Gall bladder motility after endoscopic sphincterotomy

EDITORS—We read with interest the article by Sugiyama and Atomi (Gut 1996;39:856–9) on the effect of endoscopic sphincterotomy (ES) on gall bladder motility. These authors have proved conclusively that ES causes a significant improvement in gall bladder motility in humans. We have assessed gall bladder motility in patients who underwent ES for common bile duct (CBD) stones and compared these with patients with gall bladder calculi but without CBD stones. We found significantly lower fasting and residual gall bladder volumes in the patients who had undergone ES and indicating reduced stasis after ES. However, the ejection fraction and the rate constant of gall bladder emptying in these patients was increased significantly, suggesting increased gall bladder emptying.

In another study we found a significant decrease in fasting volume (mean SD) 18.3 (8.5) v 13.9 (7.3) ml, residual volume (12.0 (8.0) v 4.4 (3.2) ml) and an increase in the ejection fraction (54.3 (9.8) v 83.5 (5.4)%) after ES, suggesting decreased stasis and increased gall bladder emptying.

In Sugiyama and Atomi’s study, all patients in whom gall bladder motility was assessed before ES had CBD stones; all stones were extracted before the motility studies were repeated. The major problem in this study is in differentiating between the effects of the CBD stones and the sphincterotomy itself. Fasting and residual volumes and maximum contraction were significantly different before and after ES. However, when one looks at these parameters in normal controls, it is clear that abnormal values before ES tend to normalise after ES, especially in patients with primary CBD stones. This strongly suggests that CBD stones adversely affect gall bladder motility. Common bile duct stones hinder gall bladder emptying by obstructing the lower CBD. In our study the patients had extracted CBD stones and some also had multiple/large CBD stones, indicating significant obstruction. We circumvented this problem by including patients without significant CBD obstruction as reflected by normal serum bilirubin, a normal CBD diameter on cholangiography, and single/small CBD stones. Therefore, we believe that in this study improvement in gall bladder motility could be due to both ES and removal of the stones.

In Sugiyama and Atomi’s study healthy volunteers were used as controls rather than patients with gallstones but without CBD stones. If gall bladder volumes and maximum contraction in patients with CBD stones were the same as in those with gallstones but without CBD stones, then one could conclude that the presence of CBD stones did not affect gall bladder emptying. However, gallbladders have small, contracted gall bladders because of associated chronic cholecystitis. However, in Sugiyama and Atomi’s study fasting gall bladder volume was the same in patients with both gall- and CBD stones as in those with primary CBD stones. Abnormal motility is not expected in the latter. Of the patients with both gall- and CBD stones, six of 15 patients had pigmented stones and would probably have had normal gall bladder motility. Behar and colleagues have shown that gall bladder muscle contractility is normal in patients with pigmented stones in comparison with patients with cholesterol stones who have reduced gall bladder muscle contractility. It is not clear whether Sugiyama and Atomi assessed gall bladder motility in a blinded fashion.

Sugiyama and Atomi mention that thickening of gall bladder wall after ES is due to reduced fasting volume. How a reduction in fasting volume can lead to thickening of the wall is not clear. After ES, bacterial biliopancreatic juices and duodenal contents can enter the gall bladder, which may lead to regurgitation, cholesterol cholecystitis and thickening of the gall bladder wall.

Improvement in gall bladder motility after ES is due to abolition of papillary resistance. With division of the papilla, resistance is lost immediately and CBD pressure equalises to duodenum. This usually leads to a rapid improvement in gall bladder emptying as demonstrated by Sugiyama and Atomi on day 7 after ES. However, why gall bladder emptying continued to improve for three months after ES was not explained. Despite a significant reduction in the size of the ES over five years, there was no evidence of a gradual decline after the initial improvement in gall bladder motility.

