Percutaneous drainage of echinococcal cysts
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Published in:
Gut

Citation for published version (APA):
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Gut 2001;48:578-
doi:10.1136/gut.48.4.578

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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 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. 3, 4 Therefore, in PAIR, as a standard procedure, cystography is performed before scolicidals are used. 5 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; left). 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. 6 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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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 study 1 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 gastrooesophageal 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. Elsewhere, 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 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>66</td>
<td>50</td>
</tr>
<tr>
<td>Measurement of BMI</td>
<td>Max Current</td>
<td>16% 15%</td>
</tr>
<tr>
<td>BMI &lt;25–24</td>
<td>16%</td>
<td>15%</td>
</tr>
<tr>
<td>BMI 25–28/24–27</td>
<td>16%</td>
<td>17%</td>
</tr>
<tr>
<td>BMI 27–30</td>
<td>0%</td>
<td>20%</td>
</tr>
<tr>
<td>BMI &gt;30</td>
<td>17%</td>
<td>30%</td>
</tr>
</tbody>
</table>
The critical question is whether 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-randomized 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 in several important aspects), more in depth clinical and epidemiological studies are needed to resolve the apparent enigma.

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Editor,—We read with interest the paper by Kronborg and colleagues (Gut 2000;46:795–800)—a large multicentre study measuring faecal calprotectin levels in high risk populations for colorectal neoplasia. The authors did not discuss their results in comparison with those of Roseth and colleagues1 or Kristinsson and colleagues2 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).

1. Median values for the control subjects were higher and median values for the colorectal cancer (CRC) and polypos groups were much lower compared with the Norwegian group (who had much greater numbers in the CRC group), continuing to markedly reduce the sensitivity of this test.

Furthermore, in the discussion, the authors claim that their results showing no fall in calprotectin levels in patients after polypectomy are similar to those of Kristinsson and colleagues before and after resection for colon cancer. This is a gross misrepresentation of their findings which clearly show that 24/26 patients who underwent colonic resection had a significant fall in faecal calprotectin levels. The other two patients had bypass operations.

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### 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)3</td>
<td>Controls 49</td>
<td>0–12</td>
<td>2.5</td>
<td>80</td>
</tr>
<tr>
<td></td>
<td>Polyps 40</td>
<td>1.5–160</td>
<td>16</td>
<td>80</td>
</tr>
<tr>
<td></td>
<td>CRC 53</td>
<td>4–1000</td>
<td>40</td>
<td>95</td>
</tr>
<tr>
<td>Kristinsson (1998)3</td>
<td>Controls 119</td>
<td>0–12</td>
<td>2.5</td>
<td>93</td>
</tr>
<tr>
<td></td>
<td>CRC 2–950</td>
<td>52</td>
<td>93</td>
<td></td>
</tr>
<tr>
<td>Kronborg (2000)</td>
<td>Controls 488</td>
<td>5–7</td>
<td>7</td>
<td>93</td>
</tr>
<tr>
<td></td>
<td>Polyps 300</td>
<td>5–10</td>
<td>9</td>
<td>43</td>
</tr>
<tr>
<td></td>
<td>CRC 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 by 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.1 A value of 1.5% had 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. 

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 5.44 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 antinuclear, anti-smooth muscle, and antimitochondrial anti-platelet antibodies were absent. The patient was positive for anti-HEV IgG and negative for anti-HEV IgM.

On abdominal sonography the liver appeared enlarged with no intra- or extra-hepatic 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, direct bilirubin 241.11 μmol/l, and alkaline phosphatase 450 IU/l. Markers for HAV, HBV, HCV, CMV, and EBV were negative; she was positive for anti-HEV IgM and negative for anti-HEV IgG. Three weeks after admission the jaundice subsided 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 countries3 and the association observed in Italy between HEV and hepatitis C clearly show that the transmission is not the only means of transmission.4 It is noteworthy that the increased prevalence of 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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Adenocarcinoma arising in columnar lined oesophagus following treatment with argon plasma coagulation

EDITOR.—Following the recent report by Van Laethem et al concerning 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

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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 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 polishes 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; it is a very rare neoplastic 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. 1 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 over 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 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 for two reasons: 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 usefulness 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,6 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

Outcomes 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 (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 (8 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 cases 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 unknown and it may be premature to conclude that the lamivudine resistant phenotype causes severe graft damage.

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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 emphasizes that the optimal management of these difficult and often complicated group of tumours depends on a multidisciplinary team approach and he has edited the text to provide the investigatory, surgical, and oncological aspects of treatment. Firm editorial control allows each of the chapters to be approximately one third of the 48 contributors from Duke University, North Carolina, and approximately 12 contributors from six countries, including the UK, on this side of the Atlantic.

The structure of the chapters is sound and each has a small but useful list of additional reading material which is presented as an addendum with short critical comments on each reference. The main reference lists are comprehensive and up to date.

An introductory section includes chapters on pathology, epidemiology, imaging, and tumour markers. I agree with the authors that the ideal sequence of the investigation of malignant liver tumours remains to be defined and that too many patients receive all possible modalities of imaging as well as a biopsy. They suggest that once the accuracy of the various scanning modalities is decided, it will be possible to reduce the high costs of current investigations. The section on tumour markers is a good critical review of both the values and limitations of the wide range of possible investigations.

