Percutaneous drainage of echinococcal cysts

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LETTERS TO THE EDITOR

Percutaneous drainage of echinococcal cysts

EDITOR,—We read with interest the critical reply of Dr Morris (Gut 2000;47:156–7) to the letter on the use of PAIR (puncture, aspiration, injection, reaspiration) in the treatment of echinococcal cysts. He questioned the safety and efficacy of PAIR and wondered whether there was any other place for PAIR than in situations where surgery was not available. We comment on the risk of sclerosing cholangitis.

We agree with Dr Morris that injection of sclocidal agents into hydatid cysts is a potential risk for sclerosing cholangitis. However, this complication can be avoided when scolicidals are used for the correct indications. Scolicidals are not advocated at surgery because they have been associated with sclerosing cholangitis. The scolicidal probably enters pericystic liver tissue through breaks in the laminated membrane which cannot be identified by the surgeon’s eyes.1 2 Therefore, in PAIR, as a standard procedure, cystography is performed before scolicidals are used.3 Scolicidals can be safely instilled into the cyst if the laminated layer is intact and a cystobiliary fistula has been excluded.

In our experience, cystography is only appropriate in Gharbi type 1 or type 2 cysts but not in type 3 cysts (so-called mother-with-daughter cysts). In type 3 cysts, the many daughter cysts prevent the injected contrast entering the cystobiliary fistula.4 Patients with type 3 cysts, scolicidals may therefore only be used, if at all, after percutaneous evacuation of all daughter cysts and subsequent exclusion of a cystobiliary fistula by cystography. Following the procedure we treat our patients with albendazole 800 mg at breakfast and dinner, for six months. During a follow up period of at least two years, ultrasound and serology are checked at regular intervals. We do not share Dr Morris’ opinion that the best indications for PAIR are only those where surgery is not available. Compared with surgery, PAIR of type 1 cysts is a simple procedure, less invasive, equally effective, and can be carried out in poorly equipped hospitals.5 Patients with type 3 cysts should be treated by experienced doctors in well equipped hospitals. Currently, most clinicians consider that surgery is the treatment of choice in these latter patients. However, the experience with percutaneous drainage as initial treatment of these complicated cases is growing. In the near future we will learn more about its pros and cons. An open mind for the clinical experience of the WHO working group and of others will be helpful in making up our minds.

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REFERENCES


Body mass and gastro-oesophageal reflux symptoms

EDITOR,—In a recent article, Lagergren et al (Gut 2000;47:26–9) reported no relation between body mass and gastro-oesophageal reflux in a Swedish population and concluded that reflux symptoms occur independently of body mass index. As the authors point out, the evidence on this subject is conflicting. A large recent US cross sectional study6 reported a strong positive association between body mass index and the prevalence of reflux symptoms (table 1). One possible explanation for the difference between the two studies is the younger age distribution of the US cohort. The prevalence of overweight has increased dramatically throughout Europe and North America in recent decades.7 As a consequence, the younger US cohort is likely to have accumulated more person years of overweight by a given age and the risk of reflux symptoms may be related to both the magnitude and years of overweight exposure. The authors also concluded, in the light of their findings, that weight reduction may not be justifiable as an antireflux therapy. Even if overweight is a poor predictor of reflux symptoms, this does not necessarily imply that weight reduction will not be of benefit in providing symptom relief. A significant beneficial effect of weight loss on symptoms of gastro-oesophageal reflux in overweight patients has recently been reported in a small study involving 34 patients.8 In addition, the degree of weight loss was directly correlated with improvement in symptom score. Elsewhere, strong and independent associations have been reported between both overweight and reflux symptoms and oesophageal adenocarcinoma.9 The evidence suggests that an overweight individual with reflux symptoms is at significantly increased risk of oesophageal adenocarcinoma. Further studies clarifying the role of weight loss in the management of reflux symptoms are clearly warranted.

