LETTERS TO THE EDITOR

The aging stomach

EDITOR,—After reading Lee and Feldman's excellent article on the aging stomach (*Gut* 1997;**41**:425–6), I have realised how many concepts we take for granted even in the absence of clear-cut scientific evidence. In their exhaustive review Lee and Feldman emphasise the paucity of human studies on the state of mucosal defence systems in the elderly. I actually had some data on file which have never been published as I felt that the results were "too obvious" to interest any medical journal. I can only blame myself for not searching Medline and not noticing earlier how little has been published on—for example, the gastric mucus-bicarbonate barrier in the elderly.

Comparative data from my laboratory on 45 patients over 65 years of age, without endoscopic gastroduodenal alterations, and 45 matched patients under 60, showed that the amount of gastric mucoproteins in fasting gastric juice was significantly reduced in the former, whereas the ratio of neutral to total mucoproteins—the so called mucoprotective index, regarded as an index of mucus quality[1]—was unaffected by age (Guslandi *et al* unpublished observations). This is in partial agreement with Lee and Feldman's findings in the rat and apparently is the only (unpublished) experience in humans. Measurement of basal gastric bicarbonate secretion in the same patients, performed using Feldman's method, yielded results comparable to those of Feldman and Cryer,[2] namely a significant decrease in elderly patients.

Gastric mucosal blood flow is either reduced or unchanged in the elderly.[3 4] Recent measurements of basal gastric microcirculation using laser Doppler flowmetry in 24 patients over 65 years of age showed that gastric blood flow is significantly lower than normal (Guslandi *et al*, unpublished observations), a finding that, although partly predictable, was previously supported only by animal studies and by a single report in humans.[3]

All in all, our data indicate that in the elderly, gastric mucus production, bicarbonate secretion, and gastric mucosal blood flow are indeed reduced. These observations are consistent with previous reports from Feldman's group of a decrease in gastric prostaglandin biosynthesis in elderly subjects and accounts for the vulnerability of their gastric mucosa to damage from ingestion of non-steroidal anti-inflammatory drugs.

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Reply

EDITOR,—We would like to thank Dr Guslandi for his interest in our paper. These unpublished observations from Dr Guslandi's laboratory confirm and extend the findings reviewed in our article. Collectively, available data suggest that aging is associated with selective and specific changes in gastric mucosal defence mechanisms that may predispose the elderly to non-steroidal anti-inflammatory drug (NSAID) gastropathy. Future studies should be focused on the mechanisms of these age related changes in gastric mucosal function, which may help us to develop novel interventions for the treatment and prevention of NSAID induced gastrointestinal injury in susceptible elderly individuals.

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Helicobacter pylori: beware "blind" eradication!

EDITOR,—The Maastricht Consensus Report (*Gut* 1997;**41**:8–13) has recommended broadening the guidelines on eradication of *Helicobacter pylori* to include certain dyspeptic patients without confirmed peptic ulceration. Although this policy may improve dyspeptic symptoms in certain subgroups of patients without ulcers,[1] an overall benefit from eradication of *H pylori* in non-ulcer dyspepsia has yet to be established. Furthermore, while a "test and treat" strategy may have cost benefits,[2] potential dangers to individual patients should be considered. Such dangers are illustrated by a patient who was recently admitted under our care with fulminant pseudomembranous colitis following triple therapy for the treatment of *H pylori*.

The 40 year old woman had complained to her general practitioner of colicky epigastric pain, associated with reduced stool frequency over the previous 12 months. Following a positive ^{13}C breath test, she was prescribed a two week course of bismuth chelate 120 mg four times daily, metronidazole 400 mg three times daily and amoxycillin 500 mg four times daily. Two weeks later she was admitted to hospital with severe diarrhoea and abdominal pain. Examination revealed a pulse of 120 beats/min and a systolic blood pressure of 80 mm Hg. Her abdomen was distended, with localised peritonism in the right iliac fossa. Following resuscitation, she underwent laparotomy, when the diagnosis of severe, generalised pseudomembranous colitis was confirmed, and a subtotal colectomy was undertaken. *Clostridium difficile* toxin was later identified on stool examination. Examination of the resection specimen revealed a partially obstructing Dukes' B carcinoma of the mid-transverse colon. She was treated with enteral vancomycin 250 mg three times daily and, after a prolonged stay on the intensive care unit, made a full recovery.

