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 entero-enteric 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 Maconi and colleagues[1] who found very low sensitivity (50%) with 95.5% specificity in ultrasound detection of entero-enteric 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 hypoechoic 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 adopt a cut off criterion of a diameter of less than 1 cm.

Strictures and abscesses are often considered to be more easily detectable by ultrasound than fistulas, but contrasting data exist even on this point. Gasche *et al* found 100% sensitivity for intra-abdominal abscesses, whereas in contrast Maconi *et al* found an overall sensitivity of 83.3%, with only 66.6% for intra-abdominal abscesses. Schwerk and colleagues[2] found levels of sensitivity for parietal and intra-abdominal abscesses that were similar to those of Gasche *et al*, although they emphasised a lower sensitivity for ultrasound in detecting retroperitoneal and perianal lesions. We agree that abscesses located in the small pelvis or in the pararectal space are the most difficult to detect, although intra-abdominal and parietal abscesses are easily recognised.

Finally, we agree with Gasche *et al* on the accuracy of ultrasound in detecting strictures; they reported 100% sensitivity and 91% specificity, with bowel wall thickening of at least 3 mm. Different values of bowel wall thickening have been considered to be pathological, which is probably due to the use of different type of probes and to operator experience. Di Candio and Sheridan[3 4] defined bowel wall thickness of greater than 5 mm as pathological, whereas Maconi and Schwerk[1 2] considered wall thickening of 4 mm or more to be abnormal. Hata and colleagues[5] reported that the mean overall wall thickness of normal bowel specimens was 2.8 mm and that no normal specimens exceeded 4 mm in thickness. More recently other studies by Solvig, Van Oostayen, and even Gasche defined bowel wall thickening of 3 mm or more as pathological.[6 7]

Previously,[8] we considered 4 mm to be the pathological value of bowel wall thickness in patients with inflammatory bowel disease, but we have now reduced this value to 3 mm or more,[9] having excluded patients with ipoalbuminaemia or portal hypertension, in which bowel wall thickness is due to an oedematous imbibition. Recently, we conducted a prospective study (unpublished data) in which bowel wall thickness was shown to 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 18 months, when compared with patients with bowel wall thickness of less than 6 mm (40%).

In conclusion, the diagnostic accuracy of transabdominal ultrasound has improved progressively and the differences 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.

V ARIENTI
L ZAMBONI
Department of Internal Medicine, Ospedale Maggiore C.A. Pizzardi, 40133 Bologna, Italy

P GIONCHETTI
F RIZZELLO
M CAMPIERI
Department of Internal Medicine, Policlinico S. Orsola, 40138 Bologna, Italy

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

Reference	*Country*	*Sites*	*Comparison*	*Odds ratio*	*95% CI*
2	USA	OGC	Highest of lowest quartile	0.3	0.1 to 0.7
3	USA	OGC	Highest of lowest quartile	0.3	0.1 to 0.8
4	USA	O	Highest of lowest quartile	0.4*	Not stated
5	Greece	O	Marginal quintile†	0.74	0.55 to 0.99

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.

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.

C GASCHE
Department of Medicine, Division of Gastroenterology, University of California, San Diego, La Jolla, CA 92093-0688, USA

Association between colon cancer and adenocarcinoma of the oesophagus

EDITOR,—Recently, Lagergren and Nyrén (*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 risk of 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 the combined 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).[2–5] 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.[2 5]

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.[6] 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.[4 7] 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-

tion, and the antioxidant effect of micronutrients in fruits and vegetables.[3]

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 oesophageal adenocarcinoma. As the authors indicated, the limited power of the study meant that they were unable to exclude the possibility of a doubled risk.

However, despite recent increases in incidence, the lifetime risk, even if elevated, 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.

R MARIC
K K CHENG
Department of Public Health and Epidemiology
University of Birmingham, Edgbaston
Birmingham B15 2TT, UK

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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 revealed an association 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.

We agree that changes in dietary fibre consumption cannot explain the increasing incidence of oesophageal adenocarcinoma. The increasing prevalence of obesity is a possible reason for this rise, but some seemingly incongruent observations need to be reconciled before this hypothesis can be verified.[2] The apparently sudden deflection of the incidence curve for oesophageal adenocarcinoma,[3] the rapidity of the increase,[4] and the noticeable (6–8 fold) male predominance,[4] do not entirely support this interpretation.

