Living related liver transplantation: a Japanese experience and development of a checklist for donors’ informed consent

In the February 2002 issue of Gut, Broelsch et al argued for a controversial therapy of living related liver transplantation (Gut 2002; 50:143). The Japanese experience is somewhat different from those of other countries.

The development of this medical procedure at our institute has entailed a strict self-regulatory process.

(1) Each case is reviewed by an institutional professional committee that examines the medical indication. The transplant team prioritises the safety of donors, and no donor deaths have been reported so far.

(2) Informed consent obtained by transplant teams is reassessed by the institutional ethics committee to check for the absence of coercion and guarantee the right to refuse surgery until the last moment. The ethics committee has developed a checklist (table 1) and basically all donors are interviewed by a member of the ethics committee before surgery. Donor candidates are restricted to a spouse or relatives within the third degree of blood relationship.

(3) Information disclosure to media. In order to facilitate social acceptance of the procedure, relevant information continues to be disclosed to the press.

While these institutional efforts are essential, we suppose there are more substantial reasons for the striking increase in this type of surgery. One obvious explanation is the assumption in Japan to accept the concept of brain dead organ donors, but another may be the strong family bonds that are fundamental to Japanese culture. Traditionally raised in a family-oriented society, Japanese people may not hesitate to give their organs to save a family member even if there is a small but perhaps fatal risk associated with the practice. This hypothesis needs further corroboration; however, on the other hand, many would assert that love for family is a universal value.

Hence we are faced with two academic questions: firstly, whether or not liver transplants using living donors will prevail to a similar extent in other countries where organ procurement from the brain dead is socially prohibited there; and secondly, whether or not this procedure can provide a solution to the lack of available organs in countries where organ procurement from the brain dead is permitted.

Japanese transplant surgeons are now going abroad to teach the living related liver transplant technique while patients and their family from countries where transplants from the brain dead are not permitted come to Japan to undergo living donor surgery. The situation described here clearly shows that while the world surgical community frequently shares advancements in techniques, regional and sociocultural values greatly influence their implementation.

A Akabayashi, M Nishimori, M Fujita, B T Slingsby
Department of Biomedical Ethics, School of Public Health, University of Kyoto Graduate School of Medicine, Kyoto, Japan

Correspondence to: Dr A Akabayashi, Department of Biomedical Ethics, School of Public Health, University of Kyoto Graduate School of Medicine, Yoshida-Konoe-cho, Sakyo-ku, Kyoto 606-8501, Japan; akirasan@phb.med.kyoto-u.ac.jp

References

Endoscopic surveillance of premalignant gastric lesions

We read with interest the study by Whiting and colleagues (Gut 2002;50:378–81). The study has further highlighted the importance of early detection of gastric cancer and also given further emphasis on ways to prevent the multistep progression in gastric carcinogenesis. However, we would like to make the following comments.

Firstly, one in five patients in this group had lesions which, according to Whiting et al, should be followed up by yearly endoscopies. Despite the low acceptance rate in screening

