Liver disease and pulmonary hypertension

Editor,—I read with interest the leading article on hepatoportal 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.1-4 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 adrenaline receptors in the pulmonary arteries can produce excessive pulmonary vasoconstrictor and proliferative responses leading to pulmonary hypertension.5

It is generally believed that pulmonary hypertension results from defective hepatic elimination of a vasoconstrictive agent produced by the liver parenchymal cells which reaches the pulmonary arteries through portal-systemic shunts.6 The mesenteric organs produce about 50% of the total NA present in the human body7 which is rapidly metabolised by liver parenchymal cells to vanillylmandelic acid before it reaches the systemic circulation.8 Following hepatectomy, circulating levels of NA have been shown to be increased by up to 10-fold in experimental animals9 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.10 Increased pulmonary vascular resistance has often been observed during the anhepatic phase of liver transplantation11 while several studies have demonstrated that pulmonary hypertension occurs relatively frequently in patients with liver cirrhosis following liver transplantation.12 Formation of a portocaval shunt without liver cirrhosis has also been shown to produce severe pulmonary hypertension.13 It has been demonstrated that hepatectomy produces a sharp increase in pulmonary vascular resistance which correlates positively with pulmonary arterial NA levels.14 Deficiency in histaminergic mechanism 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 pulmonary circulation. High circulating levels of NA or adrenaline receptors in the pulmonary arteries could greatly increase the prevalence of pulmonary hypertension in patients with advanced liver disease and portal hypertension.15 Antagonists or drugs that rapidly metabolise circulating lev-

ds 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 that in our study1 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 test results 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 parameters were studied and there was only a short follow-up period.

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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) cholestasis was only present in patients with anicteric 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 choleretic compounds or a combination of various choleretic substances could further improve responses 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 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 gastric glandular tissue.2 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 gastric levels were significantly reduced compared with those before eradication in the same patients, and others have found a rapid decrease in acid output in relation to the decline of serum gastrin.3 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 pepsinogen, and gastric emptying of liquids over six months from eradication of Helicobacter pylori on duodenal ulcer patients. Gut 1995;37:310–5.


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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 subdiuretic sodium retention that is characteristic of preascitic 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 any comparison is fig 1.8 Our studies suggest that there is a progressive increase in angiotensin II concentrations with increasing severity of sodium retention. In fact, this increase in angiotensin II is evident before any measurable derangement in systemic haemodynamic characteristics.9 The values measured in healthy volunteers are also significantly higher than those reported in the literature. We are not sure if these differences in 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 ml of 0·45% O-phenanthroline and 1% disodium 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.2 In this, we feel it should be stressed that in patients with cirrhosis and varying degrees of severity of sodium retention (preascitic (PA) cirrhosis, diuretic responsive (DR) ascites, and refractory ascites). Our results and the values reported by Girgrah et al are 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; f=f<0.05 v preascitic cirrhosis and controls; *p<0.05 v controls, preascitic cirrhosis and refractory ascites.

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tions. We agree with the increase in serum angioten-
sin II levels with deterioration of liver dis-
case. In general, we largely agree with much of
the Edinburgh group’s findings. In particular,
we agree that in cirrhotic patients with elevated
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EDITOR,—Jass has pointed out that the instability status are not in accordance with the conclusion of our study and submission of our manuscript were contemporaneous to our study in avoiding potentially confounding effects of tumour stage on microsatellite instability and mutations of the transform-}


Fibrosonic colonopathy in an adult caused by overuse of pancreatic enzyme supplements

EDITOR,—We read with interest the report by Bansi and colleagues (Gut 2000;46:285–287) describing fibrosonic colonopathy secondary to high dose pancreatic enzyme therapy in an adult patient. Some details of the patient’s history—chronic cystic fibrosis and pancreatic insufficiency—are strikingly similar to symptoms displayed by our adult patient with cystic fibrosis and fibrosonic colonopathy described previously. 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 supplemen-tation. Bansi et al. state 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, 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, nasopharyngeal differences measurements should be performed. 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 pancreatic tissue, chronic cholangitis, cholecystitis and atrophy, is also typical of cystic fibrosis, as are frequent bowel actions. Proof of fibrosonic colonopathy in a patient not suffering from cystic fibrosis may contribute considerably to a better understanding of the pathology of fibrosonic colonopathy which is still a matter of discussion. It would underline the aetiologic 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. We would therefore be interested in the patient’s sweat chloride concentration and, if normal, in the result of nasopharyngeal difference measurements. This paper strongly advocates well thought out supplementation of pancreatic enzymes but caution against overestimation of the contribution of factors possibly related to the cystic fibrosis transmembrane regulator gene mutation, such as increased intestinal absorption.

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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 report 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 of relapse, and improved patient satisfaction (Cochrane, 1999, 14) and Aid 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 summarized 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 ileorectal 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-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 down or stopping maintenance therapy. It was surprising that there was no information for adolescents, or on osteoporosis, and little mention of the dilemmas of coexisting inflammatory bowel syndrome or the implications of differentiating ulcerative colitis from Crohn's colitis. A brief mention of new therapies on the horizon would have suited the aim of the book, if only to highlight the importance of clinical and basic science research, which were simply ignored.

The patient record booklet is a good idea, it will be of little interest to the practising gastroenterologist, but it may be interesting to a handful of researchers world wide. 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 recognize the opportunities offered by the rapid, 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 realized 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 synthesize 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 as 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 biology and medicine 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 the world is striving 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 biology 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.


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 recognize the opportunities offered by the rapid, 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 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 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 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 Mak, Dr Mario Hadjivassiliou, Professor Lionel Fry, Dr Gerd Michaelson 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 Médicine 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 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 22 908 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@hkstde.org
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