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 However, the underlying mechanism(s) responsible for pulmonary hypertension in these patients is not known. It has been recently hypothesised that increased circulating levels of noradrenaline (NA) or increased activity of α1 adrenergic receptors in the pulmonary arteries can produce excessive pulmonary vasoconstrictor and proliferative responses leading to pulmonary hypertension.2

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 a portal-systemic shunt.3 The mesenteric organs produce about 50% of the total NA present in the human body which is rapidly metabolised by liver parenchymal cells to vanillylmandelic acid before it reaches the systemic circulation.4 Following hepatectomy, circulating levels of NA have been shown to be increased by up to 10-fold in experimental animals5 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.6 Increased pulmonary vasculature resistance has often been observed during the anhepatic phase of liver transplantation7 while several studies have demonstrated that pulmonary hypertension often responds to treatment in patients with liver cirrhosis following liver transplantation.8 Formation of a portocaval shunt without liver cirrhosis has also been shown to produce severe pulmonary hypertension.9 It has been demonstrated that in patients with liver cirrhosis who undergo hepatectomy there is a significant increase in pulmonary vascular resistance which correlates positively with pulmonary arterial NA levels.10 Deficiency in histaminotropic activity by diseased liver parenchymal cells could greatly increase circulating levels of NA. The resulting portal hypertension and portal-systemic shunt also transfers large amounts of NA directly from the mesenteric bed to the systemic and pulmonary circulation. High circulating levels of NA could then stimulate α1 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 portal-pulmonary hypertension,11 Antagonists or drugs that rapidly metabolise circulating levels of NA could therefore prevent the development of pulmonary hypertension in patients with advanced liver disease and portal hypertension.

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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 says that improvement in liver histology in our patients treated with UDCA (p<0.05) differs from the overall experience in other studies. However, the results are not sufficiently 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 substantially 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 patients with PBC. For example, in 1994 it was shown7 that in a focal disease such as PBC, nine liver biopsies were needed from one session to warrant a definitive histological diagnosis. Because of this, it is not possible for ethical reasons, histological findings should not be overinterpreted. Clinical data, development of complications, outcome, etc, are more relevant.

The most important objection of Lindor is the question of the relationship between normalization of liver function tests and clinically relevant findings. This is in contrast with a statement by Lindor himself (personal communication, November 9, 1999; 5th Annual Meeting, AASLD, Dallas, Texas) where he told us that in his incomplete responders the disease progressed in 38% of patients and in full responders in only 5%. We believe our results are comparable. In our series, Lindor, in full responders we found progression of the disease in 4% and in 11% of incomplete responders; in incomplete responders progression occasionally took place not only from one stage to the next, but to the next but one stage. As patient numbers were small in our study, we did not give percentage values. Hence it is clear from our results the significance of normalisation of liver function tests.


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LETTERS TO THE EDITOR

Gut 2000;47:595–600 595

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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 anicteric early stages of PBC allowed di
cholestatic indices in patients with anicteric
3–5 years and did not normalise; and (3)
Helicobacter pylori
Endoscopic gastrin test and
hypothesis.
previously, we are about to conclude such a
results in incomplete responders. As stated
compounds or a combination of various
parallelism of the curves may
strictly parallel manner; (2) in incomplete
indices in incomplete and full responders in a
levelled o
responders, the curves levelled o
percentages 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 signifi-
cance does not mean physiological relevance in
this case.
Apart from the previously mentioned over-
lapping, some patients even show an increase in
acid secretion after seven months, and oth-
ers have found no change in maximal acid
secretion 12 months after eradication of the bac-
eterium. 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
described H pylori eradication, and a high
acid output has been found in patients with
duodenal ulcer recurrence after the disap-
pearance 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, regardless of the presence of H pus/tors at least in a subset of patients with
duodenal ulcer, regardless of the presence 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
preascitic cirrhosis is improved by administra-
tion of low dose losartan. This is despite
the paradoxical observation of an angiotensin
centration 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 meas-
urements are at variance with those published
by Girgrah et al and are summarised in fig
1.† Our studies suggest that there is a
progressive increase in angiotensin II concen-
trations with increasing severity of sodium
retention. In fact, this increase in angiotensin II is evident before any measurable derange-
ment in systemic haemodynamic characteris-
tics. The measured values in healthy
volunteers are also significantly higher than those reported in the literature. We
are not sure if these differences in 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 vol%alfaf 0.45% O-
phenanthroline and 1% disodium EDTA),
or different assay techniques (were the
samples extracted prior to the radioimmuno-
assay)?
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 secre-
tion. If this was true then there should have been a significant increase in plasma angiotensin II concentrations after administration of losar-