---

**Table 1** Checklist for interviews with donors for living related liver transplantation

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>General profile of the recipient and the donor</td>
</tr>
<tr>
<td></td>
<td>(a) A brief medical history of the recipient</td>
</tr>
<tr>
<td></td>
<td>(b) Family tree</td>
</tr>
<tr>
<td>2.</td>
<td>Informed consent</td>
</tr>
<tr>
<td></td>
<td>(a) When and how did you come to know about living related donor liver transplant?</td>
</tr>
<tr>
<td></td>
<td>(b) Who explained the details of the transplant surgery, and how many times?</td>
</tr>
<tr>
<td></td>
<td>(c) Under what circumstances (one to one, or with others present)?</td>
</tr>
<tr>
<td></td>
<td>(d) Do you clearly understand the procedure of the surgery?</td>
</tr>
<tr>
<td></td>
<td>(e) Do you fully understand the risks and benefits of the treatment (including short term and long term risks for the donor, and the success rate of graft attachment for the recipient)?</td>
</tr>
<tr>
<td></td>
<td>(f) Have you been given information and explanations about alternative therapies?</td>
</tr>
<tr>
<td></td>
<td>(g) Have you been given enough time to ask questions? Have you been invited to ask questions?</td>
</tr>
<tr>
<td>3.</td>
<td>Decision making process</td>
</tr>
<tr>
<td></td>
<td>(a) Have you consulted with anyone?</td>
</tr>
<tr>
<td></td>
<td>(b) Were there any coercion by other family members or relatives (for example, if you do not agree to be a donor, the patient will surely die)?</td>
</tr>
<tr>
<td></td>
<td>(c) Is your decision completely voluntary?</td>
</tr>
<tr>
<td>4.</td>
<td>Psychosocial aspects</td>
</tr>
<tr>
<td></td>
<td>(a) Do you have any anxiety about your surgery?</td>
</tr>
<tr>
<td></td>
<td>(b) Do you have any problems in your life (for example, business or social relationships)?</td>
</tr>
<tr>
<td></td>
<td>(c) Do you have any financial problems?</td>
</tr>
<tr>
<td>5.</td>
<td>Protection of the donor’s right</td>
</tr>
<tr>
<td></td>
<td>(a) You have the right to refuse or withdraw your consent until the last moment.</td>
</tr>
<tr>
<td></td>
<td>(b) You will not suffer any disadvantage if you decide to refuse or withdrawal.</td>
</tr>
<tr>
<td>Interviewer’s assessment</td>
<td></td>
</tr>
<tr>
<td>(1)</td>
<td>The donor is well informed. ☐ Yes ☐ No</td>
</tr>
<tr>
<td>(2)</td>
<td>The donor has a good understanding of the entire process. ☐ Yes ☐ No</td>
</tr>
<tr>
<td>(3)</td>
<td>The donor is fully capable of making a decision. ☐ Yes ☐ No</td>
</tr>
<tr>
<td>(4)</td>
<td>The donor’s decision is completely voluntary and firm. ☐ Yes ☐ No</td>
</tr>
<tr>
<td>(5)</td>
<td>The decision has been reached without any evidence of coercion. ☐ Yes ☐ No</td>
</tr>
<tr>
<td>(6)</td>
<td>The donor’s right has been fully protected. ☐ Yes ☐ No</td>
</tr>
<tr>
<td>(7)</td>
<td>The donor is without significant psychosocial problems. ☐ Yes ☐ No</td>
</tr>
<tr>
<td>Time of interview</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Interviewer’s signature (a member of ethics committee)</td>
<td></td>
</tr>
<tr>
<td>Date</td>
<td></td>
</tr>
</tbody>
</table>
programmes as noted by the authors, this would create an enormous workload on already over burdened endoscopic units. Clearly, further modes of selection of high risk patients with atrophy and metaplasia is desirable. Mutations in p53, APC, and mismatch repair genes have been reported in intestinal metaplasia. Some of these mutations are associated with an enhanced progression to advanced lesions in the multistep sequence of gastric carcinogenesis. High throughput methods for the detection of gene polymorphisms associated with increased cancer risk, such as interleukin 1 polymorphisms, are likely to be available in the near future. In addition, alteration in gastric secretion of pepsinogen may be used as an aid in early detection of premalignant lesions.

Secondly, the authors have not provided us with data regarding the Helicobacter pylori status of the patients. Their results may have been different if successful eradication of H pylori was achieved in the follow up group, a situation more relevant to current practice. It is now universally accepted that H pylori infection is the most important factor in gastric carcinogenesis with both host and bacterial virulence factors playing a role.

The European Helicobacter pylori Study Group strongly recommended H pylori eradication for patients with atrophic gastritis, after gastric cancer resection, and first degree relatives of patients with gastric cancer (presented at the Maastricht 2–2000 conference). There are emerging data that intestinal metaplasia may be replaced by normal gastric mucosa following H pylori eradication. In summary, we feel that less invasive and more cost effective modes for detection and follow up of premalignant gastric lesions are required and hopefully are on the horizon. In the meantime, it appears that a screen and treat strategy for H pylori is desirable. Mutations in the CagA gene of H pylori are associated with intestinal metaplasia. Some of these mutations are associated with an enhanced proliferation in Barrett's epithelium, although approximately 30% of patients develop squamous islands within the Barrett's segment. Within each study to date, the same dose of PPI has been given to each patient. However, as oesophageal pH monitoring studies show, there is wide variation in acid reflux between patients. Effective control of acid reflux into the oesophagus is important in preventing Barrett's epithelium and our study of patients treated with omeprazole for up to six years showed that none developed dysplasia and our study of patients treated with omeprazole for up to six years showed that none developed dysplasia during follow up. Therefore, PPI dose should be that which inhibits acid reflux effectively and will vary from patient to patient. Patients may resist frequent pH monitoring to determine the effective PPI dose so we would support the views of Fass et al that consideration should be given to treating patients with longer segments of Barrett's oesophagus with higher doses of PPI. Moreover, Barrett's patients with associated moderate to severe reflux oesophagitis should also be treated with higher PPI doses.