This case illustrates two important lessons. Firstly, it shows the potential dangers of linking gastrointestinal symptoms to positive *H pylori* breath and serological tests without further investigation. A presumptive diagnosis of *H pylori* related pain was based on the combination of epigastric pain and a positive breath test. In retrospect the original symptoms were probably caused by her transverse colon carcinoma. Although rare in this age group, this diagnosis should have been considered, given her change in bowel habit. Furthermore, if a negative upper gastrointestinal endoscopy had been obtained, the nature and severity of her symptoms might have led to a search for an alternative explanation.

Secondly, this case emphasises the potential dangers of antibiotic treatment regimens in the treatment of *H pylori*. The eradication therapy given in this case has now been largely superseded by shorter treatment courses incorporating a proton pump inhibitor, combined with a combination of two antibiotics, usually amoxycillin or clarithromycin and metronidazole. Minor side effects, including taste disturbance, diarrhoea and rashes are relatively common, but more serious events have also occurred. *Clostridium difficile* positive diarrhoea and fulminant pseudomembranous colitis have been described following helicobacter eradication regimens containing amoxycillin[3 4] and clarithromycin,[5] although not previously with such dramatic consequences. The presence of a partially obstructing carcinoma in this patient's colon may have influenced the severity of the problem at presentation.

The authors of the Maastricht Consensus statement admit that the evidence for benefit from *H pylori* eradication in functional dyspepsia and in the prevention of gastric cancer is equivocal. Although life threatening complications such as this are rare, clinicians should consider potential risks when *H pylori* eradication therapy is being considered without a proven benefit.

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1 McCarthy C, Patchett S, Collins RM, *et al.* Long-term prospective study of Helicobacter pylori in non-ulcer dyspepsia. *Dig Dis Sci* 1995;**40**:114–19.
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Are complications of endoscopic sphincterotomy age related?

EDITOR,—Deans *et al* (*Gut* 1997;**41**:545–8) have supported the use of endoscopic sphincterotomy for bile duct stones in young people in preference to laparoscopic bile duct exploration. They do this because sphincterotomy causes no more complications in young than in old people. Any comparison between the two methods must also take into account the complications, particularly pancreatitis, that occurred in the remaining two-thirds of the patients undergoing endoscopic retrograde cholangiopancreatography (ERCP) without sphincterotomy. Laparoscopic duct exploration is still a relatively new technique. Rhodes *et al* reported 129 explorations with one case of postoperative pancreatitis.[1] We have had no pancreatitis in 53 explorations. Until larger series are reported judgement is impossible.

For the treatment of bile duct stones a comparison of therapeutic success and the manoeuvres required to achieve it are required on an intention-to-treat basis.

The consequences of sphincterotomy and reflux in the very long term are not known. Most bile duct stones are predominantly composed of pigment, thought to be caused by reflux through an incompetent sphincter.[2] They are seen in about 10% of patients after sphincterotomy but these data include only small numbers of young patients with long term follow up. Data for the events of 20–30 years after sphincterotomy are not available, yet a quarter of the patients in the study of Deans *et al* might be expected to live this length of time. Apart from stone formation, long term duodenobiliary reflux may have other adverse effects. Kurumado *et al* reported dysplastic change in rat mucosa after formation of biliary enteric fistulae.[3] Biliary malignancy is seen in anomalous pancreatico-biliary junctions, postulated to be caused by unusual reflux.[4] The application of these reports to the effect of sphincterotomy needs to be cautious but they act as a warning that sphincterotomy might not be entirely benign in the long term.

Laparoscopic bile duct exploration offers the possibility of safe choledocholithotomy without sphincter destruction and it merits further study.

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1 Rhodes M, Nathanson L, O'Rourke N, *et al*. Laparoscopic exploration of the common bile duct: lessons learned from 129 consecutive cases. *Br J Surg* 1995;**82**:666–8.
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Reply

EDITOR,—This study was conceived because we had heard clinicians at several national and international meetings stating that young patients with stones in the common bile duct are better treated by surgical exploration than ERCP sphincterotomy because the latter has a 1% mortality. These clinicians were advocating open exploration of the common bile duct and to our knowledge none of them were performing laparoscopic bile duct exploration. Their statements were based on the global mortality from ERCP, irrespective of age and we were unable to find any scientific evidence relating the risks of ERCP sphincterotomy with age.

