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.

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 α1 adrenergic receptors in the pulmonary arteries can produce excessive pulmonary vasoconstrictor and proliferative responses leading to pulmonary hypertension.

It is generally believed that pulmonary hypertension results from defective hepatic elimination of a vasoconstrictive agent produced in hepatic and splanchic territories which reach the pulmonary arteries through porto-systemic shunts. 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.

Following hepatectomy, circulating levels of NA have been shown to be increased by up to 10-fold in experimental animals 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. Increased pulmonary vascular resistance has often been observed during the anhepatic phase of liver transplantation while several studies have demonstrated that pulmonary hypertension occurs relatively early in histologically defined liver cirrhosis following liver transplantation. Formation of a portocaval shunt without liver cirrhosis has also been shown to produce severe pulmonary hypertension. It has been demonstrated recently that hepatectomy produces a sharp increase in pulmonary vascular resistance which correlates positively with pulmonary arterial NA levels.

Defective hepatic metabolism by diseased liver parenchymal cells could greatly increase circulating levels of NA. The resulting portal hypertension and porto-systemic shunt also transfer large amounts of NA directly from the mesenteric bed to the systemic and pulmonary circulation. Higher circulating levels of NA could then stimulate α1 adrenergic receptors 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.

Antagonists or drugs that rapidly metabolise circulating lev-}

els 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 states in his commentary that in our study patients with primary biliary cirrhosis (PBC) who initially had less abnormal liver function tests responded more favourably to ursodeoxycholic acid (UDCA) than those who had initially greater abnormal liver function values. We believe this is interesting as it is known that patients with lower abnormal liver function tests respond less favourably (for example, chronic autoimmune hepatitis to treatment with glucocorticoids) and that values do not decrease in a linear manner. Furthermore, it is well known that UDCA in PBC does not cause normalisation of liver function tests in most patients, and to date there has been no extensive examination of full and incomplete responders. Only in one study was this area addressed but few liver function tests responded more favourably to UDCA than those who had initially greater abnormal liver function values. We believe this is interesting as it is known that patients with lower abnormal liver function tests respond less favourably (for example, chronic autoimmune hepatitis to treatment with glucocorticoids) and that values do not decrease in a linear manner. Furthermore, it is well known that UDCA in PBC does not cause normalisation of liver function tests in most patients, and to date there has been no extensive examination of full and incomplete responders. Only in one study was this area addressed but few liver function tests responded more favourably to UDCA than those who had initially greater abnormal liver function values.

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Endoscopic gastrin test and Helicobacter pylori infection

EDITOR.—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 eradication. 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 secretion 12 months after eradication of the bacterium. 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 duodenal ulcer patients even 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 themselves acknowledged in their paper that this small percentage casts doubt on the unique role of H pylori in determining the augmented acid secretion of duodenal ulcer. Although the rate of increase of acid secretion in duodenal ulcer patients after eradication was higher than that reported in the literature.

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. As stated previously, we are about to conclude such a study and the results seem to support our hypothesis.

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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 substitution sodium retention that is characteristic of preasitic cirrhosis is improved by administration of losartan. 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 apetin; Newby et al and Helmy et al—0.5 ml or 0.45% O-phenanthroline and 1% disodium EDTA), or different assay techniques (were the samples extracted prior to the radioimmuno-assay)?

The authors hypothesis that the increase in renal sodium excretion observed after administration of losartan was possibly due to its effect on intrarenal angiotensin II secretion. In this was true because the results showed a significant increase in plasma angiotensin II concentrations after administration of losar-
tand? 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 high. 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.13

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vascular cutaneous blood flow in patients with cirrhosis: role of the renin-angiotensin and sympathetic nerv-


2 Hsu C, Jalan R, Newby DE, et al. Role of angio-


5 Sweedberg K, Emanuel P, Kjekshus J, et al for the CONSENSUS Trial Group. Hormone regulat-

6 Schneider AW, Kalk JF, Klein CP. Effect of losar-

7 Lang F, Tschernko E, Schulze E, et al. Hemo-


8 Jalan R, Forrest EH, Redhead DN, et al. Reduc-

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 inves-
tigation 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 the position the Edinburgh group used in their studies.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.14 Hence how do we explain these differences? We cannot explain them in terms of changes in plasma renin activity since their 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 cholangitis, giving rise to an unusually high mean serum bilirubin level (35 (12) µmol/l) 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 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.16 Relatively mild jaundice may also explain the reduced vascular responsiveness to angiotensin II found by the Edinburgh group.17

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 in preascitic liver disease; with their findings after the TIPS procedure; and with the importance of liver function and portal hypertension in the pathogenesis of sodium retention 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 blunting effect of losartan on portal pressure.18