B C SHARMA
K SINGH
Department of Gastroenterology,
Postgraduate Institute of Medical Education and Research,
Chandigarh 160 012, India

R K DHIMAN
Department of Hepatology
Correspondence to: Dr B C Sharma, 591, Sector 2, Panchkula, Haryana, India.


Specificity of small bowel biopsy findings in coeliac disease

EDITORS—We appreciate the comments raised by Sharma et al about our paper. We agree that improvement in gall bladder motility after endoscopic sphincterotomy could be due to both decreased resistance of the terminal common bile duct and clearance of CBD stones, as we described in the discussion. We have found that patients with cholesterol cholecystolithiasis have a lower maximum contraction than those with gallstones but without CBD stones (unpublished data). Therefore, the presence of the CBD stones seems to affect gall bladder emptying. In our study, gall bladder function tests were carried out in a non-blinded manner.

Patients with severe cholecystitis have a small gall bladder. However, the patients had no or mild cholecystitis and a larger fasting gall bladder volume than normal controls. Some patients with primary CBD stones may have mild cholecystitis or bile stasis, which may affect gall bladder motility. The brown pigmented stones cause cholecystitis and gall bladder dysfunction.

The gall bladder wall thickens as the gall bladder empties. Therefore, mild thickening of the gall bladder wall after sphincterotomy seems to be caused partly by the reduced fasting volume of the gall bladder. Regurgitation cholecystitis may also cause wall thickening, as Sharma et al state.

Improvement in gall bladder motility reached a plateau three months after sphincterotomy. Initial improvement may also have been caused by decreased resistance of the terminal duct and stone clearance, as mentioned earlier. Further improvement after three months cannot be fully explained but may be partly from changes in bile composition.

M SUGIYAMA
Y ATOMI
The First Department of Surgery,
Kyorin University School of Medicine,
6-20-2 Shinkawa,
Mitaka, Tokyo 181, Japan

Correspondence to: Dr Sugiyama.


LETTERS TO THE EDITOR

Gut 1998;43:146–150

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showing the “typical” coeliac sprue lesion, a clinical response to gluten withdrawal is required to diagnose the disorder unequivocally.

J C YARZE
Gastroenterology Associates of Northern New York, 9 Iron Gate Country, Glovers Falls, New York, 12801, USA


Reply
EDITOR.—I thank Dr Yarze for his interest and kind comments. He is of course correct that small bowel biopsy is not 100% specific for coeliac disease taken in isolation. (Very few tests reach 100% specificity.) However, I hope he would agree that, in conjunction with a positive anti-gliadin antibody used as the initial screening test, the specificity is high. Another way of looking at this would be the positive predictive value of a small bowel biopsy, which depends upon the prevalence of the disease in the population being studied.

As coeliac disease is relatively common and a positive anti-gliadin antibody used as the initial screening test, the specificity is high. Another way of looking at this would be the positive predictive value of a small bowel biopsy, which depends upon the prevalence of the disease in the population being studied. The small bowel biopsy would have a high positive predictive value.

J METCALF
Gloucestershire Royal Hospital, Great Western Road, Gloucester GL1 3NN, UK

Prevalence of the factor V Leiden mutation in portal and hepatic vein thrombosis
EDITOR.—We read with great interest the article by Mahmoud et al (Gut 1997;40:798–800) on the prevalence of factor V Leiden (FVL) mutation in hepatic and portal vein thrombosis. In the past few years the FVL mutation has emerged as the commonest genetic risk factor for venous thrombosis. The FVL mutation seems to be most prevalent in Europe, extending to northern India in the east and Saudi Arabia in the south. The two striking features of the population genetics of the FVL mutation are the confined racial distribution and the high prevalence in European peoples (allele frequencies ranging from 1.4% to 7%). We have found an allele frequency of 1.9% for the FVL mutation in 130 unrelated subjects from northern India who had no predisposing cause or family history of thrombosis.