Our study is a prospective, multicentre audit of 1000 endoscopic sphincterotomies. No patient underwent ERCP without sphincterotomy and there was no comparison with any other technique such as laparoscopic common bile duct exploration. Mr Thompson's comments about a comparison with laparoscopic bile duct exploration and about "the remaining two-thirds of the patients undergoing ERCP without sphincterotomy" cannot therefore be directly answered by our report. We would like to emphasise that we use a selective policy for performing ERCP in patients undergoing laparoscopic cholecystectomy.[1 2] This policy has become more selective since we began using intravenous infusion cholangiography in patients with possible bile duct stones, a technique we have shown to be as effective as ERCP but with fewer complications and less cost.[3]

We accept that "data for the events of 20–30 years after sphincterotomy are not available." Such statements are true for the long term consequences of all recent innovations, including laparoscopic bile duct exploration. Concerns about the prolonged effect of duodenobiliary reflux in animals have not be substantiated by the few reports looking at the long term effects of ERCP sphincterotomy in humans. In a study of 100 patients undergoing ERCP sphincterotomy followed for a median of 15 years, late complications, such as recurrent ascending cholangitis or malignant degeneration, were not observed.[4] Recurrent bile duct stones, however, did develop in 24% of patients. The authors concluded that stone recurrence remains the most important long term problem after ERCP sphincterotomy, but that these stones can in general be managed by further ERCP.

Like Mr Thompson, we believe that laparoscopic bile duct exploration merits further study. In the introduction we state that "laparoscopic bile duct exploration is a well established technique but the time and skill required are likely to prevent it becoming universally available to patients." The experience of most surgeons with this technique is limited as supported by the relatively low numbers of patients reported in most series.[5] The dilemma in many busy NHS hospitals is that unexpected bile duct exploration, either laparoscopic or open, is time consuming and interferes with the running of an already tight operating schedule. We therefore stand by our statement that currently "many clinicians therefore use preoperative ERCP and sphincterotomy." We are, however, enthusiastic supporters of minimally invasive therapy, particularly laparoscopic surgery, and would be interested in being involved in a comparative study of the various treatment options available.

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History of dyspepsia in patients with gastric cancer

EDITOR,—We read with interest the study by Suvakovic *et al* (*Gut* 1997;**41**:308–13) reporting that 63% of the patients with early gastric cancer had a previous history of upper gastrointestinal symptoms. These authors concluded that guidelines for the appropriateness of gastroscopy in patients with dyspepsia need to reflect the importance of not starting treatment prior to gastroscopy. General practitioners need to be made more aware of the potential for antisecretory drugs to mask early gastric cancer, which will then have a major detrimental impact on patient management. We have also studied whether patients with gastric cancer had serious and persistent dyspeptic symptoms necessitating consultation with their general practitioner (Laheij *et al*, unpublished observations). The results of our study, however, differ from Suvakovic *et al*'s.

We identified 46 patients with diagnostically verified early and late stage gastric cancer, using the Continuous Morbidity Register. This database is used to study the epidemiological aspects of diseases in primary care. The primary care population of approximately 12 000 patients has been followed since 1971. The recorded data have passed stringent quality controls and are consistent over the years of registration.[1] Controls were selected and matched for sex, social class, practice, observation period, and age.

The mean observation period between the date of registration and the first diagnosis of gastric malignancy was 12 years. In this period, 18 (39%) patients had dyspeptic symptoms necessitating consultation with the general practitioner compared with 20 of the controls (odds ratio 0.8, 95% confidence interval 0.3 to 2.0). Patients with gastric cancer had no more visits to their general practitioner for gastrointestinal symptoms than controls. Therefore, consultation rates for dyspeptic symptoms cannot be considered a warning for gastric cancer.

Every patient with gastric cancer develops gastrointestinal symptoms and there will always be a delay in diagnosis.[2] Martin *et al*

found a median delay of 17 weeks from the onset of symptoms to a definitive histological diagnosis in patients with gastric cancer. Use of an open access endoscopy service reduced this delay. There was no correlation between the delay in diagnosis and tumour stage or the success of potentially curative resection. The prognosis for gastric cancer is poor. To detect early gastric cancer, patients should be examined before the onset of dyspeptic symptoms. Fortunately, gastric cancer is rare in the Western world and epidemiological studies have shown that the incidence of and mortality from gastric cancer have decreased considerably. Screening people for gastric cancer before the onset of symptoms is therefore not feasible. Early detection of gastric cancer is not yet possible, and focusing attention on patients with a history of serious gastrointestinal symptoms may be of little value.

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1 van Weel C. Validating long term morbidity recording. *J Epidemiol Community Health* 1995;**49**(suppl 1):29–32.
2 Martin IG, Young S, Sue-Ling H, *et al*. Delays in the diagnosis of oeosphagogastric cancer: a consecutive case series. *BMJ* 1997;**314**:467–71.

