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 scolicidal 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. Therefore, in PAIR, as a standard procedure, cystography is performed before scolicidals are used. 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 from reaching and demasking a possible fistula (fig 1; right). Therefore, we do not advocate the use of scolicidals in type 3 cysts.

Can patients with type 3 cysts be treated safely with percutaneous drainage? Faced with serious complications such as biliary obstruction, cholangitis, rupture of cyst content into the biliary tree, sepsis due to cyst infection, and obstruction of portal and hepatic veins, we modified the PAIR procedure in these patients. After puncture and aspiration, the cyst content is evacuated via a 8–18 F catheter by frequent injection and reaspiration of small amounts of isotonic saline (20–40 ml) using a 60 ml syringe. The daughter cysts readily rupture when aspirated into the catheter. Puncture of each single daughter cyst is not necessary. We avoid injection of alcohol into the mother cyst because of the high occurrence of a cystobiliary fistula. Six of the 10 patients with type 3 cysts that we treated in this way had a cystobiliary fistula. In three the fistula was present before percutaneous aspiration was initiated. In the other three patients the fistula became apparent only after the procedure was completed (fig 1; right). In 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. 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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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.
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Reply

EDITOR,—We read with interest the paper by Locke et al (Gut 1999;47:26–9) primarily pertinent to an elderly Swedish population (mean age 66 years) in which we believe that effect modification by age is a less likely explanation for the discrepancy between our results and those of Locke and colleagues. 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 to find a stronger relation 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 colleagues found, if anything, a tendency towards falling rates with age. An increasing prevalence with age was found in only a minority of numerous similar epidemiological studies. Moreover, if Maric and Cheng’s hypothesis on the importance of accumulated time with overweight is correct, their 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. 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 adenocarcinoma—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. While uncontrolled non-randomised intervention studies, like the one cited by Maric and Cheng, contribute relatively little to our understanding of the importance of body weight (patients who manage to lose weight may differ from those who fail to do so due to various reasons), more in depth clinical and epidemiological studies are needed to resolve the apparent enigma.

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Reply

EDITOR,—The values in our 488 controls (median 7 mg/l, range 5–7) did not differ from those in the 125 controls in the paper by Kristinsson (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 two other studies 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 colleagues 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 calciprotein levels after polypectomy. We mentioned that the levels after colonic resection for cancer in the study by Kristinsson and colleagues median 10.3 mg/l, range 1–200) were similar or even higher than those after polypectomy in our Table 1 Median and range calciprotein 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>
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<tr>
<td>Roseth (1993)³</td>
<td>Controls 49</td>
<td>0–12</td>
<td>2.5</td>
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<tr>
<td>Polyps 40</td>
<td>1.5–160</td>
<td>16</td>
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<tr>
<td>CRC 53</td>
<td>4–1000</td>
<td>40</td>
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</tr>
<tr>
<td>Kristinsson (1998)³</td>
<td>Controls 119</td>
<td>0–12</td>
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<td>Polyps 119</td>
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<td>CRC 23</td>
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<td>Polyps 300</td>
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<tr>
<td>CRC 23</td>
<td>12–31</td>
<td>18</td>
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CRC, colorectal cancer.

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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 acute 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 the 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.1 A value of 1.5% has been reported for the general adult population of the Republic of San Marino.2 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) 1620 IU/L). Eight days after admission total bilirubin was 74.44 µmol/l, direct bilirubin 21.33 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 cirrhotic, and was resectioned with no intra- or extrahepatic bile duct dilation. 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 transaminase, 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 ultrasoundography no bile stones or obstruction were found. She had no iden-
tifiable 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 out-
patient.