Table 1 Prevalence of reflux symptoms by body mass index (BMI)

<table>
<thead>
<tr>
<th>Category</th>
<th>Lagergren et al</th>
<th>Loch et al</th>
</tr>
</thead>
<tbody>
<tr>
<td>Setting</td>
<td>Sweden</td>
<td>USA</td>
</tr>
<tr>
<td>Sample size</td>
<td>820</td>
<td>1524</td>
</tr>
<tr>
<td>Sex</td>
<td>M/F</td>
<td>M/F</td>
</tr>
<tr>
<td>Mean age (y)</td>
<td>60</td>
<td>50</td>
</tr>
<tr>
<td>Measurement of BMI</td>
<td>Maximum Current</td>
<td></td>
</tr>
<tr>
<td>BMI &lt;25–24</td>
<td>16%</td>
<td>15%</td>
</tr>
<tr>
<td>BMI 25–29–24–27</td>
<td>17%</td>
<td>20%</td>
</tr>
<tr>
<td>BMI &gt;30</td>
<td>17%</td>
<td>30%</td>
</tr>
</tbody>
</table>

Figure 1 Echinococcus cyst Gharbi type 3 in the liver dome of a patient. Left: The many daughter cysts which became apparent after injection of contrast into the mother cyst prevented reliable visualisation of a possible cystobiliary fistula. Right: Six weeks following percutaneous evacuation of the daughter cysts, a cystobiliary fistula was demonstrated by cystography.
Letters, Book reviews, Correction, Notes

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Reply

EDITOR,—Like Maric and Cheng, we are also fascinated by the conflicting results in the literature on body weight and its possible association with gastro-oesophageal reflux. The critical question is whether the variation in results is explained by biologically meaningful differences (that is, if there is relevant effect modification) or if the discrepancies should be attributed to various biases, technical flaws, or simply to chance. Although it deserves to be pointed out that our data (Unpublished analyses stratified by age in our material showed the same absence of association both in relatively young (age <60 years) and old subjects. The combination of effects of age and nationality (as a proxy for early onset of overweight in the USA), as proposed by Maric and Cheng, seems to view in an even more improbable explanation. If the occurrence of reflux symptoms is dependent primarily on accumulated “exposure” to overweight, one would expect a stronger relationship in our study, which involved older people, presumably with longer exposure time. One would then also expect that the prevalence of reflux symptoms would increase with age. But Locke and colleagues1 found, if anything, a tendency towards falling rates with age. An increasing prevalence with age was found in only a minority of numerous similar epidemiologic studies. Moreover, if Maric and Cheng’s hypothesis on the importance of accumulated time with overweight is correct, our study, which assessed body mass index at different points in time in the past should have had a greater chance of ascertaining any true association compared with the study of Locke et al which dealt with only current body mass index. Non-differential misclassification of both exposure (body mass index) and outcome (reflux symptoms) due to imperfect recollection in our study may have attenuated our estimates.1 But prevalence rates for reflux symptoms well in agreement with the previous literature, and the strong association that we observed between these measures and the risk of oesophageal adenocarcinoma1 somewhat allays this concern. A further possibility that may explain the conflicting results is if the relation between body mass index and reflux propensity is non-linear with a definite trend only in the very high end of the body mass index distribution, and hence the range of body mass index values in the negative studies was insufficient to detect it. The proportion (15%) of obese subjects (body mass index >30) in our sample was considerably lower than that in the Mayo study (23%), and few subjects (n=17) had ever had a body mass index greater than 35. The data of Locke et al were not however consistent with such a threshold effect, and although statistical precision was poor, we did not see any important tendency towards a positive relation, even in the very highest end of our body mass index distribution (unpublished data). Thus the variation in results remains unexplained. Given that there is no clear geographical pattern among positive and negative studies, it appears that genetic differences between populations is an unlikely explanation.

The authors did not discuss their results in comparison with those of Roseth and colleagues2 which dealt with only current cases and those in the 125 controls in the paper by Kristinsson et al2 (median 5.2 mg/l and not 2.5 as quoted by Brydon et al). The lower median values in our 23 cancer patients compared with those in the other two studies2,3 is probably due to the less advanced cancers found in our study (six Duke’s A, five Duke’s B), which mostly included patients in the surveillance programmes. Unfortunately, nothing is told about the size and multitude of the polyps in the study by Roseth and colleagues2 but in our study the majority of patients had small adenomas and no more than 1–2, which may explain the slightly lower median values.

Brydon et al seem to have misunderstood our discussion of calprotectin levels after polypectomy. We mentioned that the levels after colonic resection for cancer in the study by Kristinsson and colleagues2 (median 10.3 mg/l, range 1–200) were similar or even higher than those after polypectomy in our study.

