inars to the Editor

Liver disease and pulmonary hypertension

EDITOR—I read with interest the leading article on hepatopulmonary syndromes (Gut 2000;46:1–4). The author describes explicitly the various associations between the liver and lung disorders. Several clinical studies and autopsy findings have demonstrated a 20% higher prevalence of pulmonary hypertension in patients with advanced liver disease and portal hypertension, the histological findings of which show features similar to those seen in pulmonary hypertension from other causes.1 2 However, the underlying mechanism(s) responsible for pulmonary hypertension in these patients is not known. It has been frequently hypothesised that increased circulating levels of noradrenaline (NA) or increased activity of u, adrenergic receptors in the pulmonary arteries can produce excessive pulmonary vasoconstrictor and proliferative responses leading to pulmonary hypertension.3

It is generally believed that pulmonary hypertension results from defective hepatic elimination of a vasoconstrictive agent produced by the splanchnic territory which reaches the pulmonary arteries through porto-systemic shunts.4 The mesenteric organs produce about 50% of the total NA present in the human body5 which is rapidly metabolised by liver parenchymal cells to vanillymandelic acid before it reaches the systemic circulation.6 Following hepatectomy, circulating levels of NA have been shown to be increased by up to 10-fold in experimental animals7 while patients with liver cirrhosis or those undergoing extracorporeal hepatic resection or liver transplantation have levels of circulating NA up to 2.6-fold greater.8 Increased pulmonary vascular resistance has often been observed during the anhepatic phase of liver transplantation9 while several studies have demonstrated that pulmonary hypertension occurs more frequently in histologically severe liver cirrhosis following liver transplantation.10 Formation of a portocaval shunt without liver cirrhosis has also been shown to produce severe pulmonary hypertension.11 It has been demonstrated recently that hepatopathy produces a sharp increase in pulmonary vascular resistance which correlates positively with pulmonary arterial NA levels.12 Defective hepatic metabolism by diseased liver parenchymal cells could greatly increase circulating levels of NA. The resulting portal hypertension and porto-systemic shunt also transfers large amounts of NA directly from the mesentery to the systemic and pulmonary circulation. High circulating levels of NA could then stimulate u, adrenergic receptors present in the pulmonary arteries to produce excessive pulmonary vasoconstrictor and proliferative responses leading to pulmonary hypertension. Increased NA levels could also explain the association of increased cardiac output noted in patients with porto-pulmonary hypertension.13 Antagonists or drugs that rapidly metabolise circulating lev-

UDCA, PBC, and biochemistry, what does normal mean?

EDITOR—We read the commentary by Lindor (Gut 2000;46:8) with great interest and would like to raise the following points.

Lindor is right that in a focal disease such as PBC, nine liver biopsies were needed from one session to another, while several studies have demonstrated that pulmonary hypertension developed in 4% and in 11% of the disease in 4% and in 11% of the patients.5 6 We believe this is interesting as it is improbable that a further increase in bile acid concentrations in serum and a shift from the more hydrophobic to a more hydrophilic bile acid pool could be responsible for a complete response to UDCA therapy. Further results are awaited.

Lindor speculates that in our study the high percentage of patients with early stage PBC could have been an artefact because there was no correlation between histological stage at entry and biochemical response. We started UDCA therapy for PBC in 1978/79. In that time we had 120 patients under constant supervision and over this period of 21 years only three patients have undergone liver transplantation and two have died as a result of late stage liver disease. We believe that our patients underwent regular liver biopsies and some even laparoscopy. That we have seen no more deaths or complications can only be explained by the fact that patients were in the early stages of the disease and that they were treated continuously with UDCA.

Lindor says that improvement in liver histology in our patients treated with UDCA (p<0.05) differs from the overall experience in other studies. However, patients were discriminated between incomplete and complete responders whereas in other trials complete and incomplete responders were evaluated together and compared with an untreated group.

In addition, Lindor is surprised that the histological progression reported in our series, even in incomplete responders, was slow. Based on modelling studies of untreated patients with PBC, he stated that substantial more patients developed histological progression. The difference between the studies cited by Lindor and ours is that we studied patients treated long term and not untreated patients, and it is well known that UDCA retards histological progression,1 as recently shown using the Markow model.