In India portal vein thrombosis (PVT) and hepatic vein thrombosis (Budd-Chiari syndrome) are frequently encountered hepatic disorders causing portal hypertension. In a series of patients with PVT from our institute the cause of disease could be elucidated in only 17 of the 213 cases. Umbilical sepsis led to PVT in 13 patients and congenital heart disease was responsible in four. Budd-Chiari syndrome represents a spectrum of disease primarily caused by a hypercoagulable state. Analysis of our cases of Budd-Chiari syndrome revealed the pathology in 65 of the 177 patients. In four patients Budd-Chiari syndrome was associated with polycythaemia vera and in three with paroxysmal nocturnal haemoglobinuria. It has been well recognised that the clinical outcome, course and aetiological factors leading to PVT and Budd-Chiari syndrome are substantially different in the East and West.

As the FVL mutation has recently been detected in our population we attempted to study cases of PVT and Budd-Chiari syndrome to evaluate the role of this mutation in the aetopathogenesis of these two conditions. Twenty three cases of PVT and nine cases of Budd-Chiari syndrome, including the five reported in our previous study, constituted the study group. The FVL mutation was detected as described by Bertina and colleagues. Genomic DNA was extracted from leukocytes by chloroform-phenol extraction, amplified using the polymerase chain reaction using specific primers, and the product was digested with the MnlI restriction enzyme. One of the 23 patients with PVT and two of the nine patients with Budd-Chiari syndrome were heterozygous for the FVL mutation. None of the patients was homozygous for the mutation. Another case of Budd-Chiari syndrome was associated with polycythaemia vera and thus was homozygous for the FVL mutation. The prevalence of FVL mutation in our small series seems to be higher in patients with Budd-Chiari syndrome than in those with PVT or in controls. Therefore we concur with Mahmoud et al’s observation that screening for the FVL mutation is important in patients with Budd-Chiari syndrome.

However, in cases of idiopathic PVT this mutation does not seem to have a major pathogenic role.

R DAS
GAREWAL
Department of Haematology, Postgraduate Institute of Medical Education and Research, Chandigarh 160 012, India

Y CHAWLA
R K DHUMAN
Department of Hepatology

Correspondence to: Professor G Garewal


Steatorrhoea and pancreatic disease in HIV infected patients
EDITOR.—The study by Carbonnel et al (Gut 1997;41:805–10) on macro nutrient intake and steatorrhoea in HIV infected patients tackles one important clinical feature of HIV infection. As the severity of disease increases, the ability of patients to digest carbohydrates and proteins becomes impaired, resulting in reduced protein and fat absorption. Therefore, patients with AIDS are vulnerable to malnutrition. Even asymptomatic patients at an early stage of disease may have changes in body composition as shown by a reduction in body cell mass, which is associated with an increased mortality and morbidity. HIV associated malnutrition has a multifactorial origin but as shown by Carbonnel et al other authors steatorrhoea is a common problem in HIV infected patients, especially in those with opportunistic infections of the gastrointestinal tract. Inadequate energy intake is a major determinant of malnutrition in these patients, which is confirmed by the fact that enteral refedding can improve their nutritional status.

Unfortunately, Carbonnel et al’s study missed one important aspect of steatorrhoea—next to decreased fat absorption, exocrine pancreatic insufficiency may also result in lipid malabsorption. Raised pancreatic serum enzyme concentrations are frequently seen in patients with AIDS and also to a lesser extent in asymptomatic HIV infected patients, suggesting the presence of pancreatic inflammation. A recently published study reported that 54% of patients with suspected AIDS related cholangitis who underwent endoscopic retrograde cholangiopancreatography (ERCP) had a pathological pancreaticotgraphic (β, 26%; II, 47%; III, 47% classified according to Cotton bridge classification). Pathological pancreatographies were associated with opportunistic infections (candida, cytomegalovirus, cryptosporidiosis, microsporidiosis, and mycobacteria) and CD4 cell count less than 200 cells/μl. Therefore these patients have an increased risk of pancreatic involvement. To our knowledge no studies have been published on exocrine pancreatic function in HIV infected adult patients.

