Osteoporosis and liver disease: additional reasons for coeliac disease screening

We read with great interest the recently published guidelines on the management of osteoporosis associated with chronic liver disease (Gut 2002;50(suppl 1):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 would like to add a few words of comment. Gut 1998;42:91–9. Associations between coeliac disease (CD) and primary biliary cirrhosis in particular and other autoimmune liver diseases in general have been reported. It would be useful to add a few words of comment. Gut 1998;42:91–9. Associations between coeliac disease (CD) and primary biliary cirrhosis in particular and other autoimmune liver diseases in general have been reported. In addition, it has been suggested that these individuals should be considered as an at risk group for whom serological testing for CD is indicated. Patients with CD are at high risk of developing low bone mineral density and bone turnover impairment, and it has been shown that adherence to a gluten free diet has a significant positive impact on these parameters. Thus we suggest that physicians caring for patients with the above mentioned liver diseases should screen them for CD in the presence of signs and symptoms suggestive of malabsorption such as osteoporosis. This seems a reasonable strategy as detection of CD will allow for a more rational therapeutic approach to the risks determined by this association. Complications due to the presence of CD, such as malnutrition, anaemia, and osteoporosis, may have a considerable impact on liver disease management and the need/success of transplantation.

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References

Caeerulein 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 and prevent trypsinogen activation in the pancreas”. We have a few comments with regard to the interpretation of the data that were obtained in this experiment. Previous experimental work by Fossard and others have implicated HSP70 as playing a protective role in caerulein induced acute 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 subsequently induced caerulein pancreatitis. The authors hypothesised 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 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 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, acts on beta adrenergic receptors to inhibit the release of proinflammatory mediators such as tumor necrosis factor (TNF), interleukin (IL)-1β, IL-2, IL-6, 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 cytokines IL-10 which has been shown to be protective in acute pancreatitis. Thirdly, in endotoxemia models, β adrenergic stimulation results in reduced levels of cytokines and proinflammatory mediators. Fourthly, neutrophil migration to the pancreas, one of the hallmarks of acute pancreatitis, towards chemotactic stimuli such as C5a and lipopolysaccharide (LPS) is reduced by administration of β agonists but also affects LPS induced neutrophil degranulation in vivo. Fifthly, with regard to the hypothesis that HSP70 prevents the activation of trypsinogen in the pancreas, it must be noted that recent evidence suggests that neutrophils and possibly cytokines can also influence trypsinogen activation. Therefore the reduction in trypsinogen activation shown in their study might be unrelated to HSP expression and may be explained by the reduction of inflammation due to β adrenergic effects. Therefore, we believe that the conclusion by 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 catecholamines.

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

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

To prove that a cause-effect relationship exists between heat stress-induced 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 HSP70 expression. In fact, catecholamines have been used in the past to induce heat shock proteins in several experimental systems.

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

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

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References

Pathology and cost effectiveness of 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 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,1,2 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 histochemical 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,1,3 “gastric intestinalisation” per se does not carry the phenotypic and genotypic alterations pre-currying invasive neoplasm. Most importantly, the high prevalence of metaplastic lesions within subjects who will never develop adenocarcinoma exclude (non-extensive) intestinal metaplasia as the proper target of surveillance protocol.

In 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 metastatic activity.7 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.8 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>Follow up (months)*</td>
</tr>
<tr>
<td>-------------------------</td>
<td>-------------------</td>
<td>---------------------</td>
</tr>
<tr>
<td>Low grade non-invasive neoplasia (P9 cases)</td>
<td>48 (38–80)</td>
<td>1.5 [1–2]</td>
</tr>
<tr>
<td>High grade non-invasive neoplasia (23 cases)</td>
<td>30 (13–72)</td>
<td>2 [1–4]</td>
</tr>
<tr>
<td>Total</td>
<td>33 (13–80)</td>
<td>1.7 [1–4]</td>
</tr>
</tbody>
</table>

EGC, early gastric cancer (that is, UICC pathological stage II); AGC, advanced gastric cancer (that is, UICC pathological stages III–IV); 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 while the 11 cases detected within one year from the original endoscopy fully demonstrated that non-invasive neoplasia frequently coexists with advanced cancer. Both of these observations could represent valid foundations in drawing a surveillance programme aimed at secondary gastric cancer prevention.

Acknowledgements

The prospective study on gastric non-invasive neoplasia has been supported by the Veneto Region (project number 909/06-99) and granted by the Italian Office for Instruction and University Research (project number 909/06-99), and by the European Commission (CIPA 95/510). The authors wish to thank Professor C. G. Corleone, DI of Pisa, for his continuous support and encouragement, and Dr. M. S. Giuli, Director of the Department of Medical Sciences, University of Pisa, for having made this study possible.