Our description of how the histological grading was performed was not sparse; it was presented carefully and in accordance with other studies. It is correct that the histological data are mentioned in a single sentence and are not tabulated or otherwise presented. But having been a pathologist myself, I am rather sceptical towards liver histology in a patchy disease. For example, in 1994 it was shown9 that in a focal disease such as PBC, nine liver biopsies were needed from one session to another, while several studies have demonstrated that pulmonary hypertension developed in 4% and in 11% of the patients. We believe our results are comparable. In our study, even in incomplete responders, histological progression was seen no more deaths or complications can only be explained by the fact that patients were in the early stages of the disease and that they were treated continuously with UDCA. Lindor speculates that in our study the high percentage of patients with early stage PBC could have been an artefact because there was no correlation between histological stage at entry and biochemical response. We started UDCA therapy for PBC in 1978/79. In that time we had 120 patients under constant supervision and over this period of 21 years only three patients have undergone liver transplantation and two have died as a result of late stage liver disease. We believe that our patients underwent regular liver biopsies and some even laparoscopy. That we have seen no more deaths or complications can only be explained by the fact that patients were in the early stages of the disease and that they were treated continuously with UDCA. Lindor speculates that in our study the high percentage of patients with early stage PBC could have been an artefact because there was no correlation between histological stage at entry and biochemical response. We started UDCA therapy for PBC in 1978/79. In that time we had 120 patients under constant supervision and over this period of 21 years only three patients have undergone liver transplantation and two have died as a result of late stage liver disease. We believe that our patients underwent regular liver biopsies and some even laparoscopy. That we have seen no more deaths or complications can only be explained by the fact that patients were in the early stages of the disease and that they were treated continuously with UDCA.
The most important findings in our study were that: (1) UDCA improved cholestatic indices in incomplete and full responders in a strictly parallel manner; (2) in incomplete responders, the curves levelled off after about 3–5 years and did not normalise; and (3) cholestatic indices in patients with alcoholic early stages of PBC allowed differentiation between responders and incomplete responders. This parallelism of the curves may indicate that UDCA influences mainly cholestasis and that other reactions are secondary. Therefore, more potent cholestatic compounds or a combination of various cholestasis and that other reactions could further improve results in incomplete responders. As stated previously, we are about to conclude such a study and the results seem to support our hypothesis.

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Endoscopic gastrin test and Helicobacter pylori infection

EDITOR.—In their recent article in Gut, Iijima and colleagues conclude that reduced acid secretion in gastric ulcer patients and gastric hypersecretion in duodenal ulcer patients were both normalised after H pylori eradication. We agree with the recovery of gastric secretary function in the former group of patients, who constantly bear chronic gastritis which improves greatly after disappearance of the germ, with subsequent restoration of gastric glandular tissue. However, we disagree with their conclusion regarding the latter group, because it is not supported by the experimental data they obtained. It is surprising that they found an increase in acid secretion (although not significant) in duodenal ulcer patients one month after eradication. This finding is difficult to explain, because basal gastrin levels were significantly reduced compared with those before eradication in the same patients, and others have found a rapid decrease of acid output in relation to the decline of serum gastrin.

The Japanese researchers state that this disparity may depend on the premature assessment of gastrin stimulated acid output (one month), because the same evaluation performed after seven months showed significantly decreased values compared with those before eradication. It must be pointed out, however, that there is a tremendous overlap between acid outputs measured before and after seven months of eradication, and those pertaining to H pylori negative controls.

Moreover, the rate of decrease of acid secretion after seven months was only 23% in their study. This reduction is very low and similar to the level of 16% seen after six months of eradication by Parente et al, who also acknowledge in their paper that this small percentage casts doubt on the unique role of H pylori in determining the augmented acid secretion typical of duodenal ulcer. Although the data obtained by Iijima et al and Parente et al after cure of H pylori infection were significantly different from those before eradication, we believe that statistical significance does not mean physiological relevance in this case.

