LETTERS TO THE EDITOR

Cholecystectomy and bowel function

Editor,—I read with great interest the article by Hearing et al (Gut 1999;45:889–894) on the effect of cholecystectomy on bowel function. In this elegant publication, however, the authors mistakenly assume that published estimates of the incidence of diarrhoea after postcholecystectomy diar-rhoea derive from retrospective or uncontrolled data only. In this context I would like to draw attention to earlier publications derived from the Rotterdam Gallstone Study. 1

In the first paper the results are discussed of a prospective analysis of biliary and gastrointestinal symptoms (including diarrhoea) prior to and up to two years after gall stone therapy. The therapy consisted of either conventional cholecystectomy or extracorporeal shock wave lithotripsy (ESWL), allocated randomly. The second paper focused on surgery and reported on symptoms before and after conventional and laparoscopic cholecystectomy. 2 This study was based on the same concept, and treatment depended on the availability of a laparoscopic set. Generally, we found that the reported incidence of diarrhoea before and after surgery did not change. In fact, there was no difference in the reported incidence of diarrhoea at any time between cholecystectomy and gall bladder preserving therapy (that is, ESWL). We also found that there were no differences in the reported incidence or severity of diarrhoea between laparoscopic and conventional cholecystectomy at any time.

Although the study design of our two studies differed largely from that of Hearing’s, the results and conclusions are in agreement, in that clinical diarrhoea seldom develops after cholecystectomy. O’Donnell is correct that objective assessments of therapy demonstrate new onset diarrhoea after cholecystectomy. 3 I agree with Hearing et al that postcholecystectomy diarrhoea is in fact an unproved entity. Given our and Hearing’s results, I doubt if more prospective studies are needed to solve this problem.

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MALT lymphomas and Helicobacter pylori?

Editor,—Raderer et al (Gut 2000;46:133–5) present an interesting case report of a patient with a mucosa associated lymphoid tissue (MALT) lymphoma of the stomach and descending colon. Their report adds to the growing literature of gastrointestinal MALT lymphomas that respond to antibiotic treatment. In addition to the numerous reports on antibiotic sensitive gastric lymphomas of the small intestine, salivary glands, nasal mucosa, and colon 4–7 have recently been reported. Although Helicobacter pylori is generally implicated as the inducing agent, this does not always appear to be the case. Further related bacterium, H helminthiatis, has also been found in association with gastric MALT lymphomas, including H pylori negative patients whose disease was still responsive to antibiotic treatment. 4 Furthermore, other non-H pylori bacterial and protozoal 8 flora have been observed in gastric lymphomas specific to involved regions. In the report by Raderer et al, and in several of the cases previously mentioned, 9 H pylori was not identified in the extragastric lesions, leaving it open to speculation how H pylori may induce antigenic stimulation of these lymphomas. Moreover, in the report by Inoue and Chiba, 10 not only was the rectal lesion H pylori negative but upper gastrointestinal endoscopy was normal. Their patient was seronegative for H pylori and had a negative rapid urease test, culture, and histological examination. In light of this evidence, it seems that although H pylori may be the most common cause of many gastrointestinal MALT lymphomas, it is not the only causative organism. This is an important consideration when confronted with patients diagnosed with H pylori negative MALT lymphomas.

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Reply

Editor,—Hopton Cann et al again point out the fact that Helicobacter pylori is not the sole cause of mucosa associated lymphoid tissue (MALT) lymphoma in some cases. They also suggest other, as yet undefined, bacterial/ infectious causes in MALT lymphomas in extragastric sites in view of our case and the recent literature. While we believe this to be a valid point and agree with the already established notion of other contributing factors in addition to H pylori, we nevertheless advise that our findings should be interpreted with caution. In contrast with other cases reported in the literature and cited by the authors, our patient suffered from concurrent gastric and colonic MALT lymphoma and had evidence of H pylori infection. Thus one cannot rule out the fact that antigenic shedding of H pylori from the stomach throughout the gastrointestinal tract or the presence of specific T cells alone was able to provide the colonic lesion with an antigenic drive needed for maintenance of the lymphoma. In this scena-rio, one would expect eradication of H pylori to lead to regression of the (still antigen and/or T cell dependent) lymphoma. The fact that various (apparently not H pylori related) intestinal as well as extraintestinal lesions regressed with antibiotic treatment is indeed highly sug-gestive of an underlying infectious process but does not necessarily constitute proof of this assumption, as direct antiproflllerative mecha-nisms of various antibiotics, including clar-thromycin, have been reported in different set-tings. Further investigations are needed before definite recommendations for (as yet empiri-cal) antibiotic therapy in patients with extra-gastric MALT lymphomas can be given.

