, and scalloped nuclear membranes. Bridges of nucleolus associated chromatin reaching irregular chromatin deposits are seen in the nuclear membrane of dysplasia whereas carcinoma in situ 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 of 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.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 represented by categories 2–5 of Vienna1 and their correct identification for future molecular research.

The viewpoint of Dixon appears to be in concert with the develop in Western pathologists who are willing to embrace this new “doctrine” in order to acquire accurate information on the histological steps followed

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by early neoplastic lesions of the gastro-
testinal tract. Only then will we be able to
translate such events into molecular terms.

As the more enlightened hospitals form
teams, a demand develops for a comprehen-
sive textbook. This one is widely seen as one
of the best and it now that the collaboration is
revised. It is an edited collation in which Brit-
ish authors dominate but it also includes con-
tributions from key players from continental
Europe making it representative of much of
ESPEN. As such it unsurprisingly contains
some excellent chapters and others which are
less so; the balance is good.

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for the endoscopist. Endoscopy

BOOK REVIEWS

Dynamic Radiology of the Abdomen: Normal and
Pathologic Anatomy, 5th edn
M A Meyers. Germany: Springer-Verlag,

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 under-
standing 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 illus-
trated, 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 patho-
logical anatomy of the different parts of the
abdominal cavity, the liver, the biliary and
abdominal organs, and the extraperitoneal spaces
are described in detail. There are excellent de-
scriptions of the intraperitoneal spread of infections and of making it representative.

D Nolan
Artificial Nutrition Support in
Clinical Practice, 2nd edn
Edited by J Payne-James, G Grime, D Silk.
Greenwich Medical Media, £125. ISBN 1-
90015-97-9.

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 malnutri-
tion, 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 fol-
lowed efforts to improve nutrition in hospitals
through food improvement, supplement pro-
vision, 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 gastroenter-
ologists.

It is widely acknowledged that the ap-
proach to nutritional care is best made via a
multiprofessional team which combines the
skill and knowledge of dieticians, nutrition
specialists, pharmacists, and doctors.
Teams like those are the key to excellent patient
centred nutritional care and it is advisable to
undertake hospital strategies for nutritional support which seamlessly combine, in a cost effective
way, the use of food and supplements with
artificial feeding to provide the nutrients by
the simplest safest route acceptable to the patient.
Such teams need to develop more in the com-
 community too.

As the more enlightened hospitals form
teams, a demand develops for a comprehen-
sive textbook. This one is widely seen as one
of the best and it now has the collaboration is
revised. It is an edited collation in which Brit-
ish authors dominate but it also includes con-
tributions from key players from continental
Europe making it representative of much of
ESPEN. As such it unsurprisingly contains
some excellent chapters and others which are
less so; the balance is good.

J Powell-Tuck
Dyspepsia
M J Lancaster Smith, K L Koch. UK: Health

We all have a word of advice and the general
practitioner is a handy person to do this 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 usual translation, dyspepsia, in the usual
type of and context of differentiation, two
distinguished gastroenterologists have made
a worthy effort to reach out and have
successfully condensed most gastroentero-
logical 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 swallow-
ing 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 dys-
pepsia. This is accompanied by a list of tailored regimens based on acid suppression
dysmotility agents. In real life, successful
management, one fears, is more likely to be
related to 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 discom-
fort or pain related in the epigastrium; luckily
I kept at it and further down the page was
informed that this only applies where “com-
mon or uncommon structural, biochemical or
infectious agents have been excluded” – Actu-
ally, this angst, and that of further subdivid-
ng 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 other-
wise be requested to confirm an exact diagno-
sis of functional dyspepsia in younger pa-
tients. Avoiding endoscopy here also avoids
opprobrium; alas, the diagnosis of functional
dyspepsia must remain in the mind rather
than in the investigation suite.

Now one needs to consider a book which one
needs to receive gratis although I do recom-
pense if necessary. It is eminently
suiting to distribution through the good will

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

P Hungin

Reconstructive Surgery of the Esophagus


Oesophageal disease brings together many disciplines within the field of gastroenterology and the book is aimed primarily at the specialist oesophageal surgeon. Reconstruction of the oesophagus following resection for benign or malignant disease is one of the most challenging surgical procedures currently performed, and the oesophagus being such an unforgiving organ increases that challenge. Rather surprisingly, this is the first truly authoritative and evidence based volume to be devoted completely to this topic.

Dr Ferguson was trained at the University of Chicago under doyens of oesophageal surgery including Skinner, Belsey, and DeMeester, and has continued the tradition of that line 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 oesophagectomy and the elements available of the oesophageal substitute and route to bridge the gap, a chapter is then devoted to each of the principal reconstructive techniques using stomach, colon, and jejunum, as well as the use of prosthetic tubes. Each of the chapters goes into considerable detail about relevant surgical anatomy, physiology, operative technique, and complications and their management.

Reconstructive Surgery of the Esophagus is clearly and well written. While it draws heavily on the author's considerable experience, one of the attractions of this book is that it is clearly evidence based, and as well as being liberally referenced, the key references and conclusions are highlighted in tabular form in each chapter. Another strong point for its predominantly surgical audience is the wealth of line drawings, which clearly define 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 colorectalology 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 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 chapters concerning pathogenesis, investigations, and treatment of the pelvic floor disorders. However, these algorithms have been constructed to treat either the anterior pelvic floor or the posterior pelvic floor, but not to treat a patient who complains simultaneously of the two parts of the pelvic floor. It was perhaps because the book was initially so promising and the subsequent chapters devoted to each 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-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 “perineology”, it was probably not the time to furnish algorithms in this particular edition of The Pelvic Floor concerning investigations and management of associated symptoms of the anterior and posterior pelvic floor, integrating the psychological profiles of the patients. JH Pemberton, M Swash, and MM Henry must be acknowledged and congratulated for having together brought 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 that hopefully sensible answer is that clinical governance might ensure “uniform standards” across heterogeneous NHS practices, reassure the public and, in any case, it seems here to stay—at least for the moment.

It still baffles me as to what differences there are between excellent clinical practice and practice by clinical governance—presumably the latter is not meant to be quite as good, but will do. There seems to be a clamour for “acceptable” standards and this anticipating being in charge of ensuring we do our jobs properly and services are commissioned effectively. There are one or two omissions—for example, the lack of mention of varices as a cause of acute bleeding—but each chapter does have a section on health economics for those hopefully traversing the quagmire of costs, effectiveness, care, and quality. Word has it that this book is selling well; the authors have got that aspect of health economics correct and timely.

P Hungin

Gastroenterology Highlights 2001–02

Edited by E Quigley. Health press, 2002, £15.00, colour, pp 84. ISBN 1-903743-12-6

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 small bowel, to complications of liver disease, endoscopy, and colorectal cancer prevention and screening. The aim is to discuss key lines of evidence and recommendations. The 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 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, from colorectal cancer. The steps are identical for the high risk group, with preventive surgery immediately after diagnosis. These steps are again repeated for 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 never 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 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: 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

International Symposium on Viral Hepatitis and Liver Disease
This conference will take place on 6–10 April 2003 in Sydney, Australia. Further information: ISVHLD 2003 Congress Managers, GPO Box 128, Sydney NSW 2001, Australia. Tel: +612 9262 2277; fax: +612 9262 3155; email: 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: +420 241 445 799; 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
Pathology and cost effectiveness of endoscopy surveillance for premalignant gastric lesions
M Rugge, M Cassaro, G Pennelli, V M Russo, F Di Mario and F Farinati

Gut 2003 52: 453-454
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