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 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 scolicaid probably enters pericystic liver tissue through breaks in the laminated membrane which cannot be identified by the surgeon’s eyes.1,2 Therefore, in PAIR, as a standard procedure, cystography is performed before scolicidals are used.3 Scolicidals can be safely instilled into the cyst if the laminated layer is intact and a cystobiliary fistula has been excluded. In our experience, cystography is only appropriate in Ghari 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 of the fistulae 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.4 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 study6 reported a strong positive association between body mass index and the prevalence of reflux symptoms (table 1). One possible explanation for the difference between the two studies is the younger age distribution of the US cohort. The prevalence of overweight has increased dramatically throughout Europe and North America in recent decades.7 As a consequence, the younger US cohort is likely to have accumulated more person years of overweight by a given age, and the risk of reflux symptoms may be related to both the magnitude and years of overweight exposure. The authors also concluded, in the light of their findings, that weight reduction may not be justifiable as an antireflux therapy. Even if overweight is a poor predictor of reflux symptoms, this does not necessarily imply that weight reduction will not be of benefit in providing symptom relief. A significant beneficial effect of weight loss on symptoms of gastro-oesophageal reflux in overweight patients has recently been reported in a small study involving 34 patients.8 In addition, the degree of weight loss was directly correlated with improvement in symptom score. Elsewhere, strong and independent associations have been reported between both overweight and reflux symptoms and oesophageal adenocarcinoma.9,10 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>Gaster et al</th>
<th>Looch et al</th>
</tr>
</thead>
<tbody>
<tr>
<td>Setting</td>
<td>Sweden</td>
<td>USA</td>
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<tr>
<td>Sample size</td>
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</tr>
<tr>
<td>Sex</td>
<td>M/F</td>
<td>M/F</td>
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<tr>
<td>Mean age (y)</td>
<td>50</td>
<td>50</td>
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<tr>
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<td>Maximum</td>
<td>Current</td>
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<tr>
<td>BMI &lt;25–24</td>
<td>16%</td>
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<tr>
<td>BMI 27–30</td>
<td>20%</td>
<td>30%</td>
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<tr>
<td>BMI &gt;30</td>
<td>17%</td>
<td>30%</td>
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</table>

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Letters, Book reviews, Correction, Notes

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5 S. 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 in our view to be 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, presum-ably 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 epidemiologic studies. Moreover, if Maric and Cheng’s hypothesis on the importance of accumulated time with overweight is correct, our study, which assessed body mass index at different points in time in the past should have had a greater chance of ascertaining any true association compared with the study of Locke et al which dealt with only current body mass index.

Non-differential misclassification of both exposure (body mass index) and outcome (reflux symptoms) due to imperfect recollection in our study may have attenuated our estimates. 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. Although we have not carried out uncontrolled non-randomised intervention studies, like the ones 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 several important factors), more in depth clinical and epidemiological studies are needed to resolve the apparent enigma.

Reply

EDITOR,—We read with interest the paper by Roseth et al.1 We 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 Roseth et al, Kristinsson et al, and Kronborg and colleagues

<table>
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<tr>
<th>Study</th>
<th>No of patients</th>
<th>Range</th>
<th>Median</th>
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<tr>
<td>Roseth (1993)</td>
<td>Controls 49</td>
<td>0–12</td>
<td>2.5</td>
<td>80</td>
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<tr>
<td></td>
<td>Polyps 40</td>
<td>1.5–160</td>
<td>16</td>
<td>80</td>
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<tr>
<td></td>
<td>CRC 53</td>
<td>4–1000</td>
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<td>95</td>
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<td>52</td>
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CRC, colorectal cancer.

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 Kristinsson2 (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 calprotectin levels after polypectomy. We mentioned that the levels after colorectal resection for cancer in the study by Kristinsson and colleagues3 (median 10.3 mg/l, range 1–200) were similar or even higher than those after polypectomy in our study.

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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 caproctin 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.

2 Roseth AG, Kristinnson J, Fagerhol MK, et al. Fecal calprotectin: A novel test for the diagno-

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 of increasing public health concern in the UK. 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.5

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 (aspartateaminotransferase (AST) 1990 IU/l; alanine aminotransferase (ALT) 1626 IU/l). Eight days after admission total bilirubin was 528.32 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, titres had 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 had risk factors for HEV.

