Letters to the editor

Ultrasonographic findings in Crohn’s disease

Editor,—We read with interest the paper by Gasche et al (Gut 1999;44:112–117) on the accuracy of transabdominal ultrasound in the detection of complications in Crohn’s disease. The authors evaluated 33 patients with Crohn’s disease who had resective bowel surgery. The results were impressive: 87% sensitivity with 90% specificity in the diagnosis of enterocutaneous fistulas; 100% sensitivity with 92% specificity in the diagnosis of intra-abdominal abscesses; and 100% sensitivity with 91% specificity in the diagnosis of strictures. However, these data are in contrast with those reported by Schwerk and colleagues who found very low sensitivity (50%) with 95.5% specificity in ultrasound detection of enterocutaneous fistulas.

The difference in levels of sensitivity in these studies could be explained by the use of different standards and also, in our opinion, by varying definitions of fistulas. Gasche and colleagues considered fistulas to be any hypoechogenic peri-intestinal lesion measuring less than 2 cm. However, although this arbitrary cut off point may be useful to differentiate between fistulas and abscesses, it does not allow for precise differentiation between fistulas and strictures, for which we usually do not allow for precise differentiation. Previously, we considered 4 mm to be the pathological value of bowel wall thickness in patients with inflammatory bowel disease, but we now reduced this value to 3 mm or more, having excluded patients with polabulbar lesions or portal hypertension, in which bowel wall thickness is due to an oedematous m动us.

In conclusion, the diagnostic accuracy of transabdominal ultrasound has improved progressively as found in the literature are due principally to the introduction of new technologies, the level of experience of the operators, and the growing interest in the application of ultrasound to the study of the digestive tract.

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Association between colon cancer and adenocarcinoma of the oesophagus

Editor,—Recently, Lagergren and Nyren (Gut 1999;44:819–821) concluded that results from a population based cohort study in Sweden did not support a common link between colon cancer and oesophageal adeno- carcino ma. However, there is consistent evidence that low intake of dietary fibre is associated with both diseases. In an analysis of 13 case control studies involving more than 5000 colorectal cancer cases, Howe and colleagues reported an inverse association between fibre intake and colorectal cancer in 12 of the 13 studies, and an odds ratio of 0.53 (95% confidence interval 0.47 to 0.61) for the highest quintile of fibre intake compared with the lowest, in a meta-analysis. Similarly, four case control studies have reported a significant inverse association between fibre intake and adenocarcinoma of the oesophagus and gastric cardia (table 1).

In contrast, two studies which included cases of squamous cell carcinoma found no significant link between fibre intake and squamous cell carcinoma of the oesophagus.

Clearly, the dramatic increase in the incidence of adenocarcinoma of the oesophagus in the USA and parts of Europe over past decades cannot be explained by secular trends in dietary fibre consumption. A more plausible explanation links increased rates of the disease to increases in the prevalence of obesity. This view is supported by evidence from observational studies that suggests that both overweight and symptomatic gastro-oesophageal reflux are linked to increased risk of oesophageal adenocarcinoma. Possible mechanisms for the observed protective effect of dietary fibre include the mechanical cleaning effect of the lower oesophageal mucosa, increased motility of potential carcinogens across the gastro-oesophageal junc-

Table 1 Dietary fibre intake and adenocarcinoma of the oesophagus and gastric cardia

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<tr>
<th>Reference</th>
<th>Country</th>
<th>Sites</th>
<th>Comparison</th>
<th>Odds ratio</th>
<th>95% CI</th>
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*95% confidence interval (CI) does not include 1.0. †Multiple logistic regression model.