S Sebastian, J Seery, C O'Morain, S M Buckley
Department of Gastroenterology, Adelaide and Meath Hospital, Dublin, Ireland.
Correspondence to: Dr M Buckley

References

Smoking and ulcer healing
We read with interest the paper by Wong et al (Gut 2002;50:322–5) on prediction of therapeutic failure in patients with bleeding peptic ulcer but are surprised they did not include smoking in their logistic regression analysis. The background prevalence of smoking is sufficiently high in western communities to be a useful marker if found significant. The association between smoking and ulcer healing and smoking and cardiovascular and respiratory disease raises the issue of whether smoking may be a risk factor both for ulcer rebleeding and mortality. It is recognised that cardiovascular and respiratory mortality is a substantial contributor to peptic ulcer disease related mortality. Addition of smoking may improve the predictive performance of their receiver operating curve and the value of their model in clinical practice.

A Duggan, N Rutherford
Department of Gastroenterology, John Hunter Hospital, NSW 2310, Australia.
Correspondence to: A Duggan, John Hunter Hospital, Locked Bag 1, Hunter Region Mail Centre, NSW 2310, Australia; aduggan@hunter.health.nsw.gov.au

Oesophageal pH monitoring in Barrett's oesophagus
We wish to comment on an interesting paper published previously in Gut which we inadvertently overlooked at the time. Fass et al (Gut 2001;48:310–13) reported that there was a positive correlation between percentage time that oesophageal pH was less than 4 in 24 hours and the Barrett's oesophagus length. We have published previously in Thorac Cardiovasc Surg (1999;117:572–80) that the extent of Barrett's oesophagus depends on the status of the lower oesophageal sphincter and the degree of oesophageal acid exposure. J Thorac Cardiovasc Surg 1999;117:573–80.

Author's reply
I would like to thank Drs Neumann and Cooper for their comments on our article on the correlation of oesophageal acid exposure with Barrett's oesophagus length (Gut 2002;50:310–13). In recent years, laboratory investigation has focused on factors that promote the development of Barrett's oesophagus. Surprisingly, our understanding of the mechanisms that are responsible for the emergence of Barrett's epithelium remains extremely poor. Despite the tendency in the literature to group...
Influence of clinical factors, drug use, and food intake on the glutathione system

In a previous issue of Gut, Hoensch and colleagues (Gut 2002;50:235–40) using antral and duodenal biopsies, reported on a variety of factors such as sex, age, drug use, and food intake that influence the concentration of glutathione and the activity of glutathione S-transferase. All of these factors either singly or in combination significantly affect glutathione metabolism within the gastric mucosa.

Curiously, one critical factor that may have influenced their measurements, namely Helicobacter pylori infection, was not mentioned in their paper. This omission is particularly important as the majority of the patients that these investigators examined had endoscopic findings strongly suggestive of infection with H pylori (gastric erythema, erosions, or ulcers). Previous studies by some of the coauthors in the Hoensch paper1 as well as by our group have clearly demonstrated that H pylori infection is associated with marked depletion by approximately 50% of reduced glutathione within the gastric epithelium, and that concentrations of reduced glutathione are restored to normal by eradication of H pylori. Failure to stratify patients for H pylori infection makes other conclusions in the study less compelling. Consideration of the presence of H pylori may explain why the antrum, the preferred site of H pylori colonisation, had the lowest concentration of reduced glutathione in the gastrointestinal tract.