Reply

EDITOR,—The points raised by Laheij and colleagues are relevant to the debate regarding the late diagnosis of gastric cancer in Western Europe. Their figure of 39% consulting their general practitioner over a mean time period of 12 years is not far away from our figure of 30% investigated for symptoms in the 13 years leading up to diagnosis. We are not suggesting that all of these patients had gastric cancer at the initial time of presentation, but many almost certainly did (most of those investigated in the last four years—a "miss" rate of 1 in 6). There are two important points to note. Firstly, general practitioners may not think of the correct diagnosis because of previous (reassuring) investigations with the result that the diagnosis is delayed by years not months. Secondly, and related to the first point, general practitioners will prescribe powerful acid suppression for patients with a previous ulcer history which will result in resolution of symptoms and a further delay in diagnosis. This raises the question when do general practitioners re-refer a patient for gastroscopy when previous investigations showed no pathology or benign disease? It is also important to remember that early gastric cancer has "benign" symptoms and so it is effectively too late when the patient has lost weight or become anaemic.

We disagree that gastric cancer cannot be diagnosed earlier and progress will only be made if we alter current practice to include investigating patients "at risk" before starting antisecretory therapy and lowering the threshold for re-investigating patients who may have a history of dyspepsia. Screening is not feasible and we are not proposing such a policy. However within current practice we feel that there is room for improvement in the diagnosis of this disease which is not uncommon, representing the fourth commonest cancer in men in the UK.

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The genetics of ulcerative colitis based on one Swede

EDITOR,—I was interested to read the review on the role of inherited factors in inflammatory bowel disease by Satsangi *et al* (*Gut* 1997;**40**:572–4), but dismayed by the evidence provided. They claim that twin pairs provided strong evidence for genetic predisposition and that the most pertinent was that reported by Tysk *et al* using data from the Swedish Twin Registry.[1] However, the claim for ulcerative colitis rests on a single twin of the 50 000 registered. The probands were identified as hospitalised cases in Sweden. The 50 pairs of twins where one twin had suspected ulcerative colitis were whittled down to 36 pairs: 16 monozygotic and 20 dizygotic or of unknown zygosity. No hospitalised pairs were found. Twins of probands were interviewed and the occurrence of any gastrointestinal complaint was thoroughly discussed in order not to miss a mild non-hospitalised case. In spite of this approach only one twin of a proband could be claimed as a case of ulcerative colitis and he was one of the monozygotic group. This pair had only distal ulcerative colitis. From this one twin a heritability coefficient of 0.53 was calculated (with 95% confidence interval 0.24 to 0.82) and not 0.24 to 1.0 as stated by Satsangi *et al*. Thus, the 53% heritability claimed in both papers is based on one man with mild ulcerative colitis. The definition of his disease differed from that in the probands and the authors admit to deficiencies in their selection procedure. Tysk *et al* also admit that the prevalence of ulcerative colitis in the twins differs from that of the general population in Stockholm (mostly singletons). Twin studies cannot inform about disease in singletons if the prevalences are not the same. As the mean observation period was almost 20 years, it is unlikely that many more healthy twins will develop ulcerative colitis.[1] With 94% discordance in monozygotic twins, it is difficult to accept that most ulcerative colitis is inherited in either twins or singletons.

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1 Tysk C, Lindberg E, Jarnerot G, *et al*. Ulcerative colitis and Crohn's disease in an unselected population of monozygotic and dizygotic twins. A study of heritability and the influence of smoking. *Gut* 1988;**29**:990–6.

Reply

EDITOR,—We are grateful to Dr Harding for his comments, and we share his reservations regarding interpretation of twin studies in common diseases. Indeed, we have pointed out on a number of occasions that the genetic contribution in ulcerative colitis is less strong than that in Crohn's disease. Nevertheless, the model of disease inheritance which is most consistent with the epidemiological and molecular genetic data currently available is that Crohn's disease and ulcerative colitis are related polygenic disorders. The twin data are supported by epidemiological studies of familial prevalence from Scandinavia, the UK, and the USA. In recent years these epidemiological data have been supplemented by molecular genetic studies from a number of independent investigators. These studies have provided evidence for the presence of a number of genetic determinants of susceptibility and disease behaviour in ulcerative colitis. There is great optimism that these studies will result in an advance in the understanding of the pathogeneis of these common disorders. We stongly agree that both environmental as well as genetic factors are important in the pathogenesis of ulcerative colitis.