In a small preliminary study in HIV infected patients, drench five of 17 patients had pancreatic dysfunction and three of five had significant steatorrhoea.

Further investigations of lipid malabsorption and weight loss in HIV infected patients, especially in those with advanced disease and cryptosporidial or microsporidial infection, should include a diagnostic work up of the exocrine pancreas. This is important because these patients may benefit from pancreatic enzyme supplementation.

J OCKENGA
M GOKE
M P MANNS
Department of Gastroenterology and Hepatology, Medical School Hannover, 30623 Hannover, Germany

Correspondence to: Dr J Ockenga (email: ockenga@aol.com)

group, yielding a comparison of 100% vs 63.4%. Finally, the penultimate line in table 3, dealing with the healing rate for \( H. pylori \) positive patients allocated to placebo, did not make sense. These are minor criticisms in an otherwise good paper.

O M JOLUBE
Gersasim, Department of Medicine for the Elderly, Tameside General Hospital, Ashton under Lyne OLE 6BW, UK

Reply
EDITOR,—We appreciate Ockenga et al’s comments on our study. Although we did not assess pancreatic function in our patients, we would like to discuss the hypothesis that exocrine pancreatic insufficiency may contribute to steatorrhoea in malnourished HIV infected patients. As pointed out by Ockenga and colleagues, HIV infected patients frequently have elevated pancreatic serum enzymes and this can be due to several causes. Further, a recent study has shown that pancreatic abnormalities are frequently found on ERCP of HIV infected patients with cholangitis. However, these biochemical or morphological abnormalities do not necessarily imply that exocrine pancreatic insufficiency is present. Indeed, roughly 90% of the secretory capacity must be lost before fat malabsorption occurs. In addition, previous studies have shown that faecal fat concentration is higher in patients with pancreatic insufficiency than in those with gastrointestin al diseases. In the paper by Bo-Linn and colleagues, all patients with pancreatic steatorrhoea had faecal fat concentrations of more than 9.5%. In our study, the faecal fat concentration was significantly lower in HIV infected patients than in patients with small bowel disease or short bowel syndrome, suggesting higher intestinal secretion; a faecal fat concentration of more than 9.5% was found in only one of 79 patients. Intestinal involvement is sufficient to explain faecal weight and steatorrhoea in HIV infected patients. Yet, these data do not exclude that, in some patients, particularly the severely malnourished ones, some degree of exocrine pancreatic insufficiency may aggravate malabsorption.

F CARBONNEL
J COSINES
Service de Gastroentérologie et Nutrition, Hôpital Rothschild, 33 Boulevard de Pépinié, 75012 Paris, France

One week triple therapy for Helicobacter pylori infection
EDITOR,—The paper by Misiewicz et al (Gut 1997;41:735–9) is an important contribution which demonstrates the usefulness of lansoprazole and clarithromycin combined with either amoxicillin or metronidazole in the treatment of Helicobacter pylori infection. However, there are some questions which arise.

(1) Only half the patients had duodenal ulcers. There is good evidence that the response rate to eradication therapy is inferior in other patients and it is hard to justify grouping ulcer and non-ulcer patients together in the study.

(2) Why use lansoprazole 30 mg twice daily? Optimal acid reduction should be achievable with 30 mg daily, and doubling the dose merely reduces the cost effectiveness of this drug. The balance of evidence suggests that omeprazole 40 mg daily, lansoprazole 30 mg daily and pantoprazole 40 mg daily should be equivalent in these regimens.

(3) It is amazing that the combination of lansoprazole or omeprazole with twice daily amoxicillin and metronidazole was judged to be “effective”. The success rates achieved were 66% to 83% depending upon the method of analysis. These sorts of incomplete success rates rightly led to the abandoning of dual therapy with omeprazole and amoxicillin or clarithromycin, and are no better than the old triple therapy with bismuth, tetracycline and metronidazole, which has now been superseded.