Table 1 Median and range calprotectin levels (mg/l) in the studies of Roseth et al, Kristinsson et al, and Kronborg et al

<table>
<thead>
<tr>
<th>Study</th>
<th>No of patients</th>
<th>Range</th>
<th>Median</th>
<th>Sensitivity (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Roseth (1993)2</td>
<td>49</td>
<td>0–12</td>
<td>2.5</td>
<td>80</td>
</tr>
<tr>
<td>Polyps</td>
<td>40</td>
<td>1.5–160</td>
<td>16</td>
<td>80</td>
</tr>
<tr>
<td>CRC</td>
<td>53</td>
<td>4–1000</td>
<td>40</td>
<td>95</td>
</tr>
<tr>
<td>Kristinsson (1998)2</td>
<td>119</td>
<td>0–12</td>
<td>2.5</td>
<td>93</td>
</tr>
<tr>
<td>Controls</td>
<td>2–950</td>
<td>52</td>
<td>93</td>
<td></td>
</tr>
<tr>
<td>Polyps</td>
<td>488</td>
<td>5–7</td>
<td>7</td>
<td>93</td>
</tr>
<tr>
<td>CRC</td>
<td>300</td>
<td>5–10</td>
<td>9</td>
<td>43</td>
</tr>
<tr>
<td>CRC, colorectal cancer.</td>
<td>23</td>
<td>12–31</td>
<td>18</td>
<td>73</td>
</tr>
</tbody>
</table>
study (median 7.07 mg/l, range 5.26–8.67), lending support to the possibility of a general intestinal mucosal defect.

The calprotectin test still has a sensitivity for colorectal neoplasia which is higher than that of ordinary guaiac tests, but the rather low specificity limits its usefulness to high risk groups.

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Sporadic HEV hepatitis in Italy

EDITOR,—We read with great interest the paper of McCrudden et al concerning recent hepatitis E (HEV) in the UK (Gut 2000;46:732–3). We agree wholeheartedly with the authors that this form of hepatitis is on the increase in industrialised countries. In Italy, the reported prevalence of anti-HEV IgG positivity ranges from 0.74% to 1.94%, although a recent study found a prevalence of 2.6% in one small town in central Italy. A value of 1.5% has been reported for the general adult population of the Republic of San Marino.

We have recently observed two cases of acute hepatitis E with no evidence of any known risk factors for HEV.

Case 1. In September 1997, a 45 year old Italian woman (not pregnant) was admitted with a one week history of fever (38°C), dark urine, and upper abdominal pain. The past medical history was unremarkable, and the patient denied recent travel abroad. There was no history of the use of drugs, alcohol, or herbal products that would justify a suspicion of toxic hepatitis.

Transaminase levels were elevated on admission and reached maximum levels approximately one week later (aspartate aminotransferase (AST) 1990 IU/L; alanine aminotransferase (ALT) 1626 IU/L). Eight days after admission total bilirubin was 1.44 µmol/l, direct bilirubin 210.33 µmol/l, alkaline phosphatase 469 IU/L, and lactate dehydrogenase 1011 IU/L. The patient was hepatitis A (HAV) IgG positive and negative for anti-HAV IgM, hepatitis C (HCV), hepatitis B (HBV), hepatitis G (HGV), cytomegalovirus (CMV), and Epstein-Barr virus (EBV) markers. Serum autoantibodies, anti-smooth muscle and antimitochondrial antibodies were absent. The patient was positive for anti-HEV IgG and negative for anti-HEV IgM.

On abdominal sonography the liver appeared normal and underwent with no intra- or extrahepatic bile duct dilatation. One month later there was a significant increase in anti-HEV IgG, and serum transaminase levels began to drop. The patient was discharged, and six weeks later jaundice had disappeared and transaminases were within normal limits.

The patient has been followed for approximately three years, during which time she has remained asymptomatic with normal transaminases, bilirubin, alkaline phosphatase, and γ-glutamyl transpeptidase levels.

Anti-HEV IgG titres have decreased but are still positive.

Case 2. A 60 year old housewife presented in our outpatient clinic with a one week history of jaundice, pale stools, and dark urine preceded by malaise, anorexia, and fever. On liver ultrasonography no bile stones or obstruction were found. She had no identifiable risk factors for liver disease, and no history of foreign travel, contact with infected individuals, or toxic exposure. She refused hospitalisation and was followed as an outpatient.