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 −1.15 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 the factor in this group of children. 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 groups, details were not available. In the case notes we cannot use 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 the age of 13 and 11 years, respectively. We defined the upper limit of “postpuberty” as 16 years, in contrast with Alemzadeh et al. who used 22 years. The population we serve is ethnically diverse and therefore we have confined this analysis to Caucasians, as the Dutch study. There was no deficit in height of the parents of 48 mothers and 46 fathers of children with childhood onset Crohn’s disease compared with the general population. Furthermore, there was no deficit in height when examined per the Dutch study. Growth failure remains a concern to our British Crohn’s patients and although the mean deficit of 5.6 cm from target height may be considered by some to be clinically inconsequential this includes a subset with much more significant growth impairment. A better understanding of the mechanisms underlying growth failure is required to determine whether there is an identifiable group of children that may benefit from early and more intensive immunosuppression and/or nutritional therapy.

We agree with Alemzadeh et al. that only larger (population based) studies will have the power to determine the effect of factors such as site of disease activity and therapeutic intervention.

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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>Prepubertal</th>
<th>Postpubertal</th>
<th>Pre v post</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;16 years at last height</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SDS</td>
<td>−0.68</td>
<td>−0.57</td>
<td>−0.10</td>
</tr>
<tr>
<td>(cm)</td>
<td>−5.9</td>
<td>−8.6</td>
<td>−5.3</td>
</tr>
<tr>
<td>n</td>
<td>20</td>
<td>20</td>
<td>7</td>
</tr>
<tr>
<td>p-value</td>
<td>0.62</td>
<td>0.67</td>
<td></td>
</tr>
<tr>
<td>&lt;18 years at last height</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SDS</td>
<td>−0.73</td>
<td>−0.65</td>
<td>−1.07</td>
</tr>
<tr>
<td>(cm)</td>
<td>−5.3</td>
<td>−8.9</td>
<td>−1.48</td>
</tr>
<tr>
<td>n</td>
<td>16</td>
<td>13</td>
<td>7</td>
</tr>
<tr>
<td>p-value</td>
<td>0.58</td>
<td>0.10</td>
<td></td>
</tr>
</tbody>
</table>

Data are mean (95% confidence interval).

This study was supported by the Crohn’s and Colitis Foundation of America and the British Surgical Association. The authors declare no conflict of interest.

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PostScript

Gut: first published as 10.1136/gut.52.3.452-a on 1 March 2003. Downloaded from http://gut.bmj.com/ on April 6, 2022 by guest. Protected by copyright.
References


Author’s reply

I think that this study is clear. We agree with the authors that the differences are possibly caused by the other “end point” of puberty and the small population in both studies. Another difference could be that the authors did not calculate the corrected height SDS (height SDS target height SDS); this may be lower for the prepubertal group compared with the postpubertal group. Furthermore, they used another formula for target height. This formula does not include a correction for secular trend, which would 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% has been diagnosed by August 2001. Of these known cases we identified 42 (20 males) C282Y homozygotes who had been diagnosed by predictive genetic testing of family members of affected probands. At diagnosis, all 20 males (mean age 46) had evidence of biochemical iron overload, defined as a transferrin saturation of >45% (transferrin detected immunochemically) or a serum ferritin of >300 µg. Both parameters were elevated in 15 (75%) individuals, with three having an isolated transferrin saturation and two an isolated elevated ferritin.

Of the 22 females (mean age 44) identified, 18 (81%) had evidence of biochemical iron overload with 10 (45%) having raised transferrin saturation and ferritin, as defined above. A further seven patients had an isolated elevation in transferrin saturation and one had an elevated ferritin alone. Only four (9.5%) C282Y homozygotes identified by family testing had no evidence of biochemical iron overload. All the individuals were women (age range 17–48 years). Unfortunately, due to the retrospective nature of the analysis, it was not possible to assess symptoms at diagnosis.

The prevalence of biochemical iron overload in our predominantly Celtic population is high and comparable with that reported from Dublin by Ryan et al. However, the proportion of subjects 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. 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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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 last decade the concept of differentiation difficulties and some pathologists appeared reluctant to accept such controversial notions, the discussion became less intense during the second day, and at the end the Vienna paper was reached, gaining finally the pages of this journal.

The Vienna classification 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, we remain elusive whether carcinoma in situ is preceded by high grade dysplasia or develops without a protracted phase. By the same token we do not know whether carcinoma in situ antedates intramusco- sal 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 intramuscosal 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 followed the recommendations of British gastrointestinal pathologists. If those microscopic realities 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 “dysplasia” and “carcinoma in situ” and their correct identification for future molecular research.

The viewpoint of Dixon appears to be in concert with the decision of some British pathologists who are willing to embrace this new “doctrine” in order to acquire accurate information on the histological steps followed.
Dynamic Radiology of the Abdomen: Normal and Pathologic Anatomy, 5th edn


This is the fifth edition of Dynamic Radiology of the Abdomen: Normal and Pathologic Anatomy, a book that has become essential reading for all those aiming to be expert at abdominal imaging. Previous editions have been published in Italian, Japanese, Portuguese, and Spanish.