Endoscopic gastrin test and Helicobacter pylori infection

Endoscopic gastrin test and Helicobacter pylori infection

ENDOSCOPIC GASTRIN TEST AND HELICOBACTER PYLORI INFECTIO

EDITORS.—In their recent article in Gut, Iijima and colleagues concluded that reduced acid secretion in gastric ulcer patients and gastric acid hypersecretion in duodenal ulcer patients were both normalised after H pylori eradica-
tion. We agree with the recovery of gastric secretory 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 surpris-
ing that they found an increase in acid secretion (although not significant) in duode-
nal 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 outputs 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

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1994;29(suppl 204):40–6.

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 results differ from those reported by Girgrah et al in pmol/l; 1 pmol/l is approximately equal to 1 pmol/l, taking the molecular weight of angiotensin II as 1046.2. ** p<0.01 v controls; †p<0.05 v preascitic cirrhosis and controls; 2p<0.05 v controls, preascitic cirrhosis and refractory ascites.

www.gutjnl.com
Dear Dr R Jalan,

I correspond to: Dr R Jalan.

E-mail r.jalan@ucl.ac.uk


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.1,3 Before we comment on this, we will first address their second result that our findings 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 low 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. We believe the explanation for these disparate results in normals and patients is laboratory variation, which is why each investigation 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 as well. 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 renin and angiotensin-II levels, which is also at variance with much of the literature, as summarised in the review by Bernardi and colleagues.1,3 Hence how do we explain these differences? We cannot explain them in terms of decreased sodium intake as their patients were on a diet of 150 mmol of sodium per day. However, we noted that a significant percentage of their preascitic patients had primary biliary cirrhosis. These patients were cholestatic, giving rise to an unusually high mean serum bilirubin level (35 ± 12 µmol/l) for a group of preascitic cirrhotics. 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.1,3 These cholestatic patients, 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. Relatively mild jaundice may also explain the reduced vascular responsiveness to angiotensin II found by the Edinburgh group.1,3

In general, we largely agree with much of the Edinburgh group’s findings. In particular, we agree with the increase in serum angiotensin II levels and decrease in the percentage of decreases in sodium intake in preascitic cirrhotic patients after low dose losartan may well be due in part to the blunting effect of losartan on portal pressure.12

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Replication error phenotype in colorectal cancer

EDITOR,—The results presented in the article by Curran et al (Gut 2000;46:32–4) may have been different if the authors had classified DNA microsatellite instability status as stable (MS), low (MS-L) or high (MS-H), as recommended by a National Cancer Institute sponsored workshop. “RER+” group included both MS-H and MS-L cancers. The finding of bandshifts in two of eight dinucleotide markers is not specific for RER+ cancers and will pick up a proportion of MSI-L cases.1,2 Two of the three RER+ cancers with a K-ras mutation (study Nos 52 and 129) showed bandshifts at only two loci, were left sided, and were positive for nuclear p53. It would be interesting to know if these cancers are the same as those with 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 generated by earlier studies that failed to draw the fundamental distinction between MSI-L and MSI-H.


Fibrosing colonopathy in an adult caused by over use of pancreatic enzyme supplements

EDITORS,—We read with interest the report by Bansi and colleagues (Gut 2000;46:283–285) describing fibrosing colonopathy secondary to high dose pancreatic enzyme therapy in an adult patient. Some details of the patient’s history—chronic diarrhoea, occasional night-time diarrhea, and a distended abdomen—may have resulted in some MSI-L cases being misclassified. Confounding effects of tumour stage on metastatic spread and thezonosis, which is still a matter of discussion. We would point 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 It is not clear that the criteria we used may have resulted in some MSI-L cases being classified as RER+. 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 readily 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 that our study included a number of MSI-L tumours and other parameters.

Correspondence to: Dr D T Croke.