OGC, oesophagus and gastric cardia; O, oesophagus.
The primary hypothesis of our population based cohort study was not that colon cancer would subsequently develop into oesophageal cancer, but rather that there might be a common underlying link between the occurrence to these two tumours, independent of the time latency in their development. Hence, the individual follow up latency after colon cancer diagnosis was of minor importance. Therefore, it would seem reasonable to analyze the exposure to the critical underlying factors, for example, insufficient dietary intake of fibre, and the development of oesophageal or colonic adenocarcinoma. Therefore, as long as selection or ascertainment biases are deemed to be small, the time period that follows immediately after diagnosis of colon cancer is the most informative. The total number of person years was more critical, and we were able to follow up more than 500 000 person years in our study. The rarity of oesophageal adenocarcinoma is a problem in any study of the aetiology of this tumour in any country, particularly if the studied exposure is relatively weak. This problem explains our limited power to exclude a weak association. Nevertheless, we were able to identify more than 100 000 people with verified colon cancer and to follow them for subsequent cancer development. Thus, there is a substantial number of exposed people. We agree that case control studies are generally more efficient than cohort studies when rare outcomes are to be investigated. However, in the case of our register based retrospective cohort study, a case control approach would not entail any advantage, as our cohort contained all individuals exposed to colon cancer in Sweden during the period 1958 and 1992, and all individuals among them who developed oesophageal adenocarcinoma during the same period. A case control study conducted in Sweden during this period would, at best, include the same number of exposed oesophageal adenocarcinoma cases as in our cohort study. Thus, the problem with low statistical power is not owing to study design, but that the study base (all residents of Sweden 1958–1992) was too small to generate a sufficient number of individuals with the combination of colon cancer and oesophageal adenocarcinoma.

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Reply

Editor,—The influence of dietary fibre on the risk of adenocarcinoma of the oesophagus and gastric cardia is certainly interesting, but further and larger studies are needed before a link between the two can be confirmed. Although several case control studies have reported a correlation between colon cancer and fibre intake, others have failed to identify any association. Therefore, the positive finding in the Lagergren and Nyrén study is not surprising, as the average year of entry to the cohort study was 1977 and median follow up was 2.1 years. Thus, a substantial proportion of the accumulated person years relates to a time period when the Swedish population was at a very low risk of developing oesophageal adenocarcinoma after a diagnosis of colon cancer remains small, because of the late onset of colon cancer. Furthermore, case control studies are likely to continue to be the most efficient type of observational study design for the investigation of possible common links between these two diseases.

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Quality of life of parents of children on home parenteral nutrition

Editor,—Jeppesen and colleagues (Gut 1999;44:844–852) found that children receiving home parenteral nutrition (HPN) had a poorer quality of life than healthy children. However, the non-specific impact of total parenteral nutrition (TPN) was not measured. We recently completed a study to assess the quality of life of 49 patients receiving home parenteral nutrition (HPN). They found a significant reduction in the quality of life of these patients compared with patients with ana- tomically or functionally short bowel not receiving HPN.

Letters, Notes
We showed recently that having a child on HPN has a major impact on the quality of life of the parents. We studied 11 parents of children with chronic intestinal failure requiring HPN. Following an initial focus group meeting to identify important issues, semi-structured interviews were held with the parents. The General Health Questionnaire (GHQ-28) and a questionnaire developed for the British Artificial Nutrition Survey (BANS) were also administered. A control group of 11 parents with age matched healthy children also answered the BANS questionnaire.

The GHQ-28 showed that seven of the 11 parents with children on HPN exceeded the threshold for psychiatric morbidity. The BANS described a significant deterioration before and after the child’s illness for social life (p<0.007), family life (p<0.007), sex life (p<0.003), and work (p<0.004) in these parents compared with controls. Parents caring for children on HPN were also more likely to be physically tired and to have difficulties in taking holidays, going shopping and spending time with their partners. Many of them admitted to feeling frustrated, angry, stressed, and having problems sleeping.

With the advent of HPN, increasing numbers of children with chronic intestinal failure are now being managed at home. Although HPN has given life to many of these children who would otherwise have died, the burden of care on these parents is enormous and could have a significant impact on their quality of life. Health care professionals should be aware of this problem and endeavour to offer the necessary support for families who provide this demanding type of care. The services of a dedicated community nutritional support team is recommended.

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*Helicobacter pylori* infection and autoimmune pathogenesis of gastric neoplasia

EDITOR—We read with great interest the article by Kawahara et al (Gut 1999;48:20–23) reporting the increase of antibody titres to HGC-27 cells in *Helicobacter pylori* positive patients with mucosa associated lymphoid tissue (MALT) lymphoma when compared with titres in patients with other gastroduodenal diseases and in healthy subjects. Previously, other authors1 showed that antigenic mimicry between *H pylori* and the host mucosa may induce autoimmune responses which lead to the development of the disease.

Recently, we have diagnosed a few cases of synchronous gastric adenocarcinoma and low grade MALT lymphoma (unpublished data). Although the development of simultaneous primary gastric lymphoma and carcinoma is a rare event, in view of Kawahara et al’s data we think that the occurrence of both pathologies could be underestimated. In fact, the gastric glandular epithelium present inside a MALT lymphoma might be susceptible to neoplastic transformation, owing to either the presence of common oncogenic factors or to the induction of immune responses to host components. The latter mechanism may lead to tissue injury of an autoimmune nature. The possibility of coexisting MALT lymphoma and gastric adenocarcinoma should be kept in mind, especially in patients infected with *H pylori* as an aetopathogenic role for this bacterium in both diseases has been postulated.

