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.¹⁻⁴ 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.

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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 followings 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 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 centre, 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 unknown 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* (2000;46:93-97) indicates that underlying cancer will be diagnosed in about 1 in 1000 patients presenting with uncomplicated dyspepsia under 45 years of age. However, the calculated 95% confidence intervals for this are wide (1 in 2963 to 1 in 300).

An important question in considering the significance of this finding is whether the prevalence of cancer in these patients with benign dyspepsia is any different from that in the general population. In our own country, Scotland, the chance of a patient presenting with gastro-oesophageal cancer before the age of 50 is 1 in 909 (ISD Scotland Cancer Surveillance Group Data Request and Analysis Service) and half of those have presented with the cancer within the age band 45-49. Most of these patients will have had the tumour present in their stomach for a considerable time prior to clinical presentation, which would have been detected by screening endoscopy five years earlier. Even allowing for the fact that population based rates of gastro-oesophageal cancer are higher in Scotland than Alberta,¹ this suggests that the prevalence of underlying cancer in patients presenting with uncomplicated dyspepsia may not be different from that in the general population. Consequently, offering endoscopy to patients with simple uncomplicated dyspepsia to detect cancer may merely represent screening of the general population.

There has been a general assumption that a tumour growing in the stomach will produce dyspeptic symptoms. However, there is no evidence for this. Tumours developing in the colon or other parts of the gastrointestinal tract rarely, if ever, cause symptoms until they produce complications such as bleeding or obstruction.

A very small proportion of patients presenting with uncomplicated dyspepsia will have underlying cancers but this finding may be unrelated to their symptoms. Unless uncomplicated dyspepsia is confirmed to be a symptom of underlying malignancy, then one would be as well to recommend offering endoscopy to patients presenting with a

sprained ankle in order to pick up underlying gastro-oesophageal cancer.

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1 Electronic version of Parkin DM, Whelan SL, Ferlay J, Raymond L, Young J, eds. *Cancer incidence in five continents, vol. VII*. IARC Scientific Publications no. 143. Lyon, France: International Agency for Research on Cancer, 1997.

BOOK REVIEWS

Malignant Liver Tumours. Edited by P-M Clavien (Pp 363; illustrated; £112.00). USA: Blackwells, 1999. ISBN 0-632-04406-3.

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

In the preface, Professor Clavien emphasises that the optimal management 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 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 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 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

Self Assessment Colour Review of Gastroenterology. Edited by A Forbes, NH Gilinsky (Pp 188; illustrated). UK: Manson Publishing, 1999. ISBN 1 874545 47 2.

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 range of luminal 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 very 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

Pancreatic Disease. Edited by PJ Lankisch, EP DiMagno (Pp 272; illustrated; £49.50). Germany: Springer-Verlag, 1999. ISBN 3-540-65357-0.

This is a meetings book ("songs from the show") 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, 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 events, and immune mechanisms in acute pancreatitis are particularly well done, although the subsequent contributions on varieties of clinical management contain nothing new. The section on chronic pancreatitis contains some overlap between chapters but the contribution on mechanisms of fibrosis and potential therapy using inhibitors is fascinating, if still a distant dream. The chapters on cystic fibrosis are detailed and very interesting with good 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.

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