Transaminase levels were elevated (AST 1000 IU/L, ALT 2000 IU/L). Total bilirubin was 328.32 µmol/l, direct bilirubin 241.11 µmol/l, and alkaline phosphatase 450 IU/L. Markers for HAV, HBV, HCV, HGV, CMV, and EBV were negative; she was positive for anti-HEV IgM and negative for anti-HEV IgG. Three weeks later anti-HEV IgG had disappeared, 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 develop-
ed countries1 and the association observed in Italy between HEV and hepatitis C clearly show that this is not the only means of transmission.1 In light of the acute sporadic HEV cases reported in non-endemic countries with high hygienic standards, it is important that clinicians consider the possi-

bility of HEV infection in patients with clini-
cal 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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Re-epithelialisation of Barrett’s oesophagus

EDITOR,—We were interested to read the case report by Van Laethem et al concerning a carcinoid tumour arising in a re-epithelialised segment of Barrett’s oesophagus (Gut 2000;46:574–7). This raises issues in the debate over ablation 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.1,2

While we accept that columnar glands may persist under the squamous epithelium and that this may represent a continuing carcino-

ma 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 carci-

nogens 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.3

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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 adenoca-

rcinoma 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 epiga-

stric 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 oesopa-

gal wall but no obvious metastases. How-

er, at laparotomy, he was found to have an

1 Byrne JP, Armstrong GR, Artwood SEA. Restora-

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Letters, Book reviews, Correction, Notes
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 other 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 neutralises 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. Whether squamous and 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 underlined, as we did (Gut 2000;46:574–7), the major concerns about the eradication of Barrett’s mucosa by thermoablation. Their case differs from ours in the following two ways: our patient did not show any dysplasia at baseline diagnosis, has completed full eradication of the Barrett’s segment, and showed recurrence of neoplastic glands 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 not only in itself but in another concern with this type of management; as no buried glands were evidenced under the new squamous layer and the interval between endotheropy 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 endo.

Outcome of lamivudine resistant hepatitis B virus infection in liver transplant recipients in Singapore

Editor,—We read with interest the article by Mutimer and colleagues (Gut 2000;46:107–113). The Birmingham group described the clinical course of four liver transplant patients who developed graft infection with lamivudine resistant virus. Lamivudine resistant hepatitis B virus infection after a mean duration of nine months (range 8–11) after the transplant. Liver function abnormalities occurred at a mean duration of six months (range 3–12) after the emergence of lamivudine resistant virus and three of the four patients died 5–20 months later. The authors concluded that the lamivudine resistant phenotype can cause severe graft damage.

In our liver transplant unit, 12 patients with chronic hepatitis B (four with hepatocellular carcinoma) underwent liver transplantation over a five year period. All were given lamivudine before and after transplant. Lamivudine resistant hepatitis B virus infection developed in six of the nine survivors at a mean duration of 60 weeks (range 1–127) after liver transplant. Apart from weaning off immunosuppression aggressively, no further antiviral treatment was added. All six had normal liver function at their last follow up (mean 28, range 0–123 weeks after emergence of lamivudine resistant virus).

Contrary to what the Birmingham group experienced, all of our patients with lamivudine resistant virus were well, with no evidence of graft dysfunction. Long term outcome of such patients remained stable and it may be premature to conclude that the lamivudine resistant phenotype causes severe graft damage.

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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 likelihood 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
BOOK REVIEWS


Surgeons, hepatologists, and oncologists involved in the management of malignant tumours of the liver now have a wealth 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 of 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 a variety of special topics, such as the management of tumours in children, in the elderly, and in pregnancy.

This is a timely book in view of the rapid increase in the number of investigations and treatments now available for the management of liver tumours. It provides an excellent introduction for surgical trainees but at the same time includes enough thoughtful discussion, up to date information, and practical advice to be of use to any general gastroenterologist or liver specialist.

E R HOWARD


Recertification or subspeciality exit examinations may trigger a proliferation of self assessment texts, although candidates for part 2 MRCP are currently this book’s main market. In the whole it serves its purpose well and complements the similar sized MCQs in Gastroenterology (Bateson and Stephen; 1996; Petroc Press).