Reply

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 not 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 reassess our RER+ cohort using a mononucleotide repeat marker, BAT-25 or BAT-26; 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.1 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 readily 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, Mo), lends significant strength to our study in avoiding potentially confounding effects of tumour stage on microsatellite instability or other parameters.

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2 Parsons R, Myeroff LL, Liu B, et al. Microsatellite instability and mutations of the transform-
monitoring, would have been helpful—bearing in mind that treatment with azathioprine often extends for several years. It is this sort of detail, along with the errors in the main text, that gives an impression of clinical inexperience.

Nevertheless, these points are correctable and if asked by a patient, I would broadly recommend the guide. There is nothing else like it on the market and it gives far more useful information than can be readily gleaned from the internet or from pharmaceutical sponsored freebies. I hope that the authors will stand by their commitment to update the guide every two years. This means that they would be working on the 2001 edition now.

S P L TRAVIS


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 Developing Gut in Manne-Fuhs 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 and environmental influences in the 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.

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 it 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 and 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-gestate 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, if all the biological sciences are advancing so rapidly, and manipulation of genes within cells, including those of the embryo is possible, the gap between the worlds of medicine and developmental biology 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 there is a desire to be able 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”-embryology, genetics, molecular medicine, 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 recognised leaders. Ian Sanderson and Allan Walker must be congratulated for bringing their research together.

Development of the Gastrointestinal Tract is also provided as a CD-ROM, but this offers little more than the facility to read it on screen. It has no search tools, nor is it possible to cut and paste sections (for those wishing to produce a review article overnight). However, the open nature of the chapters will abolish the tedious of photocopying, and will also preserve the spine of this handsome and well produced book.

L WEAVER


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, hepatological 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, seven pages are devoted to varices...
An outline of the proposed content of the
A bibliography of relevant personal publi-
(TWENTY COPIES) should include:
Applications are invited by the Endoscopy
Committee of the British Society of Gastro-
enterology 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 manage-
ment 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 gastrooesopha-
geal 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 meeting,
sponsored by EASL International, will be held
at the National History Museum, London, on
Friday 20 October 2000. Speakers include
Professor Paul Ciclitira, Dr Tony Ellis, Dr
Geoff Holmes, Professor Markku Maki, Dr
Marios Hadjivassiliou, Professor Lionel Fry,
Dr Gerd Michalosch 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 Vandoeuv-
lès-Nancy Cedex, France. Tel & fax: +33 (0)3
81 15 35 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. Sym-
posia, posters, scientific sessions, “Pancrea-
tology at the Millennium”. Further infor-
mation: Peter A Banks, Brigham and
Women’s Hospital, 75 Francis Street, Bos-
ton, 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 Inter-
national, 17 rue du Cendrier, PO Box 1726,
CH-1211 Geneva, Switzerland. Tel: +41 22
908 0488; fax: +41 22 732 2850; 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 2635
0075; email: info@hksde.org

Sir Frances Avery Jones British Society
of Gastroenterology Research Award
2001
Applications are invited by the Education
Committee of the British Society of Gastro-
enterology who will recommend to Council
the recipient of the 2001 Award. Applications
(TEN COPIES) should include:
A manuscript (2 A4 pages ONLY)
describing the work conducted
A bibliography of relevant personal publi-
cations
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 Honorary 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 Oesopha-
geal Section of the British Society of Gastro-
enterology and the Association of Upper GI
Surgeons on 3–7 December 2000, at the Royal
College of Surgeons of England, Lincoln’s Inn
Fields, London WC2. The conference will focus
on 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 technolo-
gies that are beginning to revolutionise our
approach to patients with suspected gastro-
intestinal disorders.
Perhaps the main attraction of this book for
the visually inclined, busy trainee is that the
text is structured, succinct, and richly illus-
trated 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 gastro-
enterology and general medicine, but it will
also appeal to financially solvent medical stu-
dents who are keen to learn more about
gastroenterology.

S P PEREIRA

NOTES

Further information from: WJ Owen, Hon
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Suite 406 Emblem House, London Bridge
Hospital, 27 Tooley Street, London SE1. Tel:
(0)20 7403 3814; fax: (0)20 7403 3814.
Fibrosing colonopathy in an adult caused by over use of pancreatic enzyme supplements

M HÄUSLER, G HEIMANN, R MEILICKE and S BIESTERFELD

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**Notes**