Transaminase levels were elevated (AST 1000 IU/L, ALT 2000 IU/L). Total bilirubin was 328.32 µmol/l, and alkaline phosphatase 450 IU/L. Markers for HAV, HCV, HBV, HGV, CMV, and EBV were negative; she was positive for anti-HEV IgM and negative for anti-HEV IgG. Three weeks later alcoholic and transaminases returned to near normal. Six weeks later she was anti-HEV IgG positive, and her liver function tests were normal.

As in the McCrudden series, neither of our two patients presented risk factors for HEV.

The increased prevalence of this infection among haemodialysis patients in developed countries1 and the association observed in Italy between HEV and hepatitis C clearly show that the orofaecal route is not the only means of transmission.1,11 In light of the acute sporadic HEV cases reported in non-endemic countries with high hygiene standards, it is important that clinicians consider the possibility of HEV infection in patients with clinical and biochemical features of acute non-toxic hepatitis without evidence of exposure to the major hepatitis viruses, even if there are no known risk factors for HEV.

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Editor,—We were interested to read the case report by Van Laethem et al concerning a rare re-epithelialised segment of Barrett’s oesophagus (Gut 2000;46:574–7). This raises issues in the debate over alication of Barrett’s epithelium. There has been interest in ablating the columnar epithelium to encourage squamous regrowth which may reduce the risk of progression to adenocarcinoma. However, there have been numerous reports of buried glands under the regenerated mucosa.1,15

While we accept that columnar glands may persist under the squamous epithelium and that this may represent a continuing carcinomata risk, this is difficult to quantify. Indeed, this is the first report of such a malignant change. It may be that as any buried glands are no longer exposed to potential carcinogens in the form of acid or bile reflux, the risk is reduced.

Although the ultimate aim of treatment is to eliminate the risk of potential malignant change, any means of reducing such risk, for example by diminishing the volume of metaplastic tissue, would be worthwhile. This whole issue needs further evaluation by appropriately designed clinical trials.1

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Adenocarcinoma arising in columnar lined oesophagus following treatment with argon plasma coagulation

EDITOR,—Following the recent report by Van Laethem et al (Gut 2000;46:574–7) of adenocarcinoma developing in a patient whose columnar lined oesophagus had been treated by argon plasma coagulation, we wish to highlight a second case.

A 67 year old man presented with epigastric discomfort but no “alarm” symptoms of dysphagia or weight loss. Endoscopy revealed a 5 cm length of columnar lined oesophagus with no evidence of ulceration or stricture. Histology showed intestinal metaplasia with low grade dysplasia. He consented to enter a study of argon plasma coagulation treatment in Barrett’s oesophagus.

One half of the affected oesophagus was treated with argon plasma coagulation (Erbe APC 300, Erbe Elektromedizin GmbH, Germany). He was commenced on omeprazole 40 mg. Repeat endoscopy at two months showed macroscopic regrowth of the squamous epithelium in the area treated by argon plasma coagulation. This was confirmed histologically and the previously noted dysplasia had disappeared. He did not attend for repeat endoscopy at four months but was admitted because of significant weight loss and dysphagia. Endoscopy showed a stricture at the gastro-oesophageal junction and biopsies confirmed poorly differentiated adenocarcinoma. CT scanning of the thorax and abdomen showed thickening of the oesophageal wall but no obvious metastases. However, at laparotomy, he was found to have an adenocarcinoma arising in columnar lined oesophagus.
unresectable tumour with extensive local spread and distant metastases to the liver.

This case illustrates two key points. Firstly, carcinoma developed in spite of argon plasma coagulation treatment. Only half of the affected mucosa was treated in this study to allow the remaining half to serve as an internal control and so it is impossible to state whether this oesophageal carcinoma arose in the argon plasma coagulation treated or untreated segment. The central issue is whether squamous re-epithelialisation bolishes the malignant potential of the gastro-oesophageal junction. Destruction of columnar epithelium by argon plasma coagulation followed by restitution of squamous epithelium may reverse dysplastic changes but could simply hide them.

Secondly, and perhaps more importantly, this carcinoma went undetected in spite of rigorous endoscopic follow up and a well defined biopsy protocol, raising further doubts over the effectiveness of conventional endoscopic surveillance of columnar lined oesophagus. The surveillance process is subject to several potential sampling errors. The dysplastic process may be patchy and changes may be missed at biopsy. The histological interpretation of dysplasia is subjective and observer dependent. Finally, carcinoma may arise from the submucosal layers of the oesophagus with very little mucosal abnormality, and beyond the reach of conventional endoscopic biopsy forceps. Such carcinomas are likely to remain undetected until a very late stage.