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6 Sellers L, Shore AC, Wilkinson R. Sodium stat-
tus and the renin-angiotensin-aldosterone sys-


Repliation error phenotype in colorectal cancer

EDITOR.—The results presented in the article by Curran et al (Gut 2000;46:167–74) may have been different if the authors had classified DNA microsatellite instability status as stable (MSS), low (MSL-I) or high (MSL-H), as recommended by a National Cancer Institute sponsored workshop.1 The “RER+” group included both MSL-H and MSL-I cancers. The finding of bandshifts in two of eight dinucleotide markers is not specific for MSL-H cancers and will pick up a proportion of MSL-I cases.2 Two of the three RER+ cancers with a K-r mutation (study Nos 52 and 129) showed bandshifts at only two loci, were left sided, and were posi-
tive for nuclear p53. It would be interesting to know if these cancers are related to a distinct group of bandshifts at the mononucleotide markers BAT25, BAT26, or BAT40 (specific and sensitive for MSL-H) and/or show loss of expres-
sion of hMLH1.2 We expect these (and other cancers) will be found to be MSL-I. This would also explain the high frequency of p53 positivity, not seen by others.3 Their conclusions with respect to RER+ cancers regarding molecular profiles and prognostic signifi-
cance only compound the confusion gener-
aled by earlier studies that failed to draw the fundamental distinction between MSL-I and MSL-H.

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

Editor,—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—cholangitis, pancreatitis, and pancreatic insufficiency—are strikingly similar to symptoms displayed by our adult patient with cystic fibrosis and fibrosing colonopathy described previously.1 In this patient with cystic fibrosis, chronic cholangitis and cholecystitis required repeated endoscopic retrograde cholangiopancreatography, and severe pancreatic insufficiency was the reason for high dose pancreatic enzyme supplementation. Bansi et al assumed that their patient was not suffering from cystic fibrosis. As previously discussed in the commentary by Dodge in the same issue, negative results after even extensive mutation analysis of the cystic fibrosis transmembrane regulator gene cannot rule out cystic fibrosis. Furthermore, as outlined by the Cystic Fibrosis Foundation Consensus Panel,1 sweat testing is the standard test for diagnosis of cystic fibrosis. In patients with typical clinical symptoms but normal or borderline sweat chloride concentrations and normal genetic findings, nasal potential difference measurements should be performed.1 The clinical symptoms of the patient described by Bansi et al are highly indicative of cystic fibrosis with exclusive involvement of the gastrointestinal tract. Moreover, the histopathology of the pancreateic tissue, chronic cholangitis, cystic fibrosis and atrophy, is also typical of cystic fibrosis, as are frequent bowel actions.1 Proof of fibrosing colonopathy in a patient not suffering from cystic fibrosis may contribute considerably to a better understanding of the pathogenesis of fibrosing colonopathy which is still a matter of discussion.1,1 It would underline the aetiological impact of toxic effects of high dose pancreatic enzyme supplementation but caution against overestimating the contribution of factors possibly related to the cystic fibrosis transmembrane regulator gene mutation, such as increased intestinal absorption.1 We would therefore be interested in the patient’s sweat chloride concentration and, if normal, in the result of nasal potential difference measurements. This paper strongly advocates well thought out supplementation of pancreatic enzymes in adults who, like infants and children, are at risk of developing fibrosing colonopathy.
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 such 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 worldwide, 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 colour boxes ("Things to Remember").

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 Mammals 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 rapid and 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.

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 lactating 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, most of the biological sciences are advancing so rapidly, and manipulation of genes within cells, including those of the embryo is possible, the gap between the word of anatomy and the biological sciences 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 turn of the century it seemed possible 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 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. I could see this guide being a valuable supplement to clinical and basic science teaching for students and for patients with ulcerative colitis and such gastrointestinal conditions as irritable bowel syndrome or the implications of the 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.

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. The book contains about 90 subjects organised into 10 colour coded anatomical sections. Each section starts with a short discussion of the relevant anatomy, 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, single pages are devoted to varices and ascites, hepatological conditions were beginning to recognise the opportunities offered by the rapid and 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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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
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 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 Hadjivassiliou, Professor Lionel Fry, Dr Gerd Michaelson and Professor Tom MacDonald. Further information: Debbie Jones at SIS International. Tel: +44 (0)151 228 1992; email: djones@sishint.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 83 15 35 49.

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

This meeting will be held in Chicago, Illinois, USA on 1–3 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, The British Liver Foundation, PO Box 18, Aylesbury, Bucks HP22 4XJ, UK. Tel: +44 22 908 048; fax: +44 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 2335; fax: +852 2635 0075; email: info@hkde.org