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Guidelines for the management of iron deficiency anaemia

Editor,—It is somewhat self contradictory to suggest that “a transferrin saturation of <30% may help the diagnosis” if there is still doubt about validation of iron deficiency after receipt of the serum ferritin result, the authors having previously acknowledged that the latter is “the most powerful test for iron deficiency” (Gut 2000;46(suppl IV):v1–5). Statistical considerations which dictate that serum ferritin will always outrank transferrin, in terms of predictive power have their basis in the compari-sion between the receiver operating characteristic (ROC) curves for serum ferritin versus transferrin saturation, yielding values of 0.91 versus 0.71 (p<0.001) for the area under the curve. 1 Statistical considerations also dictate acknowledgement of mean corpuscular haemoglobin (MCH) as a predictive entity in its own right following documentation than an MCH of <27 pg was superior to a mean corpuscular volume (MCV) of <77 fl in predicting serum ferritin levels of <20 µg/l. All low MCV values had low MCH values but nine hypoferritinaemic patients with low MCH had MCV within the normal range.

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In my study, comprising 201 subjects with iron deficiency (characterised by serum ferritin <18 μg/l), the MCH was <24 pg, and this yielded a positive predictive value of 70%. By contrast, for MCV, optimum trade off between sensitivity (65.2%) and specificity (65.9%) for iron deficiency was <24 pg, and this yielded a positive predictive value of 55%. Correspondingly, the optimum MCH was either <24 pg, characterised by sensitivities, specificities, and positive predictive values of 74%, 59%, and 80%, respectively, or <23 pg, characterised by specificities, sensitivities, and positive predictive values of 58%, 75%, and 62%, respectively.

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Reply

EDITOR—Suggesting both that transferrin satiuration may help in the diagnosis and that ferritin is the most powerful test for iron deficiency anaemia (IDA) is not contradictory. Being the most powerful test does not mean it is always reliable. For example, in inflammatory conditions such as rheumatoid arthritis, ferritin may be normal even if there is iron deficiency.

We find the reference to the greater reliability of mean corpuscular haemoglobin (MCH) compared with mean corpuscular volume (MCV) in diagnosing IDA interesting. We agree that MCH can be useful in the diagnosis of iron deficiency. However, none of the papers quoted takes account of the red cell distribution width (RDW). We wonder if Dr Jolobe would still be able to demonstrate the superiority of MCH compared with MCV if anaemic patients with a normal MCV but raised RDW were excluded. We explain in our guidelines that combined deficiency (that is, iron deficiency together with B12 and/or folate deficiency) may be associated with a normal MCV and may be recognised by a raised RDW.

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Gastroenterology and Endotherapy: XIXth European Workshop

This course, to introduce the experienced gastroenterologist to the growing field of therapeutic endoscopy, will be held on 18–20 June 2001 in Brussels, Belgium. Further information: Mrs Nancy Beauprez, Gastroenterology Department, Erasme Hospital, Route de Lennik 808; B-1070 Brussels; Tel: +32 02 555 49 00; fax: +32 02 555 49 01; email: beauprez@ulb.ac.be

Falk Symposium

The symposium entitled Update in Inflammatory Bowel Diseases will be held in Liubljeva, Slovenia, on 5 May 2001. Further information: Prof Dr S Marković, University Medical Center Ljubljana, Division of Internal Medicine, Jafejeva 2, 1525 Liubljana, Slovenia; Tel: +386 (1) 231 6925; fax: +386 (1) 433 4190; email: sasa.markovic@kclj.si

Summer Abdominal Imaging Conference

A five day course designed for the practising radiologist with a primary interest in abdominal imaging, emphasising the most recent advances in helical CT, MRI, US, and gastrointestinal imaging. It will be held on 23–27 July 2001 in Banff Springs, Canadian Rockies. Twenty-five category 1 credit hours. Further information: Janice Ford Benner, University of Pennsylvania Medical Center (Radiology), 3400 Spruce Street, 1 Silverstein Building, Philadelphia, PA 19104, USA. Tel: +1 215 662 6904; fax: +1 215 349 5925.
Guidelines for the management of iron deficiency anaemia

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Updated information and services can be found at:
http://gut.bmj.com/content/48/2/283.3

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