No evidence of the phenomenon of “buried glands” was seen following argon plasma coagulation treatment in this case. Other authors have reported this appearance following thermal ablative treatment of columnar lined oesophagus. These islands of persistent metaplastic tissue may retain the potential for malignant transformation. Their significance is as yet unclear but, in this case at least, they cannot be implicated in the progression to carcinoma.

All patients with columnar lined oesophagus who have participated in clinical studies of argon plasma coagulation will require close follow up for many years to ensure that potentially malignant tissue has truly been ablated and not merely covered by a “white wash” of squamous epithelium.

Reply

EDITOR,—Dr Shand and colleagues clearly underline, as we did (Gut 2000;46:574–7), the major concerns about the eradication of Barrett’s mucosa by thermo-coagulation. Their case differs from ours in the following ways: our patient did not show any dysplasia at baseline diagnosis, has completed full eradication of the Barrett’s segment, and showed recurrence of neoplasia after a period of 18 months, clearly beneath the squamous; this last finding supports the fact that emergence of neoplastic glands was probably newly developed. The present case is interesting in two ways: another concern with this type of management; as no buried glands were evidenced under the new squamous layer and the interval between endotherapy and occurrence of unresectable tumour was very short (approximately four months), this case clearly illustrates the need for a complete and optimal staging and mapping of the target areas before starting the destruction of Barrett’s mucosa disclosing dysplasia.

As stated and discussed by the authors, the initial dysplastic process was probably patchy and changes may be missed or under staged at biopsy; in this situation, argon plasma coagulation treatment only hides the dysplastic areas.

Furthermore, submucosal origin of the carcinoma ideally should be excluded by performing endoscopic ultrasonography and profound biopsies with large forceps. Reporting these cases clearly shows that:

(a) Barrett’s mucosa destruction remains experimental and surveillance has to be strictly maintained.

(b) Selection of patients is paramount and should include accurate staging and mapping of the target areas before endotherapy.

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Gastric cancer in patients with benign dyspepsia

EDITOR,—There is an ongoing debate regarding the value of endoscopy in younger patients presenting with dyspepsia. One important consideration is the issue of detecting an underlying cancer which might be cured by early treatment. The large retrospective study by Breslin and colleagues in the January issue of Gut (Gut 2000;46:93–97) indicates that underlying cancer will be diagnosed in about 1 in 1000 patients presenting with uncomplicated dyspepsia under 45 years of age. However, the calculated 95% confidence intervals for this are wide (1 in 2963 to 1 in 300).

An important question in considering the significance of this finding is whether the prevalence of cancer in these patients with benign dyspepsia is any different from that in the general population. In our own country, Scotland, the chance of a patient presenting with gastro-oesophageal cancer before the age of 50 is 1 in 909 (ISD Scotland Cancer Surveillance Group Data Request and Analysis Service) and half of those have presented with the cancer within the age band 45–49. Most of these patients will have had the tumour present in their stomach for a considerable time prior to clinical presentation, which would have been detected by screening endoscopy five years earlier.

Even allowing for the fact that population based rates of gastro-oesophageal cancer are higher in Scotland than Alberta,1 this suggests that the prevalence of underlying cancer in patients presenting with uncomplicated dyspepsia may not be different from that in the general population. Consequently, offering endoscopy to patients with simple uncomplicated dyspepsia to detect cancer may merely represent screening of the general population.

There has been a general assumption that a tumour growing in the stomach will produce dyspeptic symptoms. However, there is no evidence for this. Tumours developing in the colon or other parts of the gastrointestinal tract rarely, if ever, cause symptoms until they produce complications such as bleeding or obstruction. A very small proportion of patients presenting with uncomplicated dyspepsia will have underlying cancers but this finding may be unrelated to their symptoms. Unless uncomplicated dyspepsia is confirmed to be a symptom of underlying malignancy, then one would be as well to recommend offering endoscopy to patients presenting with a


Surgeons, hepatologists, and oncologists involved in the management of malignant tumours of the liver now have a variety of recent books available for reference. Some of these texts are primarily concerned with surgical management, with subsidiary chapters on diagnosis, pathology, and other modes of treatment. Others are written from the point of view of the physician or oncologist. This new book has been edited with a change of emphasis in that it attempts to examine and compare critically all of the current modalities of treatment as well as some of those which may be successful in the future. I was pleased with the emphasis on maintaining the quality of life in patients with incurable disease rather than trying everything to gain a little more survival time, a very important principle for physicians and surgeons dealing with this group of malignancies.