H pylori is well known to induce formation of reactive oxygen species (ROS), particularly in the antrum,1 and result in oxidative damage to DNA. Influenmatory host cells, such as activated phagocytic leucocytes, are the primary source of this oxidative stress, although H pylori per se may generate ROS and result in stimulation of oxidative signalling pathways in gastric epithelial cells.2 Recent evidence strongly suggests that levels of reduced glutathione correlate inversely with parameters of acute and chronic inflammation in vivo.3,7 Thus attenuation of reduced glutathione in the gastric mucosa of H pylori infected patients may be due to both the direct effects of H pylori induced expression of oxidative signalling pathways and the associated inflammatory response.

Intra- and extracellular oxidative stresses induced by H pylori in association with depletion of glutathione and/or genetic polymorphisms of enzymes that control its metabolism may compromise normal epithelial cell function and enhance susceptibility to gastric cancers. In considering the gastric glutathione system, the effect of H pylori should not be ignored.

H Shirin
Tel Aviv University/Edith Wolfson Medical Center, Holon, Israel

J T Pinto
American Health Foundation, Valhalla, NY, USA

S F Moss
Brown University/Rhode Island Hospital, Providence, RI, USA

Correspondence to: S F Moss, Rhode Island Hospital, 593 Eddy St, APC 445, Providence, RI 02903, USA; Steven_Moss_MD@Brown.edu

References


Authors’ reply

We appreciate very much the comments made by Shirin et al concerning our publication (Gut 2002;50:235–40).

In our study (Gut 2002;50:235–40), we investigated a wide variety of factors which had not been evaluated entirely at the time this paper was written. In the meantime, the reported new findings of our group on H pylori were discovered in another patient population from the Netherlands.1,2

After we received the comments of Shirin et al, we looked again at the data of our patients from Germany for test of H pylori. We found that H pylori had a significant effect on one of the parameters of the gastrointestinal glutathione (GSH) system. The level of glutathione S-transferase (GST) A (alpha) in the antral mucosa was significantly depressed (p<0.05) in H pylori infected patients (4.8 (7.3) μg/mg cytosomal protein (n=63) v 5.6 (6.9) (n=60)). The values given are means (SD) using the Wilcoxon test for comparison of means.

The status of H pylori infectivity was determined in the gastric mucosal biopsy specimens using the urease test which was read as either positive (H pylori present) or negative (H pylori absent) from the colour reaction (CLO test).

The other parameters (GSH concentration, GST enzyme activity, levels of GST P (pi) and GST T (theta)) were not affected in the antral and duodenal mucosa by H pylori status. The GST A level of the duodenal mucosa was also not significantly influenced by H pylori.

These results corroborate the findings published recently by our research group3 and by Shirin and colleagues.1 In our large group of patients from Germany, H pylori infection was associated with lower GST A levels in the antral mucosa. Eradication of H pylori was performed only in patients with ulcers and erosions but these patients were not followed up by endoscopic routinely.

H pylori was the only factor that had a significant depressing effect on antral GST A level. H pylori had no influence on duodenal GST A, GST P, or antral GST T, which confirms that vegetable and fruit stimulation of these enzymes was not confounded by H pylori.
However, it has to be considered that *H pylori*-evaluation and eradication in patients from the Netherlands were done only in non-ulcer dyspepsia while patients from Germany comprised various pathological endoscopic diagnoses apart from non-ulcer dyspepsia.

Our cross sectional study confirms that *H pylori* seems to depress the GST A component of the enzymatic GSH system in the antral mucosa of the stomach. Depression of GST A levels could mean increased susceptibility of the stomach mucosa towards carcinogenic insults.

