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 respective postcholecystectomy diarrhoea 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.2 Therapy consisted of either conventional cholecystectomy or extracorporeal shock wave lithotripsy (ESWL), allocated at random. The second paper focused on the prevalence of postcholecystectomy diarrhoea before and after conventional and laparoscopic cholecystectomy.3

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.4

Although the study design of our two studies differed largely from that of Hearing’s, the results and conclusions are in agreement, that clinical diarrhoea seldom develops after cholecystectomy. O’Donnell is correct that objective assessment methods (such as measuring the upper gastrointestinal symptoms score) are needed to solve this problem.5

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MALT lymphomas and Helicobacter pylori

EDITOR,—Raderer and associates (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, those of the small intestine, salivary glands, nasal mucosa, and colon have recently been reported. Although Helicobacter pylori is generally implicated as the inducing agent, this does not always appear to be the case. Relapsing bacterium, H. heilmannii, has also been found in association with gastric MALT lymphomas, including H. pylori negative patients whose disease was still responsive to antibiotic treatment. Furthermore, other non-H pylori bacterial and protozoal flora have been observed in gastric lymphomas specific to infected regions. In the report by Raderer et al, and in several of the cases previously mentioned, 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, 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 point to consider when confronted with patients diagnosed with H. pylori negative MALT lymphomas.

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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 spite of the predictive power have their basis in the comparison between the receiver operating characteris- tic (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 MCV of <77 fl in predicting serum ferritin levels of <20 µg/l.2 All low MCV values had low MCH values but nine hypoferremic patients with low MCH had MCV within the normal range.

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Reply


In my study, comprising 201 subjects with iron deficiency (characterised by serum ferritin <18 µg/l), the MCH conferring 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 70%. By contrast, for MCV, optimum trade off between sensitivity (61.7%) and specificity (59.1%) was obtained with a cut off level of <77 fl, giving a positive predictive value of 65%. There were 31 patients with an MCH <26 pg in the presence of an MCV >80 fl compared with only four with an MCV <80 fl in the presence of an MCV >80 pg and, among these, four had an MCH <24 pg in the presence of an MCV >77 fl in contrast with only one with an MCV >77 fl in the presence of an MCH >24 pg. In my study, the most stringent cut off diagnostic level for iron deficiency was a serum ferritin level <10 µg/l found in a subgroup of 145 subjects. At this level, the MCH characterised by optimum trade off between sensitivity (65%) and specificity (66%) was <76 fl (identical with the cut off level in the guidelines), 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 sensitivities, specificities, and positive predictive values of 58%, 75%, and 62%, respectively.

Reply

EDITOR,—Suggesting both that transferrin saturation 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 sensitivity (61.7%) and specificity (59.1%) for iron deficiency anaemia in the elderly in Gut (that is, iron deficiency together with atrophic gastritis) may be associated with iron deficiency anaemia (IDA) is not contradictory. However, 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 volume (MCV) in diagnosing IDA interest- ing. 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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American College of Gastroenterology 2001 International GI Training Grants Programme

The ACG International GI Training (IGT) Grant Programme provides funding for clinical or research training in gastroenterology and hepatology so that an individual can acquire or develop new knowledge and/or a technical skill. This newly acquired knowledge and/or skill can be used to improve patient care in the applicant’s geographic area. Physicians outside of the United States and Canada are eligible to apply. At least one fellowship with a maximum of $10 000 per IGT fellowship will be awarded during 2001, for a training period of not less than six months. Awards will be made by a special committee of the ACG and will be based upon the applicant’s credentials, the merit of the proposed training, the potential for enhancing the field of gastroenterology in the applicant’s home country. Application forms can be obtained from the ACG administrative office: 4900B South 31st Street, Arlington, Virginia 22206-1656. Tel: +1 703 820 7400; fax: +1 703 931 4520; website: www.acg.org. Deadline for submission of application is 1 April 2001.

Cleveland Clinic Florida’s Gastroenterology Update 2001

Cleveland Clinic Florida will be sponsoring a postgraduate course entitled “Gastroenterology UPDATE 2001” to be held on 10–11 February 2001 in Fort Lauderdale, Florida, USA. Further information: Sally Jagelman, Manager of Continuing Medical Education, Cleveland Clinic Florida, 3000 West Cypress Creek Road, Fort Lauderdale, FL 33309, USA. Tel: +1 954 978 5539; fax: +1 954 978 5056; email: jagelmsn@ccf.org

GI malignancies can be prevented and treated: from the bench to the bedside

This international meeting will be held on 14–17 February 2001 in Jerusalem and the Dead Sea, Israel. Further information: Mari- lyn Katz, Secretariat, GI Malignancies, Tar- get Tours, PO Box 29041, Tel Aviv 61290, Israel. Tel: +972 3 5175150; fax: +972 3 5175155; email: gj@targetconf.com

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

Gut imaging. It will be held on 1–3 August 2001 in Bangkok, Thailand. Further information: Prof Dr Jolobe and the authors of A138 are L Sarli, R Costi, S Gobbi, D Usuco, D Sarli, and the authors of A138 are L Sarli, R Costi, S Gobbi, C Pavlidis, L Roncoroni, Ireland. Further information: Maddalena Massaro, Project Leader, AISC-AIM Group, Via A Ristori 38, 00187 Rome, Italy. Tel: +39 06 809681; fax: +39 06 80968229; email: gastro2001@aisc.it.

3rd European Federation of Autonomic Societies (EFAS)

The third European Federation of Autonomic Societies (EFAS) meeting in conjunction with the annual meeting of the sections “Auto- nomic nervous system” of the German Neuro- logical Society, “Diabetes and Nervous Sys- tem” of the German Neurological Society, and “Autonomic Nervous System” at the University of Erlangen-Nuremberg, Germany, will be held in Erlangen, Germany on 26–28 April 2001. Further information: Professor Dr M J Hila, Department of Neurology, University or Erlangen-Nuremberg, Schwabachanlage 6, D-91054 Erlangen, Germany. Tel: +49 0131 8534444; fax: +49 9131 8534528; website: www.neurologie.med.uni-erlangen.de/oeffentliche Veranstaltungen.htm

Falk Workshop

The workshop entitled Update in Inflammatory Bowel Diseases will be held in Ljubljana, Slovenia, on 5 May 2001. Further information: Prof Dr S Marković, University Medical Center Ljubljana, Division of Intern- Medine, Japheva 2, 1525 Ljubljana, Slovenia. Tel: +386 (1) 233 6925; fax: +386 (1) 433 4190; email: ssa.markovic@kclj.si

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Falk Symposium

The symposium Inflammatory Bowel Disease: A Clinical Case Approach to Pathophysiology, Diagnosis, and Treatment will be held in Bologna, Italy on 22–23 June 2001. Further information: Prof Dr M Campieri/ Dr P Gion- chetti, Policlinico S. Orsola - Malpighi, Dipar- timento di Medicina Interna e Gastroentero- logia, Via Massarenti 9, I-40138 Bologna, Italy. Tel: +39 (051) 6364 116 or 6364 122; fax: +39 (051) 302 92338; email: campieri@med.unibo.it or paolo@med.unibo.it

Summer Abdominal Imaging Conference

A five day course designed for the practising radiologist with a primary interest in abdomi- nal 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 Silver- strin Building, Philadelphia, PA 19104, USA. Tel: +1 215 662 6904; fax: +1 215 349 5925.
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