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.