The continued aim of the author is to present a systematic application of anatomical and dynamic principles to aid our understanding of the characteristic appearances and modes of spread of intra-abdominal disorders. Dissections and cross sectional views of cadavers are used in conjunction with a full range of imaging modalities, including plain radiographs, contrast studies, computed tomography (CT), 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 cases included 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 axial 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 malignancies. There is a new chapter on internal abdominal hernias. Pancreatic disorders and their mode of spread are described in detail; the diverse locations of pancreatic pseudocysts are well illustrated.

CT is now used to show the many places where free intraperitoneal air can collect in the abdomen. In recent years Cho and Baker have used this information to reassess the radiological appearances of free intraperitoneal air on the supine abdominal radiograph and have also described a number of new signs. They have brought this information together in their chapter and the result is an excellent and well illustrated contribution, providing information on an important subject that is not widely available elsewhere.

This edition of Dynamic Radiology of the Abdomen lives up the reputation established by previous editions. It should be of interest to all doctors who wish to learn more about the abdomen, particularly radiologists, oncologists, and surgeons. Radiologists who perform any type of abdominal imaging should find this latest edition invaluable and registrars training in radiology should become acquainted with it early in their career. In the preface to the first edition, Lloyd Nyhus commented on the innovative nature of this work and stated that it was an important reading source for surgeons; it likely that he would be equally enthusiastic about the latest edition.

D Nolan

Artificial Nutrition Support in Clinical Practice, 2nd edn


The provision of nutritional support for patients in hospital and in the community has derived from a heightened awareness of the impact of malnutrition on patient outcome and quality of life. Attention has focused on the prevalence of disease related malnutrition, both in terms of weight loss and weight for height, but also in terms of micronutrient deficiencies. Drives to enhance awareness of undernutrition in the community have followed efforts to improve nutrition in hospitals through food improvement, supplement provision, and artificial feeding. The need for routine nutritional assessment in clinical practice, especially in chronic disease, and its careful documentation has become ever more appreciated, particularly among gastroenterologists.

It is widely acknowledged that the approach to nutritional care is best made via a multiprofessional team which combines the skill and knowledge of dieticians, nutrition nurse specialists, pharmacists, and doctors. Teams like this are the key to expert patient centred nutritional care and lead to overall improvement in patient outcomes. The collaborative approach is most likely to be successful when the team is multidisciplinary.

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 had a diagnosis 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 the opprobrium; alas, the diagnosis of functional dyspepsia must remain in the mind rather than in the investigation suite. It is a useful little book which one needs to have on the shelf at hand, and which one needs to receive gratis although I do recommend its purchase if necessary. It is eminently suited to distribution through the good will of the NHS.

J Powell-Tuck

Dyspepsia


We all have a word of advice and the general practitioner is a handy person to talk 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. Hardy has the invisible ink (from the primary care practitioner’s viewpoint) dries on the Rome II definition before exhorting exhorting on the new chapter on “Functional dyspepsia” to differentiate, on clinical grounds, ulcer-like and dysmotility-like dyspepsia. The references to the new tailored regimens based on acid suppression or 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 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 had a diagnosis 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 the opprobrium; alas, the diagnosis of functional dyspepsia must remain in the mind rather than in the investigation suite. It is a useful little book which one needs to have on the shelf at hand, and which one needs to receive gratis although I do recommend its purchase if necessary. It is eminently suited to distribution through the good will of the NHS.

J Powell-Tuck
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 follows 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 chapter on reconstruction was written 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 oesophagectomy and the choice of replaceable 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 prostatic 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 superbly 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 as to appreciate the many challenges facing their oesophageal surgeons in this fascinating branch of gastrointestinal surgery.

A Watson

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 heterogenous 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 ensorucing 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. 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 tautology 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 speciality of pelvic floor clinics, where urologists, colorectal surgeons, specialist nurses, physiotherapists, neurologists, psychiatrists . . . work together to improve the management of pelvic floor disorders”. While we are waiting for these future multidisciplinary clinics of “perineurology”, it was probably not the time to furnish algorithms in this particular edition of The Pelvic Floor concerning investigations and management of assorted symptoms of the anterior and posterior pelvic floor, integrating the psychological profiles of the patients. JH Pemberton, M Swash, and MM Henry must be congratulated and acknowledged to bring together the knowledge of all the specialities involved in the pelvic floor.

P Denis

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 specialities 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 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 recognize that the authors have their ear to the ground, shifting though it may be.

It would be deuding to assume that all we know about gastroenterology, and that which matters to patient care can be compressed into 94 pages, but there is sufficient to keep governance types well busy. The information is well accessible but this is, of course, not a textbook. The chapter on symptoms such as “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 anticipating being in charge of ensorcurring 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. Clinical Governance in Gastroenterology 2001–02


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, and biliary tree with its complications to prokinetic drugs which are no longer, or were never, available in the UK.

P Hungin

Gastroenterology Highlights 2001–2

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P Hungin

PostScript

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.
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 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, 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.

**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: ISVHLD 2003 Congress Managers, GPO Box 128, Sydney NSW 2001, Australia. Tel: +612 9262 2277; fax: +612 9262 3155; email: isvhlhd@tourhosts.com.au; website: www.tourhosts.com.au/isvhlhd