Percutaneous drainage of echinococcal cysts
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Percutaneous drainage of echinococcal cysts

H G SCHIPPER, P A KAGER and J S LAMÉRIS

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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 scolicidals into hydatic 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 bile duct 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 an 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.

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 study1 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.2 As a consequence, the younger US cohort is likely to have accumulated more person years of overweight by any 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.3 In addition, the degree of weight loss was directly correlated with improvement in symptom score. Overall, strong and independent associations have been reported between both overweight and reflux symptoms and oesophageal adenocarcinoma.4 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 may be warranted.

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

<table>
<thead>
<tr>
<th>Category</th>
<th>Lagergren et al</th>
<th>Loche 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>66</td>
<td>50</td>
</tr>
<tr>
<td>Measurement of BMI</td>
<td>Maximum Current</td>
<td></td>
</tr>
<tr>
<td>BMI &lt;25 &lt;24</td>
<td>16%</td>
<td>15%</td>
</tr>
<tr>
<td>BMI 25–24 &lt;27</td>
<td>17%</td>
<td>17%</td>
</tr>
<tr>
<td>BMI 27–30</td>
<td>No data</td>
<td>20%</td>
</tr>
<tr>
<td>BMI &gt;30</td>
<td>17%</td>
<td>30%</td>
</tr>
</tbody>
</table>

Risk of oesophageal adenocarcinoma1 some-
what allays this concern. A further possibility that
may explain the conflicting results is if the rela-
tion between body mass index and reflux propor-
tion is non-linear with a definite trend only in the
very high end of the body mass index distribu-
tion, and hence the range of body mass index values in the
negative studies was insufficient to detect it. The propor-
tion (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
Loke et al were not however consistent with such
a threshold effect, and although statisti-
cal precision was poor, we did not see any
important tendency towards a positive rela-
tion, even in the very highest end of our body
mass index distribution (unpublished data).

Thus the variation in results remains unex-
gained. Given that there is no clear geo-
graphical pattern among positive and nega-
tive studies, it appears that genetic differ-
etions between populations is an unlikely explana-
tion. While uncontrolled non-randomisation
interventions 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), more in depth clinical and epidemiological
studies are needed to resolve the apparent
equation.

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Facial calprotectin levels and colorectal
neoplasia

EDITOR,—We read with interest the paper by
Kronborg and colleagues (Gut 2000;46:795–
800)—a large multicentre study measuring
facial calprotectin levels in high risk popula-
tions for colorectal neoplasia.

The authors did not discuss their results in
comparison with those of Rosenst and col-
leagues1 or Kristinsson and colleagues1 who
did the ground breaking work in this area and
where calprotectin levels were shown to be far
higher in patients with colonic polyps and
cancer compared with normal controls (table 1).

Table 1 Median and range calprotectin levels (mg/l) in the studies of Rosenst 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>Rosenst (1993)</td>
<td>49</td>
<td>0–12</td>
<td>2.5</td>
<td>94</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)</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>76</td>
</tr>
<tr>
<td>CRC</td>
<td>300</td>
<td>5–10</td>
<td>9</td>
<td>43</td>
</tr>
<tr>
<td>Kronborg (2000)</td>
<td>23</td>
<td>12–31</td>
<td>18</td>
<td>73</td>
</tr>
</tbody>
</table>

CRC, colorectal cancer.
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 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.1 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 44 µmol/l, direct bilirubin 210.33 µmol/l, 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 the transaminase titres returned to normal. Approximately one week later (aspartate aminotransferase (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, HCV, HBV, HGV, CMV, and EBV were negative; she was positive for anti-HEV IgM and negative for anti-HEV IgG. Three weeks later the transaminase titres returned to 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 countries2 and the association observed in Italy between HEV and hepatitis C clearly show that this form of hepatitis is not the only means of transmission.3 In light of the recent spadaneous HEV cases reported in non-endemic countries with high hygienic 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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Re-epithelialisation of Barrett's oesophagus

EDITORS,—We were interested to read the case report by Van Laethem et al concerning a carcinomatous progression of a re-epithelialised segment of Barrett's oesophagus. (Gut 2000;46:574–77). 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.1,2,3