M C BATESON
Department of Gastroenterology, Bishop Auckland and County Hospital, Cockton Hill Road, Bishop Auckland, Co Durham DL14 6AD, UK

Reply
EDITOR,—We thank Dr Bateson for his interesting comments. We should like to reply to each of his questions in turn.

(1) The success of \( H. pylori \) eradication therapy depends on the patients’ compliance with treatment and the sensitivity of the bacterium to antimicrobials. We are unaware of studies reporting significantly greater efficacy in the other treatment arms, compared with patients with non-ulcer dyspepsia.

(2) When combined with clarithromycin and a nitroimidazole, we agree that at present there seems to be no therapeutic advantage in increasing the dose of lansoprazole above 30 mg, of omeprazole above 20 mg, or pantoprazole above 40 mg daily. However, no such comparative data exist for the combination of a proton pump inhibitor (PPI) with clarithromycin and amoxicillin. When we planned the study, the data available supported the twice daily use of a PPI with antimicrobials, and also the patients’ compliance seems to be better.

(3) A PPI together with amoxicillin and metronidazole was “judged to be effective” as this combination produced eradication of \( H. pylori \) in 88 to 94% of patients with metronidazole sensitive strains, according to intention to treat analysis. In such circumstances, these regimens were as effective as the other treatment arms. However, these regimens are significantly less effective in the presence of metronidazole resistant strains, with eradication between 46 and 62%. As discussed in the paper, these regimens are not adequate in the circumstances where antimicrobial sensitivities of \( H. pylori \) are unknown or in areas where the prevalence of such strains is at least 30%, where a PPI in combination with amoxicillin and clarithromycin should be the first choice of treatment.

A HARRIS
Kent and Sussex Hospital, Mount Ephraim, Tunbridge Wells, Kent TN4 6AT, UK

J MISIEWICZ
Central Middlesex Hospital, London NW10 7NS

Letters, Book reviews, Notes

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I may get a reputation for this, but I’m going to do it anyway. I reviewed a book for Gut a few months ago, and grumbled vociferously about the title; specifically, I didn’t like the phrase “clinical pathology” in its title because I didn’t know what the authors would mean by “clinical pathology”. This time, the title is easily understood; it’s just that it’s deeply unhelpful. Pancreatic Growth and Regeneration is really about the growth and regeneration of the pancreas, and very little else. There’s precious little about any other endocrine cell, and the exocrine pancreas only appears when its interaction with the islets is discussed. The phrase “tissue engineering” appears on the cover, but that doesn’t seem to be a major clue to the book’s contents, either.

This book consists of a series of very detailed reviews, concentrating on the cellular and molecular biology of the β-cell, and in particular the role of growth factors. If this is all you want to know about, then this book is fine: coverage is detailed and thorough, the text is densely and appropriately referenced, and most of it seems to be more up to date than most books at the time of publication. The book seems to be written by molecular biologists for molecular biologists, with most of the chapters providing a precise description of the authors’ work. I found it hard to place the work within a slightly broader scientific context. There is very little concession to the interested amateur, though members of other disciplines may be rewarded if they persist. There is also an effort to put matters into an appropriate clinical context, and abbreviations come thick and fast; not all are explained.

Doctors tend to know a little about many things, and basic scientists know an enormous amount about a few things. If we let it, this dichotomy can become an insuperable cultural divide, with each side holding the other in entirely inappropriate contempt. Struggling across the chasm can be enormously productive and rewarding, but texts like Pancreatic Growth and Regeneration are hardly attractive bait to tempt one to make the effort. It would probably not be much more tempting even if it were adorned with an appropriate title, such as Molecular and Cellular Biology of the Growth and Regeneration of the Pancreatic β-Cell. However, that would be much more accurate, and less likely to deceive the naive into expecting something quite different between its covers.