*H pylori* plays a key role in the natural history of gastric MALT lymphoma and represents an example of antigen mediated tissue stimulation and lymphoproliferation, with possible subsequent lymphomagenesis. We agree with Kawahara et al that undefined bacterial components or the host immune response to the bacterial infection could promote autoimmune responses to host antigen in certain subjects. Further studies are needed to clarify the role of antibodies to Hsp60 and HGC-27, but it is possible that other as yet unidentified antibodies may also be involved.

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11th Annual International Colorectal Disease Symposium

The 11th Annual International Colorectal Disease Symposium will be held at the Marriott Harbor Beach Resort, Fort Lauderdale, Florida, USA, on 17–19 February 2000. Further information from: Cleveland Clinic Florida, Department of Continuing Education, 2950 West Cypress Creek Road, Fort Lauderdale, Florida 33309, USA. Tel: +1 954 978 5056; fax: +1 954 978 5539; email: jagemls@cclf.org

5th World Congress on Trauma, Shock, Inflammation, and Sepsis

The 5th World Congress on Trauma, Shock, Inflammation, and Sepsis will be held in Munich, Germany, from 29 February to 4 March 2000. Further information from: Prof Eugen Faist, Department of Surgery, Ludwig Maximilians University Munich, Klinikum Grosshadern, Marchioninistrasse 15, 81377 Munich, Germany. Tel: +49 89 7095 5461/2461; fax: +49 89 7095 2460; email: faist@ghch.med.uni-muenchen.de

Second Annual Gastrointestinal Cancer Update: A Multidisciplinary Approach

The Second Annual Gastrointestinal Cancer Update conference will be held at the Yarrow Hotel and Conference Centre, Park City, Utah, USA, on 15–19 March 2000. Further information from: Rosalie Lammle. Tel: +1 801 581 8664; fax: +1 801 581 3647; email: rosalie.lammle@hsc.utah.edu

European Courses on Laparoscopic Surgery

The European Courses on Laparoscopic Surgery will be held at the University Hospital Saint Pierre, Brussels, Belgium, on 4–7 April 2000 and 21–24 November 2000. Further information from: Conference Services S.A., Drève des Tumuli, 18, B-1170 Brussels, Belgium. Tel: +32 2 375 1648; fax: +32 2 375 3299; email: conference.services@skynet.be

Third Scandinavian Course on Inflammatory Bowel Diseases

The Third Scandinavian Course on Inflammatory Bowel Diseases will be held at the Wilandersgren, Orebro Medical Centre, Orebro, Sweden, on 12–14 April 2000. Further information from: Kurskliniken, Regionsjukhuset, S-701 85 Orebro, Sweden. Tel: +46 19 15 37 05; fax: +46 19 15 37 95.

XVIIIth European Workshop on Gastroenterology and Endotherapy

The XVIIIth European Workshop on Gastroenterology and Endotherapy will be held in Brussels, Belgium, on 26–28 April 2000. Further information from: Administrative Secretariat, Ms Nancy Beauprez, Gastroenterology Department, Erasme Hospital Route de Leuven 808, B-1070 Brussels, Belgium. Tel: +32 2 555 4900; fax: +32 2 555 4901; email: beauprez@ulb.ac.be

Digestive Week

The Digestive Week will be held at the San Diego Convention Centre, San Diego, California, USA, on 21–24 May 2000. Further information from: DDW Administration, 7910 Woodmont Avenue, 7th Floor, Bethesda, Maryland 20814, USA. Tel: +1 301 272 0022; fax: +1 301 654 3978; website: www.ddw.org

International Hepato-Pancreato-Biliary Association 4th World Congress

The International Hepato-Pancreato-Biliary Association 4th World Congress will be held in Brisbane, Australia, from 28 May to 1 June 2000. Further information from: Intermedia Convention and Event Management, PO Box 1280 (Intermedia House, 11/97 Castlemaine Street), Milton, Queensland 4064, Australia. Tel: +61 (07) 3369 0477; fax: +61 (07) 3369 4512; email: hpb2000@im.com.au
Helicobacter pylori infection and autoimmune pathogenesis of gastric neoplasias

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