In the preface, Professor Clavien emphasises that the optimal management is often difficult and often complicated group of tumours depends on a multidisciplinary team approach and he has edited the text to integrate the investigatory, surgical, and oncological aspects of treatment. Firm editorial control allows each of the chapters to be read as a complete essay but I found that the book also read well as a sequential text with minimal duplication of material in each of the sections. The up to date nature of the book is well illustrated by an example from the section on gene therapy which gives details of a phase one study, from November 1998, of recombinant p53 adenovirus gene therapy.

The book is based on the experience at the University of Giessen, Germany: Springer-Verlag, 1999. ISBN 3-540-65357-0.

This is a meetings book (“songs from the cytotype”) containing 24 contributions in just over 260 pages on the state of the art in pancreatic disease, as of September 1998. It is a virtual textbook with eight chapters on acute pancreatitis, eight on chronic pancreatitis, through cystic fibrosis, for four on cancer and on epidemiology (“lessons from”). The chapter titles are intriguing, focusing on biological mechanisms and current management attitudes. Genetics features strongly with a strong emphasis on clinical care and directions for research. The flavour is strongly European: for pancreatic inflammatory disease, both acute and chronic, 11 of the 16 contributions are from Germany (the meeting was held in Munich) giving a welcome access to a literature which is not often cited in English language journals. Most of the chapters are approximately 10 pages long, fully referenced, and up to date. As is inevitable, there is a fair amount of overlap and repetition and the quality is certainly uneven, ranging from detailed molecular pathology suitable for research workers (for example, the chapters on cystic fibrosis, mechanisms of fibrosis in chronic pancreatitis, and growth factors in carcinoma) to what would be more suitable for a lecture to undergraduates (exocrine pancreatic secretion).

However, for those interested in pancreatic disease, this little book (it is a pocket size paperback) offers a useful work of reference. The introductory chapters on the genetics of cellular injury, intracellular, extracellular, and immune mechanisms in acute pancreatitis are particularly well done, although the subsequent contributions on varieties of clinical management contain nothing new. The section on chronic pancreatitis contains some overlap between chapters but the contribution on mechanisms of fibrosis and potential therapy using inhibitors is fascinating, if still a distant dream. The chapters on cystic fibrosis are detailed and very interesting with excellent reviews on the state of gene therapy today and problems with enzyme therapy. The chapter on what we now call idiopathic chronic pancreatitis is certainly worth a careful read. The section on pancreatic cancer is, like the disease, disappointing, representing the essentially bleak situation of specialists searching around for mechanisms and treatment modalities with little success.

In all, as meetings books go, this one should be worth a place in the departmental library if you can afford it. There are lots of good references, figures, and diagrams, and it covers the ground of pancreatic disease very thoroughly.