Apart from the previously mentioned overlapping, some patients even show an increase in acid secretion after seven months, and others have found no change in maximal acid secretion 12 months after eradication of the bacterium. It is clear that the deregulation of gastric physiology in duodenal ulcer is caused by a combination of factors and H pylori is only one of them. In addition, it should not be forgotten that 20% of patients with duodenal ulcer have been shown to relapse despite ascertained H pylori eradication,1 and a high acid output has been found in patients with duodenal ulcer recurrence after the disappearance of H pylori. These findings seem to suggest that a genetic predisposition to secrete more acid is present at least in a subset of patients with duodenal ulcer recurrence independent of H pylori status. Therefore, overenthusiastic statements that eradication of H pylori is followed as a rule by normalisation of gastric acid output are deceiving and should be attenuated.

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Losartan and renal sodium handling

EDITOR.—We read with great interest the paper by Girgrah et al (Gut 2000;46:114–120). Their report suggests that the subtle sodium retention that is characteristic of precsisic cirrhosis is improved by administration of low dose losartan. This is despite the paradoxical observation of an angiotensin concentration that is significantly lower in patients compared with healthy volunteers (mean (SEM) patients 6 (2); controls 40 (10) pmol/l). Our results of angiotensin II measurements are at variance with those published by Girgrah et al and are illustrated in fig 1. Our studies suggest that there is a progressive increase in angiotensin II concentration with increasing severity of sodium retention. In fact, this increase in angiotensin II is evident before any measurable derangement in systemic haemodynamic characteristics. The values measured in healthy volunteers are also significantly higher than those reported in the literature. We are not sure if these differences are due to the measured values are the result of different patient populations, differences in the method of collection of the sample (Girgrah et al—EDTA and aprotinin; Newby et al and Helmy et al—0.5% trisodium EDTA) or different assay techniques (were the samples extracted prior to the radioimmunoassay?).

The authors hypothesise that the increase in renal sodium excretion observed after administration of losartan was possibly due to its effect on intrarenal angiotensin II secretion. If this was true then there should have been a significant increase in plasma angiotensin II concentrations after administration of losar-

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Figure 1 Measured angiotensin II concentrations in healthy volunteers (HV) and in patients with cirrhosis and varying degrees of severity of sodium retention (preascitic (PA) cirrhosis, diuretic responsive (DR) ascites, and refractory ascites). Our reference range for serum values reported by Girgrah et al are as follows: HV <1 pmol/l, PA <2 pmol/l, DR <10 pmol/l. For our reference range for angiotensin II levels are shown as mean (SEM) and *p<0.01 v preascitic cirrhosis and controls; †p<0.05 v preascitic cirrhosis and controls; ‡p<0.05 v controls, preascitic cirrhosis, and refractory ascites.

Letters, Book reviews, Notes
tan? Values of angiotensin II observed in this study are contradictory to expected values. Indeed, in patients with severe heart failure, mean values of 60–70 pg/ml are reported and a value >10 pg/ml in patients given angiotensin converting enzyme inhibitors is considered higher. These findings question the pathophysiological interpretation of the main results of the study by Girgrah et al. It is more likely that improvement in sodium excretion after administration of losartan was due to its effect on reducing portal pressure which in turn alters renal function through the hepato-renal axis.1,2

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Reply

Editor,—We thank Drs Jalan and Newby for their comments on our recent study (Gut 2000;46:114–120). We understand that our findings of decreased angiotensin II levels in preascitic cirrhotic patients compared with normals are at variance with their findings of elevated levels in such patients.1 Before we comment on this, we will first address their second point that our results in healthy volunteers are higher than those previously reported.1 On reviewing the literature, we noted that our values were within the same “ballpark” as the reported reference values of 20 (7 pg/ml) whereas those from the Edinburgh group (3.2 (0.3) pg/ml) are on the lower side. Furthermore, their angiotensin II levels in cirrhotic patients with ascites (238 (30) pg/ml) are several times higher than those reported in patients with severe heart failure.1 We believe the explanation for these disparate results in normals and patients is laboratory variation, which is why each investiga- tion needs its own reference values.