H Hoensch, I Morgenstern
Leitender Arzt, Innere Abteilung, Gastroenterologie und Onkologie, Kreiskrankenhaus Groß-Gerau, Wilhelm Sepp-Stajle 3, 64521 Groß-Gerau, Deutschland

Correspondence to: H Hoensch; H.P.Hoenisch@vfl.uni-frankfurt.de

References


*Mycobacterium avium* subspecies *paratuberculosis* as a cause of Crohn’s disease

The debate by Professor Quirke (Gut 2001;49:757–60) was an interesting review of the hypothesis of a microbiological aetiology of Crohn’s disease. He indicates that “the hypothesis remains controversial and unproven.” The point is that proof is never absolute, and indeed the objective of research is not correct. Proof is pragmatic not absolute, and indeed the objective of research is to disprove the hypothesis rather than to prove it. Koch himself however recognised the weakness of his postulates in that although he felt that the plausibility was clear even though the concept which itself lacks “proof”, and so conforming to the paradigm of “autoimmunity”, a concept which itself lacks “proof”, and indeed the criteria of proof in putative autoimmune disease have never been defined. A paradigm of a microbiological causation of Crohn’s disease has to be based on two factors. The first of these is the statistical association between the disease and a putative microbe but the difficulty of this is the lack of robust detection methods for identification. The second is plausibility. It is interesting to reflect that for many years acute hepatitis was accepted as being a viral disease and indeed became known as “viral hepatitis”, well before the viruses had been identified. The plausibility was clear even though microbiological science had not progressed so far to identify the viruses themselves. In respect of Crohn’s disease, we need to continue to think as to whether it is plausible that the disease might be microbiological, even in the absence of a definite microbe. The development of the paradigm and identification of a specific microbe are different scientific processes.

Plausibility is founded on existing knowledge and models, based on epidemiology and pathology, the main foundations of Western clinical medicine. What therefore do we think of the pathology of Crohn’s disease? Firstly, it is clear that Crohn’s disease is not a homogeneous disease but a variety of different patterns of inflammatory disease of the intestinal tract. The hallmark of Crohn’s disease is firstly a patchy inflammation of the gastrointestinal tract, including perianal areas of skin. Granulomas are another hallmark and fissuring a third. We can go on in this way but the more criteria that we add, the more it would appear that the disease is a heterogeneous group. In this case cannot define Crohn’s disease, we do not really know what it is, and so a concept of causation is going to be based on a very fragile foundation.

However, we can make progress, especially if we look at the “classical” type of Crohn’s disease involving the right side of the colon, the caecum, and the terminal ileum, with fissuring and granulomatous disease. This type of disease looks very much like tuberculosis, so much so that if it presents in an Asian patient the disease is usually called tuberculosis whereas if it presents in a non-Asian patient it is usually called Crohn’s disease. The similarities to tuberculosis are so powerful, then clearly causation is likely to be very similar. A further important feature is the epidemiological observation of family clustering across genetic boundaries, the husband/wife associations which point very much towards a transmissible agent. Finally, there are the parallels with Johne’s disease in animals which is usually called Crohn’s disease being an equivalent in the human. In terms of response to antimicrobial compounds, do we feel that some studies suggesting benefit are more, less, or equally important to those that suggest no benefit? It depends on the attractiveness of the microbiological paradigm to the individual—some people are anxious to find a cause for Crohn’s disease whereas others see no practical advantage of this and are happy to remain without a paradigm other than “inflammatory bowel disease”. Response to one or more given antibiotics cannot be laid down as a criterion of proof of microbiological causation but could help strengthen a cause of disease. Although we can always be wrong, and indeed we often are, to be totally sceptical denies the opportunities for scientific progress. Research must be based on hypothesis and paradigm.

D S Grimes
Blackburn Royal Infirmary, Blackburn, Lancs B81 3LQ, UK; susan.rogers@mail.bhrv.nwest.nhs.uk

www.gutjnl.com
Modern Management of Cancer of the Rectum

This is a remarkable little book. A brief review of contributors will whet the appetite: a quick read of the first chapter by Drs Shelton and Goldberg will soon confirm your decision to buy. This initial chapter is an engaging review of the key writings of the leaders of surgical thought over the centuries and provides a rare insight into how we have arrived where we are today. The book continues with the rich but often all too brief reviews of the many components of the colorectal cancer scene. This is the most important of all of the human cancers as we already know enough to cure more people of bowel cancer than all of the other internal cancers put together. Nevertheless, even though the disease has changed where knowledge may be incomplete: this book will provide a brief summary of what the surgeon must know about PS3 or the medical oncologist about TME. The obvious and the necessary are mercifully omitted while the uncommon is usually well covered. Rare tumours, for example, are splendidly complete and the book provides valuable detail and formidable lists of references.