M SARNER

Picture the scene. An international conference on gastroenterology, delegates flown in from the four corners of the earth, a nice hotel near the sea and golf courses, and one of those keypad voting systems. Dyspepsia? Easy! Dish out a PPI and lets get on to the really interesting stuff like fecal transferases and K-vu gene point mutations. But wait! The audience has been asked what it would do with a 43 year old man with an 18 month history of vague upper abdominal pain, a stressful life, and a variable response to OTCH blocker. The voting screen reveals an astonishing divergence of opinion about management. A Helicobacter pylori test followed by endoscopy if positive. Plenty of PPI and symptomatic review in a couple of months? Urgent or once in a lifetime endoscopy? The Austrian delegation are muttering about psychotherapy and a shady group of surgeons in the corner are all for an emergency laparotomy. This is why people keep writing books about dyspepsia and why this book by Gerald Holtmann and Nick Talley is particularly welcome. It succeeds in combing Germanic thoroughness with a degree of didacticism wrapped in a clarity of thought and a healthy scepticism about what passes for the "literature". The book can clearly be sponsored by Byk Gulden whose top product is pantoprazole because one of their staff has written the preface. However, the authors are scrupulous and objective about their references to individual drugs, and there is nowhere a hint of commercial bias. The arrangement of the material is traditional but contains some little gems. The section on the definition and clinical presentation of abdominal syndromes includes very helpful information about subgroups of dyspepsia and ways of distinguishing between functional dyspepsia and irritable bowel syndrome. The epidemiology is, as you would expect, thorough, and the chapter on the pathophysiology of functional dyspepsia, with almost 100 references, is a mine of information with implications for research as well as clinical practice. There is a good section on psychosomatic factors, once again well referenced and reasonably up to date. The chapters on diagnosis and management also cover most of the recent publications but although the cisapride problem is mentioned it is unhappily underplayed. There is a sensible summary of the Genval workshop with two clear flowcharts and some specific recommendations on treatment strategies and dosages, which I found particularly helpful. Interestingly, in the "Special management problems" chapter, a different author gives a completely different viewpoint on the Helicobacter pylori/pump inhibitor debate, which adds a bit of spice. There is a sensible summary of nonspecific chest pain and clear guidelines on drug treatment of reflux disease in pregnancy. Within the confines of a very short chapter, Barrett's is sensitively handled, as well as other complications of reflux disease, and in the final chapter the indications for surgery are discussed. There follows a description of surgical techniques, including laparoscopic fundoplication, and a detailed analysis of short and long term complications. Overall, this book packs a fair amount into its diminutive size and is sensibly priced. It deserves to be widely read.

A IRELAND


This book addresses 10 topics in which there has been significant development over the past decade. The subjects discussed are diverse, ranging from the combined surgical treatment for advanced pelvic malignancy to incontinence surgery, and from imaging of the anal canal and rectum to the management of anal fissure.

R JONES


I started reading this book not entirely sure whose bookshelf it was designed to be placed on. It is written largely by mainland European gastroenterologists, with one American contribution, and most contributors will be unfamiliar to British readers. The first chapter gets off to an inauspicious start, being in a very inauspicious style and giving a rather simplistic overview. The use of various reflux terms is not clear and there seems a surprising statement about the lack of utility of 24 hour pH studies in endoscopy negative reflux patients. In addition, no mention is made of the videoeastoscopy eatheter in the manometry section, which is an oversight in view of the fact they are discussing LOS relaxation. Thankfully the content and presentation improve dramatically after chapter 1, giving a very useful and informative book on the subject which can be appreciated at all levels of medical training. Specialist registrars will probably find it most helpful as consultants may wish for something a bit more "meaty".

There is a clear concise chapter on short term management, with useful supplementary information and good references, but I detect a slight commercial bias with the PPI terminology. It would be a tedious exercise to include every recipe as this is clearly a sponsored publication. Long term management is up to date, with even a discussion on the recent conflicting views on Helicobacter pylori and proton pump inhibitors, coming down, rightly in my view, on the side of non-eradication. There is a useful summary of the Genval workshop with two clear flowcharts and some specific recommendations on treatment strategies and dosages, which I found particularly helpful. Interestingly, in the "Special management problems" chapter, a different author gives a completely different viewpoint on the Helicobacter pylori inhibitors debate, which adds a bit of spice. There is a sensible summary of functional chest pain and clear guidelines on drug treatment of reflux disease in pregnancy. Within the confines of a very short chapter, Barrett’s is sensitively handled, as well as other complications of reflux disease, and in the final chapter the indications for surgery are discussed. There follows a description of surgical techniques, including laparoscopic fundoplication, and a detailed analysis of short and long term complications. Overall, this book packs a fair amount into its diminutive size and is sensibly priced. It deserves to be widely read.

A LEATHER

CORRECTIONS

Errors occurred in the UEGW abstracts supplement Gut. 2000 May;47(Suppl. II). For abstracts A136 and A160, the complete author list for both abstracts is M M Diculescu, E M Ionescu, M Ciocirilan, M Prunescu, R Iacob, S Iacob, C Aparetchiocia, A Oprea. For abstract A271, the complete author list is H J Tan and E P Nasmyth.
The authors of a case report published in March (Gut 2001;48:425–9) would like to add C McKenzie as the second last author. Her affiliation is the University of Southamp-ton. The authors would also like to acknowledge that the work was supported by the Bio-technology and Biological Sciences Research council (BBSRC).