Concerning the differences between our findings and those of Helmy et al of increases in angiotensin II levels in preascitic patients, the Edinburgh group not surprisingly found an increase in plasma renin activity also. They acknowledge in their publications that this is at variance with much of the literature on the subject in which several studies found suppression of the renin-angiotensin-aldosterone system in preascitic patients in the supine position,1,1 the position the Edinburgh group used in their studies.1,1 In addition, their preascitic patients had normal levels of atrial natriuretic peptide, which is also at variance with much of the literature, as summarised in the review by Bernardi and colleagues.1,1 Hence how do we explain these differences? We cannot explain them in terms of the patients were on a diet of 150 mmol of sodium per day. However, we noted that a significant per- centage of their preascitic patients had primary biliary cirrhosis. These patients were cholestatic, giving rise to an unusually high mean serum bilirubin level (35 (12 mmol)) for a group of preascitic cirrhotics.1 This in turn may have contributed to some of their preascitic patients being classified as Child B whereas such patients are generally in the Child A category.1,1 These cholestatic pa- tients, with or without jaundice, also have elevated levels of serum bile acids which are vasodilators and could be partly responsible for the decreased effective blood volume in the jaundiced patients, even in the absence of cirrhosis.2 Relatively mild jaundice may also explain the reduced vascular responsiveness to angiotensin II found by the Edinburgh group.11 In general, we largely agree with much of the Edinburgh group’s findings. In particular, we agree with the increase in serum angio- tensin II levels seen in patients with liver dis- ease; with their findings after the TIPS procedure; and with the importance of liver function and portal hypertension in the pathogenesis of sodium retension in chronic liver disease. Therefore, we concur with their concluding remark that improvement in renal sodium handling found in preascitic cirrhotic patients after low dose losartan may well be due in part to the lowering effect of losartan on portal pressure.11

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Replication error phenotype in colorectal cancer

Editor,—The results presented in the article by Curran et al (2000;46:121–4) may have been different if the authors had classified DNA microsatellite instability status as stable (MSS), low (MSI-L) or high (MSI-H), as recommended by a National Cancer Institute sponsored Workshop.1 Of the 254 tumours, “RER+” group included both MSI-H and MSI-L cancers. The finding of bandshifts in two of eight dinucleotide markers is not specific for MSI-H cancers and will pick up a proportion of MSI-L cancers.2 Two of the three RER+ cancers with a K-ras mutation (study Nos 52 and 129) showed bandshifts at only two loci, were left unclassified, and were positive for nuclear p53. They would be interesting to know if these cancers are characterised by specific bandshifts at the mononucleotide markers BAT25, BAT26, or BAT40 (specific and sensitive for MSI-H) and/or show loss of expression of hMLH1.3 We expect these (and other cancers) will be found to be MSI-L. This would also explain the high frequency of p53 positivity, not seen by others.4 Their conclusions with respect to RER+ cancers regarding molecular profiles and prognostic significance only compound the confusion gener- ated by earlier studies that failed to draw the fundamental distinction between MSI-L and MSI-H.

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1 Curran AJ, Marr P, Bell SJ, et al. Characterisation of a subtype of colorectal cancer (study Nos 52 and 129) showed bandshifts at only two loci, were left unclassified, and were positive for nuclear p53. They would also explain the high frequency of p53 positivity, not seen by others.4 Their conclusions with respect to RER+ cancers regarding molecular profiles and prognostic significance only compound the confusion generated by earlier studies that failed to draw the fundamental distinction between MSI-L and MSI-H.

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Gut: first published as 10.1136/gut.47.4.595 on 1 October 2000. Downloaded from http://gut.bmj.com/ on October 2023 by guest. Protected by copyright.
EDITOR,—Jass has pointed out that the criteria we used to define microsatellite instability (MSI) status are not in accordance with the recommendations produced by the National Cancer Institute workshop on microsatellite instability.1 We would point out that the conclusion of our study and submission of our manuscript were contemporaneous with the publication of these recommendations. It is clear that the criteria we used may have resulted in some MSI-L cases being included in the RER+ cohort for the purpose of the analysis. Clearly, the best way to address this issue would be to reassign our RER+ cohort using a mononucleotide repeat marker, BAT-25 or BAT-26,2; however, sufficient clinical material is no longer available to us.