For any book the scene is moving too rapidly to be completely up to date. Details of the potential of magnetic resonance imaging (MRI) for example and the currently confused state of knowledge about who should have which type of radiotherapy. Nevertheless, even in these difficult areas, the writers have a constant sense of direction which will seldom be felt, even by an expert, to be off target. Seen through the eyes of a somewhat reactionary reviewer, the importance of laparoscopic surgery in the management of colorectal cancer seems a little which I consider spurious the argument tends to be off target. Seen through the eyes of a quick read of the first chapter by Drs Shelton and Goldberg will soon confirm your decision to buy. This initial chapter is an engaging review of the key writings of the leaders of surgical thought over the centuries and provides a rare insight into how we have arrived where we are today. The book continues with the rich but often all too brief reviews of the many components of the colorectal cancer scene. This is the most important of all of the human cancers as we already know enough to cure more people of bowel cancer than all of the other internal cancers put together. Nevertheless, even though the disease has changed where knowledge may be incomplete: this book will provide a brief summary of what the surgeon must know about PS3 or the medical oncologist about TME. The obvious and the necessary are mercifully omitted while the uncommon is usually well covered. Rare tumours, for example, are splendidly complete and the book provides valuable detail and formidable lists of references.

For any book the scene is moving too rapidly to be completely up to date. Details of the potential of magnetic resonance imaging (MRI) for example and the currently confused state of knowledge about who should have which type of radiotherapy. Nevertheless, even in these difficult areas, the writers have a constant sense of direction which will seldom be felt, even by an expert, to be off target. Seen through the eyes of a somewhat reactionary reviewer, the importance of laparoscopic surgery in the management of colorectal cancer seems a little which I consider spurious the argument tends to be off target. Seen through the eyes of a somewhat reactionary reviewer, the importance of laparoscopic surgery in the management of colorectal cancer seems a little which I consider spurious the argument tends to be off target. Seen through the eyes of a somewhat reactionary reviewer, the importance of laparoscopic surgery in the management of colorectal cancer seems a little which I consider spurious the argument tends to be off target. Seen through the eyes of a somewhat reactionary reviewer, the importance of laparoscopic surgery in the management of colorectal cancer seems a little which I consider spurious the argument tends to be off target.

Abdominal Ultrasound

Mike Stocksley moved from a career in clinical ultrasound to teaching and is now senior lecturer in the Faculty of Health at South Bank University. His background as an educator is readily apparent in this excellent book which for him was clearly a labour of love.

Despite the increasing complexity of investigations available, ultrasound remains an important tool in the investigation of abdominal pathology, its ready availability and resulting popularity do not however imply that it is a straightforward or simple skill. It is probably the most operator dependent imaging modality, and mastery of the underlying concepts, proper performance of a scan, awareness of normal appearances, detection of relevant findings, and their correlation into a unifying diagnosis requires not only appropriate training but also extensive hands-on experience. These factors are often under appreciated by physicians, and if there is one thing guaranteed to aggravate the busy radiologist, it is a request for a “quick ultrasound” or for one to “just have a look”. Mr Stocksley clearly appreciates the complexities of the topic and has produced a book which, while quite short and inexpensive, manages to be both practical and informative. The opening chapter covers the basics including choice of probe, use of coupling gel, patient preparation, and scanning positions. This follows a straightforward explanation of the principles and applications of Doppler ultrasound; reading this chapter caused the reviewer to heartily wish that Mr Stocksley had been in close proximity while he was studying for his part 1 FRCR physics! Having dealt with the basics, the book proceeds with the nitty gritty of practical abdominal ultrasound and there are excellent chapters on the “usual suspects” – the liver, biliary tree, pancreas, spleen, and urinary tract, as well as
more esoteric subjects such as the adrenal glands, muscle, and bowel. Each chapter is laid out similarly, with an initial description of functions and anatomy of the organ followed by the optimal scanning technique and normal ultrasound appearances before a discussion of pathology. The book is lavishly illustrated with over 250 illustrations, including line drawings, photographs to demonstrate scanning positions, and ultrasound images, with a nice balance between normal and pathological appearances. Advice boxes scattered throughout the text give useful tips on pitfalls to avoid, measures to improve scanning technique, and the relevance of findings.