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 between their development. Hence, the individual follow up latency after colon cancer diagnosis was of minor importance. Therefore, it would seem reasonable to assume similar latencies between exposure to the critical underlying factor—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 uncommon. 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; this 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 between 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.

J LAGERGREN
P NYREN
Division of Surgery,
Karolinska Institutet,
Danderyd Hospital,
S-182 88 Danderyd, Sweden

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Diversion colitis as a trigger for ulcerative colitis

EDITOR,—Lim and colleagues recently presented three cases of diversion colitis which seemed to act as a trigger for instream ulcerative colitis (*Gut* 1999;**44**:279–282). We would like to present a fourth case which has recently come to our attention and which adds to the literature.

In 1994, a 75 year old woman presented with a five year history of faecal soiling and urinary incontinence. A former smoker of 50 years, the patient had had a traumatic forceps delivery and episiotomy when she was 30 years old. Her mother had a history of unspecified colitis. Rigid sigmoidoscopy and barium enema were normal, but anorectal ultrasound showed defects of the internal and external sphincters. Anterolateral repair of the anal sphincter did not control the symptoms and in September 1997 an end colostomy was performed for her disabling faecal incontinence.

In July 1998, the patient presented with blood and mucus per rectum, and an anterior mucosal prolapse was diagnosed and repaired. However, her symptoms persisted and in October 1998 flexible sigmoidoscopy showed a granular, congested, and oedematous mucosa with contact bleeding throughout the rectosigmoid stump. Histology showed a mixed inflammatory cell infiltrate with distortion of the crypt architecture and cryptitis, and a diagnosis of diversion colitis was made; the rectal symptoms responded quickly to topical steroid enemas.

Four months later, the patient developed increased stomal frequency and bleeding into the stoma bag. Colonoscopic examination of the instream colon, via the colostomy, revealed an active distal colitis with a granular, oedematous, congested, and friable mucosa. Histology showed a mixed inflammatory cell infiltrate in the lamina propria with cryptitis, crypt abscess formation, and a reduction in the number of goblet cells. Ulcerative colitis was diagnosed and treated with oral mesalazine and topical steroid enema per stoma. Symptoms quickly improved and there has been no further bleeding from the rectum or stoma.

Although the histological features of ulcerative colitis and diversion colitis are indistinguishable, the clinical history in this case suggests that ulcerative colitis developed after true diversion colitis. It is possible that this patient's colitic process represented an idiopathic ulcerative colitis, but it seems much more likely that the colostomy with faecal diversion was the initiating factor and that, as speculated in the cases studied by Lim and colleagues, diversion colitis is a risk factor for ulcerative colitis. Hypotheses on the pathogenesis of both diversion colitis and ulcerative colitis should take into account cases such as these.

S L JOWETT
I COBDEN
Department of Gastroenterology,
North Tyneside General Hospital, Rake Lane,
North Shields, Tyne and Wear NE29 8NH, UK

Quality of life of parents of children on home parenteral nutrition

EDITOR,—Jeppesen and colleagues (*Gut* 1999;**44**:844–852) used the 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 anatomical or functional short bowel not receiving 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, annoyed, 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,[1] 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.

C WONG
A K AKOBENG
V MILLER
A G THOMAS
Department of Paediatric Gastroenterology, Booth Hall Children's Hospital, Charlestown Road, Blackley, Manchester M9 7AA, UK

Correspondence to: Dr Thomas.

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Helicobacter pylori infection and autoimmune pathogenesis of gastric neoplasias

EDITOR,—We read with great interest the article by Kawahara *et al* (*Gut* 1999;**45**: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 authors[1] 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 aetiopathogenic 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 hypothesise that other as yet unidentified antibodies may also be involved.

G CAMMAROTA
G GASBARRINI
Policlinico "A. Gemelli", Università Cattolica del Sacro Cuore, Istituto di Medicina Interna e Geriatria, Largo A. Gemelli, 8, 00168 Rome, Italy

Correspondence to: Dr Giovanni Cammarota. email: dora.erasmo@tiscalinet.it

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NOTES

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: jagelms@ccf.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@gch.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 Wilanderselen, Örebro Medical Centre, Örebro, Sweden, on 12–14 April 2000. Further information from: Kurskansliet, 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: Intermedia Convention and Event Management. PO Box 1280 (Intermedia House, 11/97 Castlemaine Street), Milton, Queensland 4064, Australia. Tel: +61 (0)7 3369 0477; fax: +61 (0)7 3369 1512; email: hpb2000@im.com.au