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Iron reduction therapy in hepatitis C

EDITOR,—Three papers in the July issue report on the possible association between iron and tissue damage in conditions other than haemochromatosis. Tan *et al* (*Gut* 1997;**41**:14–18) found that gastric cancer cells are more susceptible to photodynamic therapy when iron is removed. Boucher *et al* (*Gut* 1997;**41**:115–20) found that treatment of hepatitis C with interferon leads to a decrease in liver iron content. Bacon (*Gut* 1997;**41**:127–8) briefly commented on the association between iron and hepatitis C, including some evidence that iron depletion may be beneficial in patients who fail to respond to interferon α.

Shortly after Bacon *et al*'s pioneering report in 1993 on iron reduction therapy in hepatitis C,[1] and mainly because of a lack of any other option at the time, we started applying this form of therapy in our growing population of patients who had failed to respond to interferon and who had the unfavourable 1b genotype.

The simplest and cheapest way to reduce body iron stores is repeated drawing of one unit of whole blood (as for haemochromatosis). However, we encountered several unexpected difficulties in our attempts to implement a phlebotomy programme for patients with chronic hepatitis C. A small group of patients are reluctant to undergo repeated phlebotomy because of ethnic or psychological reasons (there is a belief that blood equals life and therefore that blood loss depletes the body of life giving power). A second group cannot tolerate blood loss because of recurrent episodes of faintness and presyncope. These patients need several hours' observation, a chaperone, and sometimes intravenous fluid before they can be discharged home. In a third group venous access is a problem. The

large bore needles of standard phlebotomy bags can be inserted into large straight veins only. Most female and some male patients do not have suitable veins and this problem becomes more serious with age. This obstacle can be overcome by improvisation with smaller bore needles but this has the disadvantage of slower flow, increased stasis and coagulation within the tubing, sometimes necessitating reinsertion of the iv line. A fourth impediment in a busy gastroenterology/hepatology department is lack of enthusiasm among nursing staff and a shortage of beds and space in the recovery room. It also seems unwise to place patients with chronic hepatitis C undergoing phlebotomy and large quantities of potentially hazardous blood alongside uninfected patients recovering from endoscopy. This also limits the time available for phlebotomy.

We devised a simple way to circumvent all of these difficulties: all new patients with chronic hepatitis C (unless they have an iron deficiency) are shown a 50 ml syringe as early as possible in their workup. They are told that from now on they should ask for all blood tests to be taken only with such syringes and that any surplus blood should be discarded with the syringe in the biohazard containers. This also applies when vacutainer tubes are used. As iron overload in these patients is not as great as in those with haemochromatosis, iron depletion can be accomplished in 20–40 phlebotomy sessions (a 50 ml syringe can contain ~70 ml blood). Thus iron reduction therapy is achieved more slowly than with conventional phlebotomy but is integrated into the routine workup and is accepted by both patients and staff alike.

We hope that our method may be useful to other clinicians in the field.

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1 Bacon BR, Rebholz AK, Fried MW, *et al.* Beneficial effect of iron reduction therapy in patients with chronic hepatitic C who failed to respond to interferon α. [abstract]. *Hepatology* 1993;**18**:150A.

NOTES

6th Southeast European Congress of Paediatric Surgery: Short Bowel Syndrome

The 6th Southeast European Congress of Paediatric Surgery: Short Bowel Syndrome will be held in Graz, Austria, on 22–23 May 1998. Further information from: Dr Günther Schimpl, Department of Paediatric Surgery, Auenbruggerplatz 34, A-8036 LKH-Graz, Austria. Tel: +43 316 385 3762; Fax: +43 316 385 3775.

9th British Association of Day Surgery Annual Scientific Meeting

The 9th British Association of Day Surgery Annual Scientific Meeting and Exhibition will be held at the Harrogate International Centre, Harrogate, UK, on 4–6 June 1998. Further information from: Kite Communications, The Silk Mill House, 196 Huddersfield Road, Meltham, West Yorkshire HD7 3AP, UK. Tel: 01484 854575; Fax: 01484 854 576; email: info@kitecomms.co.uk.

9th International Symposium on Cells of the Hepatic Sinusoid

The 9th International Symposium on Cells of the Hepatic Sinusoid will be held in Christchurch, New Zealand, from 27 September to 1 October 1998. Further information from: Professor Robin Fraser, I.S.C.H.S., Christchurch School of Medicine, PO Box 4345, Christchurch 8001, New Zealand. Tel: +64 3 3640 587; Fax: +64 3 3640 593; email: grogers@chmeds.ac.nz.