Quibbles with this book are relatively minor. I would have welcomed a chapter on endoscopic ultrasound including endoanal; this area is underrepresented in the text. Also, one feels the clinical advice is in places oversimplistic: for example, in a table on abdominal pain, stating that “pain in both left sides and right = cancer” is of limited value. The impression that this book is predominantly aimed at ultrasonographers in training is reinforced by emphasis on such topics as planning an ultrasound room and report wording. However, these caveats aside, this is an excellent book with pitfalls to avoid, measures to improve scanning technique, and the relevance of findings.

N Power

Due to an error in the production process, parts B and C of Figure 2 in the paper by Rueemmele et al in the December issue of the journal (Gut 2002; 51: 842–8) were printed incorrectly. The figure is reprinted here.
The Future of Gastro-entero-hepato-pancreatology is bright

This Academic Farewell Symposium of Guido NJ Tytgat will be held on 12 December 2002 in Amsterdam, the Netherlands. Deadline for registration is 1 November 2002 (no registration fee) and registration should be done via email to: j.goedkop@amc.uva.nl.

Cancer of Oesophagus and Gastric Cardia: from Gene to Cure

This conference will be held on 13–15 December 2002 in Amsterdam, The Netherlands. Further information: European Cancer Centre, PO Box 9236, NL 1006 AE Amsterdam, The Netherlands. Tel: +31 (0)20 346 2547; fax: +31 (0)20 346 2525; email: ecc@ikca.nl

Imaging of the Abdomen: an Update

This will be held on 23–24 January 2003 in Amsterdam, the Netherlands. Further information: visit the website www.epgs.nl or email epgs@amc.uva.nl. Tel: +31 20566 3926/4386.

The Sheila Sherlock Memorial Symposium

Dame Sheila Sherlock, who died earlier this year, was responsible for creating hepatology at the Royal Free Hospital, London. This memorial symposium will take place on 26–28 January 2003 at the Royal Free Hospital, London, UK. Further information: Terri Dolan, Royal Free and University College Medical School, Royal Free Campus, Centre for Hepatology, Upper 3rd Floor, Rowland Hill Street, London NW3 3PF, UK. Tel: +44 (0)207 433 2851; email: t.dolan@rfc.ucl.ac.uk

3rd Chester International Inflammatory Bowel Disease Meeting

This meeting will be held on 10–11 February 2003 in Chester, UK. An international programme includes speakers from the USA, France, Italy, and the UK, and will cover clinical problems, pathogenesis, medical and surgical treatment. Registration details and programme from: Professor Jonathan Rhodes, Department of Medicine, University of Liverpool, Daulby Street, Liverpool L69 3GA, UK. Tel: +44 (0)151 706 3358; fax: +44 (0)151 706 5832; email: rhodesjm@liverpool.ac.uk

Surgery of the Foregut

This meeting will be held on 17–18 February 2003 in Florida, USA. Further information: Cleveland Clinic Florida, Office of CME, 2950 Cleveland Clinic Boulevard, Weston, FL 3331, USA. Tel: +1 954 659 5490; (toll free: +1 866 293 7866); fax: +1 954 659 5491; email: cme@ccf.org

38th EASL Annual Meeting

The European Association for the Study of the Liver will be holding its 38th annual meeting on 29 March–1 April 2003 in Istanbul, Turkey. Further information can be found on the website www.easl.ch/easl2003.

International Symposium on Viral Hepatitis and Liver Disease

This conference will take place on 6–10 April 2003 in Sydney, Australia. Further information: ISVHLD 2003 Congress Managers, GPO Box 128, Sydney NSW 2001, Australia. Tel: +61 9262 2277; fax: +61 9262 3135; email: isvhld@tourhosts.com.au; website: www.tourhosts.com.au/isvhld
Smoking and ulcer healing

A Duggan and N Rutherford

Gut 2003 52: 153
doi: 10.1136/gut.52.1.153

Updated information and services can be found at:
http://gut.bmj.com/content/52/1/153.1

These include:

References
This article cites 2 articles, 1 of which you can access for free at:
http://gut.bmj.com/content/52/1/153.1#BIBL

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/