The book presents almost 200 illustrated case histories, with questions and well informed answers from 28 gastroenterologists, half from the UK and half from the USA. This is good transatlantic collaboration. Cases cover the whole gamut of gastrointestinal and luminal gastrointestinal disease (including biliary and pancreatic disease), from the common and uncomplicated to the obscure. They are interesting and informative. Some questions are insufficiently concise for MRCP although it is only fair to say that the authors do not set out to follow the format of this examination. Other questions ask the reader to match statements and data, which are good tests of knowledge, especially that of basic gastrointestinal physiology. Indeed, I would like to see more physiological questions at the expense of some “picture recognition” cases. This is because the photographic reproduction of some of the 350 or so images is variable.

Some endoscopic and radiographic images have not reproduced well or are too small to be interpretable. The variety of cases and illustrated answers are, however, stimulating.

Doctors taking MRCP may want to buy a copy although many topics are more appropriate for specialist trainees. Consultant gastroenterologists will find it an entertaining and instructive exercise to dip into the book but I suspect that this will be from the library shelf where it will be one of a series of self assessment titles.

S P L TRAVIS


This is a meetings book (“songs from the distant dream.”) 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, three on cystic fibrosis, four on cancer, and one on epidemiology (“lessons from”). The chapter titles are intriguing, focusing on biological mechanisms and current management attitudes. Genetics features strongly, with an 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 signalling, 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 a good synopsis 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 reviews on the status 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 treatments with minimal real hope.

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.

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 real interesting stuff like fucosyltransferases and Ki- ras 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 OTC blocks. 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 Australian 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 combining Germanic thoroughness with a degree of didacticism with clarity of thought and a healthy scepticism about what passes for the “literature”.

The book has clearly been sponsored by Byk Gulden and the whole book 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 reputation of the colorectal unit at Singleton Hospital (where the editors are based) will certainly be further enhanced by this well collated and useful book.

A LEATHER


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.

A IRELAND


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 stilted 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 oesophageal 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 recommendations, which is unfortunate 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-eradicating. 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/proton pump inhibitor debate, which adds a bit of spice. There is a sensible summary of non-acidic 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

Corrections

Errors occurred in the UEGW abstracts supplement Gut 2000;47(suppl III). For abstracts A136 and A160, the complete author list for both abstracts is M M Diculescu, E M Ionescu, M Ciocirlan, M Prunescu, R Iacob, S Iacob, C Apetrechioaei, A Oprea. For abstract A271, the complete author list is H J Tan and D 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 Southhampton. The authors would also like to acknowledge that the work was supported by the Biotechnology 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: 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: WJ.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

**Redefining Priorities in Gastroenterology**
This congress will be held on 11–14 April 2001 in Monte Carlo, Italy. It will be chaired by Professor Massimo Crespi (Rome, Italy) and Professor Eammon Quigley (Cork, Ireland). Further information: Maddalena Massaro, Project Leader, AISC-AIM Group, Via A Ristori 38, 00187 Rome, Italy. Tel: +39 06 809681; fax: +39 06 80968229; email: gastro2001@aisc.it

**3rd European Federation of Autonomic Societies (EFAS)**
The third European Federation of Autonomic Societies (EFAS) meeting in conjunction with the annual meeting of the sections “Autonomic nervous system” of the German Neurological Society, “Diabetes and Nervous System” of the German Neurological Society, and “Autonomic Nervous System” at the University of Erlangen-Nuremberg, Germany, will be held in Erlangen, Germany on 26–28 April 2001. Further information: Professor Dr M J Hilz, Department of Neurology, University or Erlangen-Nuremberg, Schwabachanlage 6, D-91054 Erlangen, Germany. Tel: +49 0131 8534444; fax: +49 9131 8534328; website: www.neurologie.med.uni-erlangen.de/oefentliche_Veranstaltungen.htm

**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.

**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 21 223 3633; email: merat@ams.ac.ir; website: www.ams.ac.ir/icgh. Deadline for submission of abstracts is 31 May 2001.