Growth Factors and Nutrients in Intestinal Health and Disease

An International Symposium on Growth Factors and Nutrients in Intestinal Health and Disease will be held at the Rihga Royal Hotel, Osaka, Japan, from 31 October to 3 November 1998. Further information from: Kinya Sando, MD, Department of Pediatric Surgery, Osaka University Medical School, 2-2 Yamadoaka, Suita, Osaka 565, Japan. Tel: +81 6 879 3753; Fax: +81 6 879 3759; email: gut@pedsurg.med.osaka-u.ac.jp.

XXXth Annual Meeting of the European Pancreatic Club

The XXXth Annual Meeting of the European Pancreatic Club will be held in Thessaloniki, Greece, from 10 to 13 June 1998. Further information from: Diastasi, Congress Secretariat, 30 Katsimidou Street, Thessaloniki 54639, Greece. Tel: 30 31 938 203 or 905 110; Fax: 30 31 909 269.

XXIIIth International Update on Liver Disease

The XXIIIth International Update on Liver Disease will be held at the Royal Free Hospital School of Medicine, London, UK, from 16 to 19 July 1998. Further information from: Professor Neil McIntyre, University Department of Medicine, Royal Free Hospital, Pond Street, London NW3 2QG, UK. Tel: 0171 794 0500 ext 3969; Fax: 0171 830 2321.

Hong Kong Academy of Medicine—First International Congress

The Hong Kong Academy of Medicine will be hosting its First International Congress from 26 to 29 November 1998 in commemoration of the grand opening on its new building. Further information from: Ms Colour Lee, Conference Manager or Miss Phoebe Wong, Administrative Assistant, Hong Kong Academy of Medicine, 9/F, Multicentre Block A, Pamela Youde Nethersole Eastern Hospital, 3 Lok Man Road, Chai Wan, Hong Kong. Tel: 852 2515 5755; Fax: 852 2505 3149; Email: hkam@hkam.org.hk.

Hepatic and Splanchnic Circulation in Health and Disease

An International Conference on Hepatic and Splanchnic Circulation in Health and Disease will be held in Inverness, Scotland, from 20 to 23 June 1999. The conference is designed to provide an international forum of discussion among those interested in the circulatory control of the liver and splanchnic region. Free communication (including keynote lectures) and poster sessions will cover: physiological control, endothelial function, innervation, ischaemia-reperfusion injury, inflammation, portal hypertension, transplantation. **Abstract deadline: 1 November 1998**. Further information from: Dr Robert T Mathie, Department of Gastrointestinal Surgery, Imperial College School of Medicine, Hammersmith Hospital, London, UK. Tel/Fax: 0181 383 2267; Email: rmathie@rpms.ac.uk; Internet: http://www.otago.ac.nz/inverness.

Falk Symposia and Workshops

The Symposium on New Aspects in Hepatology and Gastroenterology will be held in Tbilisi, Georgia, on 29 and 30 May 1998.

The Symposium on Advances in Inflammatory Bowel Diseases will be held in Brussels, Belgium, on 18–20 June 1998.

The Symposium on Diseases of the Liver and the Bile Ducts—New Aspects and Clinical Implications will be held in Prague, Czech Republic, on 12 and 13 June 1998.

The XV International Bile Acid Meeting: Bile Acids in Cholestasis will be held in Titisee, Germany, on 12 and 13 October 1998.

The Symposium on Colorectal Cancer: Molecular Mechanisms, Premalignant State and its Preventions will be held in Titisee, Germany, on 14 and 15 October 1998.

The Symposium on Intestinal Mucosa and its Diseases—Pathophysiology and Clinics will be held in Titisee, Germany, on 16 and 17 October 1998.

For further information on any of these symposia, please contact: Falk Foundation e.V.—Congress Division, Leinenweberstr. 5, PO Box 6529, D-79041 Freiburg, Germany. Tel: +49 761 130 340; Fax: +49 761 130 3459.

Clinical Training and Research Opportunities in Gastroenterology in Italy

The Società Italiana di Gastroenterologia (SIGE) have produced a booklet on clinical training and research opportunities in gastroenterology in Italy. Further information and a copy of the booklet are available from: SIGE Società Italiana di Gastroenterologia, Via Salvatore di Giacomo 66, 00142 Rome, Italy. Email: roma99sige@uni.net.