We based our analysis on eight dinucleotide repeat markers and defined tumours as RER+ if two or more markers (that is, 25%) exhibited allelic shifts.3 This analysis categorised 14% of tumours (22 of 159) as RER+. The NCI recommendations for analyses involving greater than five markers were that MSI-H would be defined as having allelic shifts in >30–40% of markers. This would suggest that our RER+ cohort must contain a number of MSI-L tumours but that, by the NCI criterion, the majority are likely to have been MSI-H. Therefore, while we fully concede that our study included a number of MSI-L tumours in the RER+ category, we believe that this number was small (in the context of a total patient cohort of 159) and does not completely invalidate our conclusions. Furthermore, as we have pointed out in our paper, we believe that our decision to include only patients who underwent potentially curative surgery for cancers which had penetrated beyond the bowel wall but which had not breached the peritoneal surface, spread to other organs or metastasised to lymph nodes or distant sites at the time of operation (T3, N0, M0), lends significant strength to our study in avoiding potentially confounding effects of tumour stage on microsatellite instability or other parameters.

B CURRAN K LENEHAN H MULHOLLAND O TIGHE M A BENNETT E ECLAY D P O'DONOGHUE M LEADER T D CROKE

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2 Parsons R, Myeroff LL, Liu B, et al. Microsatellite instability and mutations of the transform-
**BOOK REVIEWS**


A booklet a little larger than the size of a two column review may seem unimportant. But this is an exception. This publication is for patients with ulcerative colitis and some sources of information should be the concern of gastroenterologists. It has been written by Andrew Robinson, whose self management programme for patients with colitis leads to fewer outpatient visits, more rapid treatment of relapse, and improved patient satisfaction (1997, A12), and Anne Kennedy, a research fellow in primary care. They have been assisted by a professional writer and sensibly had the guide endorsed by the Plain English Campaign.

The guide consists of two booklets in a single plastic folder. Part One includes an overview of ulcerative colitis, tests, treatment and surgery. Part Two is an individual patient record. There is much to be commended, with detailed information helpfully summarised in coloured boxes (“Things to Remember”), or treatment options discussed (“Your Choice”) and anecdotes from patients that give a personal appeal. Clinical views and opinions are, on the whole, well balanced, and I could see this guide being a valuable contribution to patient information. Faults, however, qualify this commendation. The surgical subsection on ileocolectal anastomosis for ulcerative colitis is wholly inappropriate and there is confusion in terminology in the section on pouch surgery. Factual errors (such as a “2% risk” of ulcerative colitis in offspring, or “5-sulf mercaptopurine”) and statements such as “immunosuppressants may make your baby very small and can lead to abnormalities” are simply misleading. Indeed the whole section on pregnancy is poor, with two anecdotes from patients advising cutting out. A mention about the dilemmas of coexisting substances from mother to infant. Until this has been reviewed and possibly rewritten, the whole section on pregnancy is poor, with factual errors (such as a “2% risk” of ulcerative colitis in offspring, or “5-sulf mercaptopurine”) and statements such as “immunosuppressants may make your baby very small and can lead to abnormalities” are simply misleading. Indeed the whole section on pregnancy is poor, with two anecdotes from patients advising cutting out. A mention about the dilemmas of coexisting substances from mother to infant.

When I was a fellow with Allan Walker fifteen years ago, gut development was a topic of interest to a handful of researchers worldwide. A classic review by Grand, Watkins and Torti published in Gastroenterology in 1976, and Koldovsky’s monograph Development of the Functions of the Small Intestine in Man: Fetus and Man in 1969, brought together much of what was then known about the ontogeny of the human gut. Developmental biologists were beginning to recognise the opportunities, offered by the rapidly differentiating organ to understand the interactions of genetic endowment and environmental influences in early life. The focus of much research was on the process of adaptation to milk feeding. With the survival of ever more preterm infants the function of the immature gut and its capacity to deal with enteral feeds prematurely, were questions of increasing practical concern.