The increased prevalence of this infection among haemodialysis patients in developed countries6 and the association observed in Italy between HEV and hepatitis C clearly means that this virus is not the only means of transmission.7 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-


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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 adenocarci-

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 oesophagal wall but no obvious metastases. However, at laparotomy, he was found to have an

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Letter, Book reviews, Correction, Notes

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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 restoration 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 which are 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 thermocoagulation. 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 new 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 because it raises 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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**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 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 (four 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. 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 remains unclear 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 effectiveness 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 may well 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 choice 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 these difficult and often complicated group of tumours depends on a multidisciplinary team approach and he has edited the text to integrate the investigative, surgical, and oncological aspects of treatment. Firm editorial control allows each of the chapters to be read as a complete essay but I found that the book also read well as a sequential text with minimal duplication of material in each of the sections. The up to date nature of the book is well illustrated by an example from the section on gene therapy which gives details of a phase one study, from November 1998, of recombinant p53 adenovirus gene therapy.

The book is based on the experience at Duke University, North Carolina, and approximately one third of the 48 contributors are from that institution. However, European practice is well represented by 11 of the 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 value 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, cryoablation, 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 to the current management but at the same time includes enough thought provoking 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 texts, although candidates for part 2 MRCP are currently this book’s 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. This is good transatlantic collaboration. Cases cover the entire range of 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 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 crypt”) 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 actually 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 and extracellular immune mechanisms in acute pancreatitis are particularly well done, although the subsequent contributions on varieties of clinical management contain nothing new. The section on chronic pancreatitis contains some overlap between chapters but the contribution on mechanisms of fibrosis and potential therapy using inhibitors is fascinating, if still a distant dream. The chapters on cystic fibrosis are detailed and very informative 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 worth a careful read. The section on pancreatic cancer is, like the disease, disappointing, representing the essentially bleak situation of specialists searching around for mechanisms and treatment modalities with little success.

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

M SARNER

Picture the scene. An international conference on gastroenterology, delegates flown 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 really interesting stuff like fucosyltransferases and Ki-ru 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 OTCH2 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 combing Germanic thoroughness and 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 Gauggnol, as 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 material is traditional but contains some little gems. The section on the definition and clinical presentation of abdominal syndromes includes very helpful information about subgroups of dyspepsia and ways of distinguishing between functional dyspepsia and irritable bowel syndrome. The epidemiology is, as you would expect, thorough, and the chapter on the pathophysiology of functional dyspepsia, with almost 100 references, is a mine of information with implications for research as well as clinical practice. There is a good section on psychosomatic factors, once again well referenced and reasonably up to date. The chapters on diagnosis and management also cover most of the recent publications but although the cisapride problem is acknowledged, the recent work on a *Helicobacter pylori* test, and treat strategy from Bytzer's group did not quite get in. There is a terrifying looking algorithm on the manage- ment of dyspepsia which, on closer inspection, turns out to be perfectly sensible. Two randomized controlled trials of herbal medicines in the treatment of dyspepsia are referenced. The section on psychological treatment is short but contains an invaluable table describing the basis of the therapeutic relationship between patient and physician.

There is something here for everyone. The family physician will have to be selective but will find useful guidance on diagnostic and management approaches. Trainees in gastroenterology will find the book stimulating and might well start asking some new research questions about dyspepsia. Established gastroenterologists will need to read this book on the plane when they are next subjected to the days of lengthy inpatient care. Thankfully the content and arrangement of the material is clear and the book has clearly been sponsored by Byk Gauggnol, as 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.

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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 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 “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 0131 8534444; fax: +49 1931 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 Markovic, University Medical Center Ljubljana, Division of Internal Medicine, Japljeva 2, 1525 Ljubljana, Slovenia. Tel: +386 (1) 231 6925; fax: +386 (1) 435 4190; 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 Surgical-Gastrointestinal 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

**Gastroenterology and Endotherapy: XIXth European Workshop**

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

**Falk Symposium**

The symposium Inflammatory Bowel Disease: A Clinical Case Approach to Pathophysiology, Diagnosis, and Treatment will be held in Bologna, Italy on 22–23 June 2001. Further information: Prof Dr M Campieri/ Dr P Gionchetti, Policlinico S. Orsola - Malpighi, Dipartimento di Medicina Interna e Gastroenterologia, Via Massarenti 9, I-40138 Bologna, Italy. Tel: +39 (051) 6364 116 or 6364 122; fax: +39 (051) 3923588; 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@mafervizi.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 3638; email: merat@ams.ac.ir; website: www.ams.ac.ir/igch. Deadline for submission of abstracts is 31 May 2001.