There are an increasing number of CD-ROMs being peddled in the medical textbook market, either as de novo products of variable quality, or, as in this example, an electronic version of an established textbook. The disk’s contents include not one, but four books. These are verbatim reproductions of the second (1995) edition of Yamada’s text-book of gastroenterology, together with the bonus of the accompanying self assessment question book, the atlas of gastroenterology and its self assessment book. This certainly represents value for money, saving at least £80 if one was to buy the printed set. There is also a considerable saving in weight (100 g compared with several kilograms), although it does not look quite so impressive sitting on the bookshelf and could easily be mislaid. Of course, one does require a reasonable computer (with 8 megabytes of RAM, 11 megabytes of hard disk space if using Windows) to use the book. This is probably available in most homes, but, given the usual financial constraints, not yet in most gastroenterology departments. However, installation was simplicity itself, and the software ran rapidly on my three year old Macintosh.

I will not review the textbook itself, as this has previously received favourable comments, with which I concur. I will instead concentrate on the electronic part of this edition.

Anyone expecting the gastroenterology equivalent of the Hitch Hiker’s Guide to the Galaxy may be somewhat disappointed, although at least the CD-ROM publishing, and optimistic. The presentation mirrors that of the book, although one views less text per page on a standard monitor. The text is peppered with highlighted links that enable the reader to jump to other definitions of related interest, including references. Luckily you can easily retrace your steps if you have decided to hop around. Tables and figures appear at the side of the screen, and blow up to full size after a short delay. Text and figures can be copied to a printer, and reproduction is very good, even using an inkjet machine. Word searches are certainly more convenient than using the printed index, and the software allows restriction to one or all of the textbooks. However, you may be faced with a long list of (sometimes tenuous) entries if you do not consider your search criteria carefully. These problems will be familiar to anyone who uses Medline or the Internet. One can also annotate the text electronically, which is somehow more acceptable than defacing the printed edition.

The main problem I have with such CD-ROMs is that their potential is rarely fully realised, which includes this one. Only 55% of the disk space is used, implying that there would be ample space for additional material. Examples could include moving ultrasound images, and videos of procedures such as liver biopsy or endoscopic procedures mentioned in the text. In theory the references could include the whole abstract, copyright permitting. The accompanying self assessment books are weakly interactive: one does not have to look up the answer as it tells you straight away, but I somehow feel that a CD-ROM could offer yet more. Again, in theory it could be linked via the Internet to the publisher’s web site for up-to-date amendments. In common with fellow registrars and medical students, I am anxious as to the relative ease of use of CD-ROM text books, and the tiring nature of reading long passages on screen rather than paper.

I bought this CD-ROM six months ago with my own hard earned cash, and generally have not been disappointed. It has proved invaluable in my own studies, as well as for ideas in teaching undergraduate and postgraduate medicine. I have tested the images on a lecture theatre video projection system with good results, the only problem being that the licence prevents multi-user access (like all computer software one doesn’t actually own the disk or its contents, just permission to use it). These problems aside, I would heartily recommend this CD-ROM to all specialist registrars and gastroenterology departments that do not already own this excellent text, which is as good as any other general gastroenterology textbook, but better value. One cannot exactly leaf through it next to an open fire supping a glass of port, but this is the price of progress I suppose.

W P GODDARD


The first formal right hepatectomy was reported by Lortat-Jacob and Robert in 1952. Before that time surgeons tried to strip clear of liver surgery because of the risk of uncontrollable haemorrhage and the operative mortality rate of at least 20%. However a widening knowledge of segmental hepatic anatomy described by Couinaud in 1952 was elaborated by Bismuth in 1982, led to the development of safe resectional techniques for the various major segments of the liver so that inoperable deaths have become rare events in elective liver surgery. The techniques of hepatobiliary surgery have developed rapidly and the subject is now recognised as a subspecialty in its own right to encompass very advanced procedures such as transplantation from living, related donors, split liver transplantation and neontal liver replacement.

