5 mm as pathological, whereas Maconi and Schwerk 7 considered wall thickening of 4 mm or more to be abnormal. Hata and colleagues 8 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. 9

Previously, 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, having excluded patients with psoas bursa or ileal hypertrophy, 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 Recently, we conducted a prospective study (unpublished data) in which bowel wall thickness was shown to have a prognostic value.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, 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 is now 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 10 (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 0.64) for the highest quintile of fibre intake compared with the lowest, in a pooled analysis. 11 Similarly, four case control studies have reported a significant inverse association between fibre intake and adenocarcinoma of the oesophagus and gastric cardia (table 1). 12

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. 13

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. 14 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 juncture.
tion, and the antioxidative effect of micronutri-
ents in fruits and vegetables.

The lack of a significant link between colorectal cancer and oesophageal cancer in the Lagergren and Nyren 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 oesopha-
gal adenocarcinoma. As the authors indi-
cated, the limited power of the study meant that they were unable to exclude the possi-
bility of a double risk.

However, despite recent increases in inci-
dence, the 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.

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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 reported a relation 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 con-
msumption cannot explain the increasing inci-
dence of oesophageal adenocarcinoma. The increasing prevalence of obesity is a possible reason for this rise, but some seemingly inconsistent observations need to be con-
ciled before this hypothesis can be verified.2 The apparently sudden deflection of the inci-
dence curve for oesophageal adenocarcinoma,3 the rapidity of the increase, and the noticeable (6–8 fold) rise in incidence, 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 of 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 rea-
sonable to analyze differences 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 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 ratio of oesophageal adenocarcinoma is a problem in any study of the aetiology of this tumour in any country, particularly if the studied expo-
se is relatively rare. This problem explains our limited power to exclude a weak association. Nevertheless, we were able to identify more than 100 000 people with veri-
fid colon cancer and to follow them for sub-
sequent cancer. Therefore, there 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 years 1958 and 1992, and all individuals among them who devel-
opied 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-
dents 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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90.
3 Pera M, Cameron AJ, Trastek VF, et al. Increasing incidence of adenocarcinoma of the esophagus and esophagogastric junction. Gastroen-

Divergion colitis as a trigger for ulcerative colitis

Editor,—Lim and colleagues recently pre-
sentted three cases of diversion colitis which seemed to act as a trigger for instream ulcer-
aver colitis (Gut 1999;44:279–282). We would like to present a fourth case which has

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 and one sister suffered from ulcerative 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 re-
paired. However, her symptoms persisted and in October 1998 flexible sigmoidoscopy showed a granular, congested, and oedema-
tous mucosa with contact bleeding through-
out the rectosigmoid segment. 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 steroids.

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 granular, oedematous, congested, and friable mucosa. Histology showed a mixed inflam-
matory cell infiltrate in the lamina propria with cryptitis, crypt abscess formation, and a reduction in the number of goblet cell. Ulcerative colitis was diagnosed and treated with oral mesalazine and topical steroid enema per stoma. Symptoms quickly im-
proved and there has been no further bleeding from the rectum or stoma.

Although the histological features of ulcer-
aver colitis and diversion colitis are indistin-
guishable, 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 idio-
pathic 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 patho-
genesis of both diversion colitis and ulcerative colitis should take into account cases such as these.

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 qu-

nenaire (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.

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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 new 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.

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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 authors1 shown 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 aetipathogenic 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 antigens in certain subjects. Further studies are needed to clarify the role of antibodies to Hp60 and HGC-27, but it is possible hypothesis that other yet undefined antibodies may also be involved.

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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

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 Wilandersplatsen, Örebro Medical Centre, Örebro, Sweden, on 12–14 April 2000. Further information from: Karolinska Institute, Regionssjukhuset, 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 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 (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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