While we accept that columnar glands may persist under the squamous epithelium and that this may represent a continuing carcinoma 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 ensuring 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
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 abolishes 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 underlined, 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 neoplasms 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, but it is 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:

1. Barrett’s mucosa destruction remains experimental and surveillance has to be strictly maintained.

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

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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;46:107–13). The Birmingham group described the clinical course of four liver transplant patients who developed graft infection with lamivudine resistant virus. Lamivudine resistant hepatitis B developed 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 (4 with hepatocellular carcinoma) underwent liver transplantation over a five year period. All were given lamivudine before and after transplant. Lamivudine resistant hepatitis B 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. 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 remains controversial 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, 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
sprained ankle in order to pick up underlying gastro-oesophageal cancer.

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BOOK REVIEWS


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 viewpoint 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 this 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 UK, on this side of the Atlantic.

The book includes four further main sections. The second section concerns systemic and local therapies such as hepatic artery ligation. This section is well illustrated, as is the remainder of the book, and is followed in the next section by a series of chapters on methods of tumour ablation which include standard liver resection techniques, transplantation, cryoslation, and ethanol injection. Although this is not a book primarily concerned with the details of surgical technique, the important surgical points are described clearly.

The fourth section is an exciting glimpse into the future of gene therapy, immunotherapy, and angiogenesis, and is completed with a clearly written essay on apoptosis (programmed cell death) and its significance in the possible development of new strategies in cancer therapy. The book concludes 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 to the whole subject for 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 main market in the UK. On the whole it serves its purpose well and complements the similar sized MCQs in Gastroenterology (Batson 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 a wide range of gastroenterology (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 have liked 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 West”) 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 signaling, 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 contributions 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 an overview of the status of gene therapy today and problems with enzyme therapy. The chapter on what we now call idiopathic chronic pancreatitis contains some useful reading. The section on pancreatic cancer is, like the disease, disappointing, representing the essentially bleak situation of specialists searching around for mechanisms and treatments in the last few years. 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

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Letters, Book reviews, Correction, Notes

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Clinician's Manual on Managing Dyspepsia

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 let's get on to the hotel near the sea and golf courses, and one of those from the four corners of the earth, a nice ench on gastroenterology, delegates flown in to vote. The first chapter gets you off to an auspicious start, being in a very readable 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 Barree catheter 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 "mature".

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-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 pylorippi cancer debate, which adds a bit of spice. There is a sensible summary of non-cardiac 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, 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 pylorippi cancer debate, which adds a bit of spice. There is a sensible summary of non-cardiac 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, and proton pump inhibitors, coming down, rightly in my view, on the side of non-eradication.

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A IRELAND

Recent Advances in Coloproctology

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 LEATHER

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 Aprentioaei, A Oprisu. For abstract A271, the complete author list is H J Tan and B Nasmuth.
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 Southampton. 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 Folli Barat, 50480 Kuala Lumpur, Malaysia. Tel: +603 2530100/2530200; fax: +603 2530900; email: gastro2001@homestead.com/files/index.htm

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, 10129 Cork, Ireland. Tel: +353 061 605961; email: madmassaro@aimgroup.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 Hitz, Department of Neurology, University or Erlangen-Nuremberg, Schwabachanlage 6, D-91054 Erlangen, Germany. Tel: +49 9131 8534444; fax: +49 9131 8534328; website: www.neurologie.med.uni-erlangen.de/oeffentliche_Veranstaltungen.htm

Falk Workshop
The workshop entitled Update in Inflammatory Bowel Diseases will be held in Ljubljana, Slovenia, on 5 May 2001. Further information: Prof Dr S Marković, University Medical Center Ljubljana, Division of Internal Medicine, Japljeva 2, 1525 Ljubljana, Slovenia. Tel: +386 (1) 231 6925; fax: +386 (1) 433 4196; email: sasa.markovic@kclj.si

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 Goodhook, European Postgraduate Gastro-Surgical School, Meibergdeef 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

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) 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 1 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.