This book (dedicated to the memory of Professor Gozetti of Bologna) is an atlas of every type of surgical technique applicable to liver surgery and is based on the work of the surgical department in Bologna over more than 15 years. A brief summary of the concepts of hepatic anatomy is followed by descriptions of the standard methods of liver resection as well as the more unusual and difficult operations designed to resect single segments such as the caudate lobe (segment 1), segment 4 or segment 8. Other topics include total vascular occlusion of the liver, which is a very valuable surgical technique for extensive tumours, intraoperative ultrasound, portal embolisation and the management of trauma. The surgery of cystic disease of the liver and tumours of the bile ducts is included and almost one third of the volume is devoted to a comprehensive review of the techniques of liver transplantation. For the inclusion of discussions on possible technical complications in many of the sections, especially as the authors detail the methods of management. These comments are valuable and extremely useful for any newcomer to liver surgery.

As a minor criticism I would have preferred the more general sections of the text to have been grouped at the beginning of the book to form an introduction to the chapters con-


There are increasing numbers of CD-ROMs being marketed in the medical textbook market, either as de novo products of variable quality, or, as in this example, an electronic version of an established text title.
cerned with specific resectional techniques. It seems a little unusual to describe and illustrate all of the standard operations before introducing a discussion on the general principles of liver resection in section 14, and the indications for liver resection in section 18. Throughout the text there are references to the results of the various operations, but these are, of necessity, rather restricted in scope and the reader would have to refer to other texts for further data.

The superb quality of the illustrations, produced in the department of anatomy in Bologna, provides the outstanding feature of this book, although it was a little disappointing to find that the section devoted to the resection of tumours of the extrapleural bile ducts was not illustrated in the same manner and the intraoperative photographs used in this chapter lack the clarity of the illustrations in the other sections. A potential purchaser of a book on liver resection can now choose from a wide range of texts but in my opinion this book should be high on the list. The technique of illustration avoids the simplification of the more usual type of line diagram and avoids the lack of clarity which often accompanies the reproduction of intraoperative photographs. It really does give a good impression of the “feel” of liver surgery and does not avoid discussion of the potential hazards. Furthermore although the book was designed primarily as a manual for liver surgeons I am sure that any gastroenterologist with an interest in liver medicine would appreciate the illustrations both as an extremely useful guide to the current surgical repertoire of liver surgery and as an extremely fine example of the art of the medical illustrator.

E R HOWARD

NOTES

International Symposium on Cytokines and Inflammatory Bowel Disease

The International Symposium on Cytokines and Inflammatory Bowel Disease will be held in Bath, UK, on 16–17 July 1998. Further information from: Mrs Carolyn Stock, Organising Secretary, School of Pharmacy and Pharmacology, University of Bath, Bath BA2 7AY, UK. Tel/Fax: +44 1249 821 225; Email: prsjcs@bath.ac.uk.

Hepatocellular Carcinoma: Eastern and Western Experiences

An international conference on Hepatocellular Carcinoma: Eastern and Western Experiences will be held at the Institut Curie, Paris, France, on 28–29 August 1998. Further information from: Agence BCA, Michele Centonze, 6 bd Général-Leclerc, 92115 Clichy, France. Tel: +33 1 41 06 67 77; Fax: +33 1 41 06 67 78.

16th Leeds Course in Clinical Nutrition

The 16th Leeds Course in Clinical Nutrition will be held at the University of Leeds, UK, on 13–18 September 1998. Further information from: Ma Samantha Armitage, CVE Section, School of Continuing Medical Education, Continuing Education Building, Springfield Mount, Leeds LS2 9NG, UK. Tel: +44 113 233 3241; +44 113 233 3240; Email: s.armitage@leeds.ac.uk.