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, cryosurgery, 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 numbers of investigations and treatments now available for the management of liver tumours. It provides an excellent introduction for specialist 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 subspecialty exit examinations may trigger a proliferation of self assessment tests, although candidates for part 2 MRCP are currently the main market in the UK. On 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. It is a good transatlantic collaboration. Cases cover a variety 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 conclusions, up to date information, and practical advice as well as 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 cyste") 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, chronic cystic fibrosis, four on cancer, and four on epidemiology ("lessons from"). The chapter titles are intriguing, focusing on biological mechanisms and current management attitudes. Genetics features strongly, as well as 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, after all, 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 pathways 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 virtual 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 interrelated, and the 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 most worthwhile.

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 fucosyltransferases and Ki-ev 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 blockers. 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 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 while it is tempting to follow 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 strength 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, supported by 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 also cover most of the recent publica-
tions but although the cisapride problem is mentioned it is clear concise chapter on short term management, with useful supplement-
ary 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 inhibi-
tors, 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 recom-
mendations on treatment strategies and dos-
ages, which I found particularly helpful. Interestingly, in the "Special management problems" chapter, a different author gives a complete different viewpoint on the Helicobacter pylori/proton pump inhibitor debate, which adds a bit of spice. There is a sensible summary of non-erosive 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 indica-
tions for surgery are discussed. There follows a description of surgical techniques, including laparoscopic fundoplication, and a de-
tailed analysis of short and long term compli-
cations. 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.

All topics have dual authorship with the exception of the useful chapter on legal mat-
ters by PF Schofield. These authors are all UK based apart from MR Salum and SD Weexer (Cleveland Clinic, Florida).

Do you have comprehensive answers to the following questions? If yes, do not read this book!

Question 1. What surgical procedures are now possible for the elderly unfit patient in whom you have just discovered a small malignant rectal polyp?

The outstanding chapter by Cook and McC Mortensen (John Radcliffe Hospital) on transanal endoscopic microsurgery describes this recent advance. This is an unusually large number of good illustra-
tions and tables in this chapter which cover all aspects of this minimally invasive technique.

Question 2. What’s the latest on troublesome haemorrhoids?

The chapter by EA Carapeti and RKS Phillips (St Mark’s Hospital) on the treat-
ment of haemorrhoids is very thorough, end-
ning by focussing on the perioperative care package that has made day case surgery possi-
ble. The goal posts really have moved since the days of lengthy inpatient care for all.

Question 3. Do doctor, what is the chance that this pouch surgery will work?

Are you up to date on the extensive knowl-
edge that has been gained over the past 15 years on complications and long term out-
come of pouch surgery? In the UK, none has performed more first time pouches nor has anyone as great an experience in revi-
sional pouch surgery as John Nicholls, who addresses this topic.

Question 4. Is laparoscopic colorectal surgery here to stay? What are the indications that there is any evidence that it is better than open surgery?

Interestingly, the editors decided to look further afield for the answers in this contro-
versial area.

Coloproctology is the most popular sub-
specialty among general surgical trainees. One reason for this is that it is a specialty on the move. There are recent advances in many other areas left for future editions of this book: management of acute colitis, colonic stenting, training, the input of colorectal nurse specialists, to name a few. I hope these future editions will also have updated gastro-
tenterology, nursing, oncology, and radiol-
ogy. After all, the editors acknowledge in the preface that coloproctology has now been transformed from a purely surgical to a multidisciplinary specialty.

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

CORRECTIONS

Errors occurred in the EUGW abstracts supplement Gut 2000;47(suppl IV). For abstracts A136 and A160, the complete author list for both abstracts is M M Diculescu, E M Ionescu, M Caciorean, M Prunescu, R Iacob, S Iacob, C Apetrechioae, A Oprun. For abstract A271, the complete author list is H J Tan and D 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 Southamp- ton. The authors would also like to acknowled- edge that the work was supported by the Bio- technology and Biological Sciences Research council (BBSRC).

NOTES

GASTRO 2001

The Annual Scientific Meeting of the Malay- sian Society of Gastroenterology and Hepa- tology (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; website: 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, 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 “Autono- mic nervous system” of the German Neurolo- gical Society, “Diabetes and Nervous Sys- tem” of the German Neurological Society, and “Autonomic Nervous System” at the Univer- sity 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, Schwabachenlage 6, D-91054 Erlangen, Germany. Tel: +49 0131 8534444; fax: +49 9131 8534328; website: www.neurologie.med.uni-erlangen.de/oeffentliche_Veranstaltungen.htm

Falk Workshop

The workshop entitled Update in Inflamma- tory Bowel Diseases will be held in Lubljana, Slovenia, on 5 May 2001. Further informa- tion: Prof Dr S Marković, University Medical Center Lubljana, Division of Internal Medicine, Japljeva 2, 1525 Lubljana, 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@aphe- nix.com

EPGS Endosonography Live in Amsterdam

This European Postgraduate Surgical-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: 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 bio- medical 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, Gastro- enteralogy 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 Gion- chetti, Policlinico S. Orsola - Malpighi, Dipar- timento di Medicina Interna e Gastroenterolo- gia, 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 abdo- minal imaging, emphasising the most recent advances in helical CT, MRI, US, and gastrointesinal 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, Con- gress 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 gastroenterolo- gists 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 sub- mission of abstracts is 31 May 2001.