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I had the grand idea at that time to produce a short book bringing the field all together. But I quickly realised that not only was growing too fast, but that a full understanding of gut development and function also required an understanding of the composition and properties of human milk and the metabolism of the newborn. The developing gastrointestinal tract is inextricably linked to the developing mammary gland are complementary organs, jointly involved in the transfer of nutrients and other substances from mother to infant. Until weaning, the neonate is an extra-gestation fetus, and breast and gut are analogous to the uterine–placental interface.

This book goes a long way to recognising this. Each chapter (essentially a stand alone review) is written by a leading figure or group expert in its field. Together they cover the major aspects of gut development and function but, apart from a short preface, there is no overview or attempt to synthesise the book’s contents. It would be impossible for one author to write this book now. The impact of molecular biology has moved the subject from an essentially descriptive science, with some experimental work in vivo, to the level of the cell and gene. This has shifted it away from the womb, breast, or incubator and into the laboratory. This book is a valuable starting point for students or researchers wishing to get up to date with the basic biology of human gut development, but it will be of little interest to the practising neonatologist struggling to define rational approaches to feeding the preterm neonate.


Medicine is fast becoming a major branch of biology, concerned with the application, often experimentally, of novel therapies based on insights and new understanding of biological processes. However, the biological sciences are advancing so rapidly, and manipulation of genes within cells, including those of the embryo is possible, the gap between the world of modern biomedical science and the patient is widening rather than narrowing.

The last century saw the integration of medicine and science, and a determination to base the practice of the former on the latter. At the beginning of this century attempts are being made to define a core of knowledge, skills, and ideas to teach our medical students. The wide scope of what we currently regard as the province of medicine now includes sociology, psychology, epidemiology, etc, and the basic sciences have been squeezed. We may be making a mistake in failing to equip medical students and young doctors with a firm understanding of the “new biology”—embracing genetics, molecular and developmental biology. This book deals with these things and, although its subject is a small part of the totality of human biology, it is dealt with in depth by recognized leaders. Ian Sanderson and Allan Walker must be congratulated for bringing their research together.


To paraphrase Mark Twain, reports of the impending demise of the print media have been greatly exaggerated—a trainee can still spend hours browsing new editions in a medical bookshop and, usually during frantic preparation for higher exams while fulfilling DSM-IV criteria for anxiety disorder, part with large sums of money on illustrated texts. There also seems to have been a small explosion of abridged versions of textbooks and specialty handbooks, although some of these “handbooks” can weigh in at more than 500 pages, and entail some serious fitness training if carried around in a coat pocket.

Almost qualifying for the cruiserweight division at just over 200 pages, A Colour Handbook of Gastroenterology provides a concise, richly illustrated summary of clinical gastroenterology. Apart from oesophageal varices and ascites, hepato logical conditions are not included. The book contains about 90 subjects organised into 10 colour coded anatomical sections. Each section starts with a short discussion of the relevant physiology, as well as techniques for imaging, and functional assessment. Most major areas of gastroenterology are covered, although the level of detail is sometimes a little uneven. For example, some pages are devoted to varices.
disorders of the small bowel and colon, but the less visually glamorous conditions of constipation and irritable bowel syndrome are relegated to a single page or less. The text on disease management is usually limited to a few lines on each subject, so that a trainee will still need to consult more detailed references when making treatment decisions. There is also a paucity of newer imaging techniques, including magnetic resonance imaging and endoscopic ultrasonography, two technologies that are beginning to revolutionise our approach to patients with suspected gastrointestinal disorders.

Perhaps the main attraction of this book for the visually inclined, busy trainee is that the text is structured, succinct, and richly illustrated with over 300 high quality radiographs, colour photographs, and tables. Given the increasing availability of electronic textbooks and medical images, one wonders about the future of such handbooks—although, unlike any other medical text on my computer or bookshelf, it was certainly easy to read from cover to cover. The preface states that it is directed towards junior doctors who are preparing for higher qualifications in gastroenterology and general medicine, but it will also appeal to financially solvent medical students who are keen to learn more about gastroenterology.

S P PEREIRA

NOTES

Sir Frances Avery Jones British Society of Gastroenterology Research Award 2001

Applications are invited by the Education Committee of the British Society of Gastroenterology who will recommend to Council the recipient of the 2001 Award. Applications (TWENTY COPIES) should include:

A manuscript (2 A4 pages ONLY) describing the work conducted
A bibliography of relevant personal publications
An outline of the proposed content of the lecture, including title
A written statement confirming that all or a substantial part of the work has been personally conducted in the UK or Eire.

