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 fistulae; 100% sensitivity with 92% specificity in the diagnosis of intra-abdominal abscesses; and 100% sensitivity with 91% specificity in the diagnosis of stric- tures. However, these data are in contrast with those reported by Di Candio and colleagues (J Gastroenterol Hepatol 1999;14:395–9). In our experience the accuracy of ultrasound is dependent principally on the use of revised definitions for these entities. Gasche et al considered fistulas to be any hypoechoic peri-intestinal lesion measuring more than 2 cm. However, in our opinion, fistulas are defined as the presence of a communication between intra-abdominal and parietal abscesses. We agree that abscesses located in the retroperitoneal and perianal regions have a prognostic value. We found that patients with Crohn's disease with a bowel wall thickness greater than 6 mm, who are in clinical remission, showed a significantly higher relapse rate (90%) in the subsequent 12 months, compared with patients with bowel wall thickness of less than 6 mm (40%).

In conclusion, the diagnostic accuracy of transabdominal ultrasound has improved progressively as seen in the literature. Therefore, it is of interest in the application of ultrasound to the study of the digestive tract.

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Reply

EDITOR,—We thank Dr Arienti and colleagues for their attention to our work. It is correct that improved technology and operator experience alone do not explain our better results. Indeed, the high accuracy of transabdominal bowel sonography in our study is based principally on the use of revised definitions for the detection of intestinal complications. It is, therefore, a pleasure to have consensus on these definitions.

Despite some unresolved issues, many (mostly European) investigators have shown the value of bowel sonography in patients with Crohn's disease. The time is ripe to offer the benefits of this imaging method to patients with Crohn's disease worldwide.

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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 adenocarcinoma. 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 pooled analysis.1 Similarly, four case control studies have reported a significant inverse association between fibre intake and adenocarcinoma of the oesophagus and gastric cardia (table 1).2,3,4,5

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.6,7 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.8 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 adenocarcinoma of the oesophagus.9 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>
<thead>
<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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<tr>
<td>1</td>
<td>USA</td>
<td>OGC</td>
<td>Highest of lowest quartile</td>
<td>0.3</td>
<td>0.1 to 0.7</td>
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<td>2</td>
<td>USA</td>
<td>OGC</td>
<td>Lowest of lowest quartile</td>
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<td>0.1 to 0.8</td>
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<tr>
<td>3</td>
<td>USA</td>
<td>O</td>
<td>Highest of lowest quartile</td>
<td>0.4*</td>
<td>Not stated</td>
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<tr>
<td>4</td>
<td>Greece</td>
<td>O</td>
<td>Marginal quintile</td>
<td>0.74</td>
<td>0.55 to 0.99</td>
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Odds ratios adjusted for alcohol and tobacco use.
*95% confidence interval (CI) does not include 1.0. | Multiple logistic regression model.
OGC, oesophagus and gastric cardia; O, oesophagus.
tion, and the antioxidant effect of micronutri-
ents in fruits and vegetables.1 The lack of a significant link between colorectal cancer and oesophageal cancer 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 oesophag-
al adenocarcinoma. As the authors indi-
cated, the limited power of the study meant that they were unable to exclude the possi-
bility of a doubled risk. However, despite recent increases in incidence, the relative risk, even if elevated, of developing oesophageal adenocarcinoma after a diagnosis of colon cancer remains small, because of the late onset of colon can-
cer. Furthermore, case control studies are likely to continue to be the most efficient type of observational study design for the investi-
gation of possible common links between these two diseases.

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 underlining 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 reason-
able to assume that theses differences in 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 fol-
lows 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 rate of oesophageal adenocarcinoma is a problem in any study of the aetiology of this tumour in any country, particularly if the studied expo-
sure is relatively varied. 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 sub-
sequent cancer diagnosis. This is a sub-
stantial 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 1977-92, and all individuals among them who devel-
oped 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 resi-
idents of Sweden 1958–1992) was too small to generate a sufficient number of individuals with the combination of colon cancer and oesophageal adenocarcinoma.

reply

Editor,—The influence of dietary fibre on the risk of adenocarcinoma of the oesophagus and gastric cancer 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 reached the conclusion between colon cancer and fibre intake, others have failed to identify such a link.1 Hence, the suggested link between fibre intake and both colon cancer and oesophageal cancer is debatable.

Further changes in dietary fibre con-
sumption cannot explain the increasing inci-
dence of oesophageal adenocarcinoma. The increasing prevalence of obesity is a possible reason for this rise, but some seemingly incongruent observations need to be con-
celled before this hypothesis can be verified.1 The apparently sudden deflection of the inci-
dence curve for oesophageal adenocarcinom-
a, the rapidity of the increase,1 and the noticeable (6–8 fold) increase that do not entirely support this interpretation.

2 Kabat GC, Ng SK, Wynder EL. Tobacco, alco-
hol intake, and diet in relation to adenocarci-
nomas of the esophagus and gastric cancer. Can-
cer Causes Control 1993;4:123–32.
3 Zhang ZF, Kurtz RC, Yu GP, et al. Adenocarci-


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Quality of life of parents of children on home parenteral nutrition

Editor,—Jeppesen and colleagues (Gut 1999;44:814–825) studied a non-disease specific sickness impact profile (SIP) and the disease specific inflammatory bowel disease questionnaire (IBDQ) 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 receiv-
ing HPN.
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, HPN has given life to many of these children who would otherwise have died,1,2 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 nutrition support team is recommended.

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NOTE

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 gastro-duodenal diseases and in healthy subjects. Previously, other authors1,2 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 aetiological 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 Hp60 and HG27, but it is possible hypothesis that other as yet unidentified antibodies may also be involved.

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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 Vilandersele, Örebro Medical Centre, Örebro, Sweden, on 12–14 April 2000. Further information from: Kurskännet, Regionsjukhuset, S-701 85 Örebro, 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 Lennik 808, B-1070 Brussels, Belgium. Tel: +32 2 555 4900; fax: +32 2 555 4901; email: beauprez@ulb.ac.be

Digestive Disease Week

The Digestive Disease 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: International Convention and Event Management, PO Box 1280 (Intermediate 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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