Current Trends in Colon and Rectal Surgery

The second postgraduate course in Current Trends in Colon and Rectal Surgery will be held in Sorrento (Naples), Italy, on 24–26 September 1998. Further information from: Dr Angela Palma, Organising Secretariat, Convention Planning s.a., via Fuornmura 20, 80067 Sorrento, Napoli, Italy. Tel: +39 81 807 1981; Fax: +39 81 807 3039; Email: convplan@syrene.it; WWW: http://www.syrene.it/convplan/.

European Mucosal Immunology Meeting, The Cells and Molecules Important in Mucosal Tolerance and Inflammation

The European Mucosal Immunology Meeting: The Cells and Molecules Important in Mucosal Tolerance and Inflammation will be held at the Charterhouse Square Campus of St Bartholomew’s and the Royal London School of Medicine and Dentistry, London, UK, on 2–3 October 1998. Further information from: Professor T T MacDonald, Department of Paediatric Gastroenterology, St Bartholomew’s Hospital, London EC1A 7BE. Email: t.t.macdonald@mds.qmw.ac.uk.

Laparoscopic Surgery

A Course on Laparoscopic Surgery will be held at the University Hospital Saint Pierre, Brussels, Belgium, on 17–20 November 1998. Further information from: Conference Services S.A., Avenue de l’Observatoire 3, bte 17, B-1180 Brussels, Belgium. Email: conference.services@skynet.be.

Second Inflammatory Bowel Disease Meeting

The Second Inflammatory Bowel Disease Meeting will be held at Chester Town Hall, Chester, UK, on 23–24 November 1998. Further information from: Professor J M Rhodes, Royal Liverpool University Hospital, Prescot Street, Liverpool L7 8XP. Tel: +44 151 706 3558; Fax: +44 151 706 5832.

13th International Workshop on Therapeutic Endoscopy

The 13th International Workshop on Therapeutic Endoscopy will be held at the Endoscopy Centre, Prince of Wales Hospital, Hong Kong, on 1–3 December 1998. Further information from: Professor Sydney Chung, Endoscopy Centre, Prince of Wales Hospital, The Chinese University of Hong Kong, Shatin, N.T., Hong Kong. Tel: +852 2632 2233; Fax: +852 2635 0075.

European Postgraduate Gastro-Surgical School Symposia

The 7th Course on Digestive Endoscopy - Live will be held at the Academic Medical Centre, Amsterdam, The Netherlands, on 3–4 September 1998. Registration fee: NLG 450.00.

H. pylori: from Bench to Bedside will be held at the Academic Medical Centre, Amsterdam, The Netherlands, on 24–25 September 1998. Registration fee: NLG 300.00.

Minimally Invasive Surgery: A Critical Evaluation will be held at the Academic Medical Centre, Amsterdam, The Netherlands, on 13 November 1998. Registration fee: NLG 200.00.

From Gene to Cure II: Bilio-pancreatic malignancy will be held at the Academic Medical Centre, Amsterdam, The Netherlands, on 4–5 February 1999.

Further information from: Helma Stockmann, Managing Director, European Postgraduate Gastro-Surgical School, G-4-ziud, Academic Medical Centre, Meibergdreef 9, 1105 AZ Amsterdam, The Netherlands. Tel: +31 20 566 3926; Fax: +31 20 566 6569 or 691 458; Email: w.j.stockmann@amc.uva.nl.

Sir Francis Avery Jones BSG Research Award 1999

Applications are invited by the Education Committee of the British Society of Gastroenterology who will recommend to Council the recipient of the 1999 Award. Applications (TWENTY COPIES) should include:

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- 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.

Entrants must be 40 years or less on 31 December 1998 but need not be a member of the BSG. The recipient will be required to deliver a 40 minute lecture at the Annual Meeting of the Society in March 1999. Applications (TWENTY COPIES) should be made to the Honorary Secretary, BSG, 3 St Andrews Place, London NW1 4LB, by 1 December 1998.
Gall bladder motility after endoscopic sphincterotomy

B C SHARMA, K SINGH and R K DHIMAN

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