An applicant need not be a member of the Society. The recipient will be required to deliver a 20 minute lecture at the Annual meeting of the Society in Glasgow in March 2001. Applications (TEN COPIES) should be made to the Endoscopy Section Secretary, British Society of Gastroenterology, 3 St Andrews Place, London NW1 4LB by 1 December 2000.

Joint Meeting of Oesophageal Section of the BSG and Association of Upper GI Surgeons

There will be a joint meeting of the Oesophageal Section of the British Society of Gastroenterology and the Association of Upper GI Surgeons exploring some important issues in oesophageal disease at the Royal College of Surgeons of England, Lincoln’s Inn Fields, London WC2 on Wednesday 1 November 2000. The meeting will take the form of four debates on:

1. The place of chemotherapy in the management of cancer of the oesophagus
2. The appropriate management of high grade dysplasia
3. Identifying the role of anti-reflux surgery in the current management of gastrooesophageal reflux disease and
4. The relevance of helicobacter pyloridis in oesophageal disease.

Further information from: WJ Owen, Hon Secretary, Oesophageal Section of the BSG, Suite 406 Emblem House, London Bridge Hospital, 27 Tooley Street, London SE1. Tel: (0)20 7403 3814; fax: (0)20 7403 3814.

Gluten Sensitivity Symposium

The Gluten Sensitivity Symposium, sponsored by SHS International, will be held at the Natural History Museum, London, on Friday 20 October 2000. Speakers include Professor Paul Ciclitira, Dr Tony Ellis, Dr Geoff Holmes, Professor Markku Maki, Dr Mario Hadijavansilou, Professor Lionel Fry, Dr Gerd Michaelsson and Professor Tom MacDonald. Further information: Debbie Jones at SHS International. Tel: +44 (0)151 228 1992; email: djones@shsint.co.uk.

Food Allergy and the Gut

The Allergy Research Foundation presents Food Allergy and the Gut, to be held at the Royal Society of Medicine, London on 29 November 2000. Further information: Philip N Goddard, Executive Secretary, The Allergy Research Foundation, PO Box 18, Aylesbury, Bucks HP22 4XJ, UK. Tel & fax: +44 (0)1296 655818.

13th European Intensive Course of Digestive Endoscopy

This course will be held in Strasbourg, France on 18 and 19 December 2000. Further information from Professor G Gay, Service de Médecine Interne J, Hôpital de Brabois, Allée du Morvan, 54511 Vandœuvre-lès-Nancy Cedex, France. Tel & fax: +33 (0)3 85 13 55 49.

Joint Meeting of the American Pancreatic Association and the International Association of Pancreatologists

This meeting will be held in Chicago, Illinois, USA on 1–5 November 2000. Symposia, posters, scientific sessions, “Pancreatology at the Millennium”. Further information: Peter A Banks, Brigham and Women’s Hospital, 75 Francis Street, Boston, MA 02115, USA. Tel: +1 617 732 6747; fax: +1 617 566 0338.

36th Annual Meeting of the European Association for the Study of the Liver (EASL)

This meeting will be held in Prague, Czech Republic on 18–22 April 2001. Abstract deadline: 27 November 2000. EASL will offer 10 travel bursaries to selected young investigators and 30 to Eastern European, pending on submission of an abstract. In addition, first authors under 35 years of age, and in training, who submit abstracts will have free registration. Further information: EASL Liaison Bureau, c/o Kenes International, 17 rue du Cendrier, PO Box 1726, CH-1211 Geneva, Switzerland. Tel: +41 22 1211 965; fax: +41 22 1211 965; email: info@easl.ch; website: www.easl.com.

15th International Workshop on Therapeutic Endoscopy

This workshop will be held in Hong Kong on 5–7 December 2000. Further information: Miss Claudia Mak, Endoscopy Centre, Prince of Wales Hospital, Shatin, N.T., Hong Kong. Tel: +852 2632 2233; fax: +852 2632 0075; email: info@hksde.org.