NOTES

GASTRO 2001
The Annual Scientific Meeting of the Malaysian Society of Gastroenterology and Hepatology (MSGH) will be held on 5–8 April 2001 in Sabah, Borneo. Further information: GASTRO 2001, 19, Jalan Folly Barat, 50480 Kuala Lumpur, Malaysia. Tel: +603 2530100/2530200; fax: +603 2530900; email: acadmed@po.jaring.my; website: gastro2001.homestead.com/files/index.htm

11th International Workshop of Digestive Endoscopy, Ultrasonography, and Radiology
This workshop will be held on 17–18 May 2001 in Marseille, France. Further information: Nathalie Fontant, Atelier Phenix, 41 rue Docteur Morucci, 13006 Marseille, France. Tel: +33 (0)4 91 37 50 83; fax: +33 (0)4 91 57 15 28; email: nfontant@aphenix.com

EPGS Endosonography Live in Amsterdam
This European Postgraduate Gastro-Surgical School congress will take place on 31 May and 1 June 2001 in Amsterdam, the Netherlands. Further information: Mrs Helma Stockmann/Mrs Joy Goedkoop, European Postgraduate Gastro-Surgical School, Meibergdreef 9, 1105 AZ Amsterdam, The Netherlands. Tel: +31 20 566 3926; fax: +31 20 566 6569; email: W.J.Stockmann@amc.uva.nl; website: www.epgs.nl

33rd European Pancreatic Club
The meeting will take place on 13–16 June 2001 in Toulouse, France. A training course will be organised on 13 June on “Genomics and post genomics: developments in biomedical sciences”. Further information: Dr Nicole Vaysse, Inserm U531, CHU Rangueil, 31403 Toulouse, France. Tel: +33 (0)5 61 32 24 02; fax: +33 (0)5 61 32 24 03; email: nicole.vaysse@rangueil.inserm.fr; website: www.e-p-c.org.

Gastroenterology and Endotherapy: XIXth European Workshop
This course, to introduce the experienced gastroenterologist to the growing field of therapeutic endoscopy, will be held on 18–20 June 2001 in Brussels, Belgium. Further information: Mrs Nancy Beauprez, Gastroenterology Department, Erasme Hospital, Route de Lennik 808, B-1070 Brussels. Tel: +32 02 555 49 00; fax: +32 02 555 49 01; email: beauprez@ulb.ac.be

Falk Symposium
The symposium Inflammatory Bowel Disease: A Clinical Case Approach to Pathophysiology, Diagnosis, and Treatment will be held in Bologna, Italy on 22–23 June 2001. Further information: Prof Dr M Campieri/Dr P Gionchetti, Policlinico S. Orsola - Malpighi, Dipartimento di Medicina Interna e Gastroenterologia, Via Massarenti 9, I-40138 Bologna, Italy. Tel: +39 (051) 6364 116 or 6364 122; fax: +39 (051) 392538; email: campieri@med.unibo.it or paolo@med.unibo.it

Summer Abdominal Imaging Conference
A five day course designed for the practising radiologist with a primary interest in abdominal imaging, emphasising the most recent advances in helical CT, MRI, US, and gastrointestinal imaging. It will be held on 23–27 July 2001 in Banff Springs, Canadian Rockies. Twenty-five category I credit hours. Further information: Janice Ford Benner, University of Pennsylvania Medical Center (Radiology), 3400 Spruce Street, 1 Silverstein Building, Philadelphia, PA 19104, USA. Tel: +1 215 662 6904; fax: +1 215 349 5925.

Torino-Toronto First Joined Workshop on Therapeutic Endoscopy
This workshop will be held on 13–15 September 2001 in Turin, Italy. Further information: Anna Botto, MAF Servizi, Congress Division, Via GB Vico, 7, 10128 Turin, Italy. Tel: +39 011 505 900; fax: +39 011 505 976; email: abotto@mafservizi.it

ICGH-2: The Second Iranian Congress of Gastroenterology and Hepatology
The main Iranian meeting of gastroenterologists and researchers in this field will be held on 27 October to 1 November 2001 in Tehran, Iran. Further information: Dr Shahin Merat, Digestive Diseases Research Center, Shariati Hospital, N. Kargar Street, Tehran 14114, Iran. Tel: +98 911 717 3966; fax: +98 21 225 3635; email: merat@ams.ac.ir; website: www.ams.ac.ir/icgh. Deadline for submission of abstracts is 31 May 2001.