

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 prevalence of 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.^{1,2}

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.¹ 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 assessment rarely demonstrates 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.

P W PLAISIER

Department of General Surgery, Medisch Spectrum Twente,
PO Box 50 000,
NL-7500 KA Enschede, Netherlands
pplaisier@planet.nl

- 1 Plaisier PW, van der Hul RL, Nijs HGT, *et al*. The course of biliary and gastrointestinal symptoms after treatment of uncomplicated symptomatic gallstones: results of a randomized study comparing extracorporeal shock wave lithotripsy with conventional cholecystectomy. *Am J Gastroenterol* 1994;89:739-44.
- 2 Plaisier PW, van der Hul RL, Nijs HGT, *et al*. Quality of life and the course of biliary and gastrointestinal symptoms after laparoscopic and conventional cholecystectomy. *Dig Surg* 1995;12:87-91.
- 3 O'Donnell LJD. Post-cholecystectomy diarrhoea: a running commentary. *Gut* 1999;45:796-7.

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,¹ those of the small intestine,² salivary glands,^{3,4} nasal mucosa,⁵ and colon^{6,7} have recently been reported.

Although *Helicobacter pylori* is generally implicated as the inducing agent, this does not always appear to be the case. A related 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.⁸ Furthermore, other non-*H. pylori* bacterial⁹ and protozoal¹⁰ flora have been observed in gastric lymphomas specific to involved regions. In the report by Raderer *et al*, and in several of the others previously mentioned,³⁻⁷ *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,⁷ 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.

S A HOPTION CANN

J P VAN NETTEN

Special Development Laboratory,
Royal Jubilee Hospital,
Victoria, BC, Canada and
Department of Biology, University of Victoria,
Victoria, BC, Canada

C VAN NETTEN

Department of Health Care and Epidemiology,
University of British Columbia, Vancouver, BC,
Canada V6T 1Z3

Correspondence to: Dr J P van Netten, Special Development Laboratory, Royal Jubilee Hospital, 1900 Fort Street, Victoria, BC, Canada V8R 1J8. hvannetten@caphealth.org

- 1 Ng WW, Lam CP, Chau WK, *et al*. Regression of high-grade gastric mucosa-associated lymphoid tissue lymphoma with *Helicobacter pylori* after triple antibiotic therapy. *Gastrointest Endosc* 2000;51:93-6.
- 2 Fischbach W, Tacke W, Greiner A, *et al*. Regression of immunoproliferative small intestinal disease after eradication of *Helicobacter pylori*. *Lancet* 1997;349:31-2.
- 3 Alkan S, Karcher DS, Newman MA, *et al*. Regression of salivary gland MALT lymphoma after treatment for *Helicobacter pylori*. *Lancet* 1996;348:268-9.
- 4 Berrebi D, Lescœur B, Faye A, *et al*. MALT lymphoma of labial minor salivary gland in an immunocompetent child with a gastric *Helicobacter pylori* infection. *J Pediatr* 1998;133:290-2.
- 5 Gupte S, Nair R, Naresh KN, *et al*. MALT lymphoma of nasal mucosa treated with antibiotics. *Leuk Lymphoma* 1999;36:195-7.
- 6 Matsumoto T, Iida M, Shimizu M. Regression of mucosa-associated lymphoid-tissue lymphoma after eradication of *Helicobacter pylori*. *Lancet* 1997;350:115-16.
- 7 Inoue F, Chiba T. Regression of MALT lymphoma of the rectum after anti-*H. pylori* therapy in a patient negative for *H. pylori*. *Gastroenterology* 1999;117:514-15.
- 8 Morgner A, Lehn N, Andersen LP, *et al*. *Helicobacter heilmannii*-associated primary gastric low-grade MALT lymphoma: complete remission after curing the infection. *Gastroenterology* 2000;118:821-8.
- 9 Jonkers D, Gisbertz I, De Bruine A, *et al*. *Helicobacter pylori* and non-*Helicobacter pylori* bacterial flora in gastric mucosa and tumour specimens of patients with primary gastric lymphoma. *Eur J Clin Invest* 1997;27:885-92.
- 10 Otrajki CL, Albores-Saavedra J, Martinez AJ. Gastric malignant lymphoma with superimposed amebiasis. *Am J Gastroenterol* 1990;85:72-5.

Reply

EDITOR,—Hoption 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 scenario, 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 suggestive of an underlying infectious process but does not necessarily constitute proof of this assumption, as direct antiproliferative mechanisms of various antibiotics, including clarithromycin, have been reported in different settings. Further investigations are needed before definite recommendations for (as yet empirical) antibiotic therapy in patients with extragastric MALT lymphomas can be given.

M RADERER

Department of Internal Medicine I,
Division of Oncology, University of Vienna,
Währinger Gürtel 18-20,
A-1090 Vienna, Austria

ANDREAS CHOTT

Department of Clinical Pathology,
University of Vienna,
Währinger Gürtel 18-20,
A-1090 Vienna, Austria

Correspondence to: Professor M Raderer.
markus.raderer@akh-wien.ac.at

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):iv1-5). Statistical considerations which dictate that serum ferritin will always outrank transferrin saturation in predictive power have their basis in the comparison 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 that an MCH of <27 μg was superior to a mean corpuscular volume (MCV) of <77 fl in predicting serum ferritin levels of <20 $\mu\text{g/l}$.² All low MCV values had low MCH values but nine hypoferritinaemic patients with low MCH had MCV within the normal range.²

In my own 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%.³ 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 MCH >26 pg³ 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 MCV 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.

O JOLOBE

Department of Medicine for the Elderly,
Tameside General Hospital,
Fountain Street,
Ashton under Lyne OL6 9RW, UK
andrea.hirst@exchange.tgcp-tr.nwest.nhs.uk

- Guyatt GH, Patterson C, Ali M, et al. Diagnosis of iron-deficiency anaemia in the elderly. *Am J Med* 1990;88:205-9.
- O'Broin SD, Kelleher BP, McCann SR, et al. The value of erythrocyte indices as a screening procedure in predicting nutritional deficiencies. *Clin Lab Haematol* 1990;12:247-55.
- Jolobe OMP. Prevalence of hypochromia (without microcytosis) vs microcytosis (without hypochromia) in iron deficiency. *Clin Lab Haematol* 2000;22:79-80.

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 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.

B B SCOTT

Department of Gastroenterology,
Lincoln County Hospital,
Lincoln LN2 5QY, UK
drbbscott@aol.com

CORRECTION

Two abstracts in *Gut* 2000;47(suppl III) had incomplete author lists. The authors of A136 are L Sarli, R Costi, S Gobbi, D Iusco, D Sarli, and the authors of A138 are L Sarli, R Costi, S Gobbi, C Pavlidis, L Roncoroni.

NOTES

American College of Gastroenterology 2001 International GI Training Grants Programme

The ACG International GI Training (IGT) Grant Programme provides funding for clinical or clinical research training in gastroenterology and hepatology so that an individual can acquire or develop new cognitive knowledge or a technical skill. This newly acquired knowledge or skill would then 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 by the selected host training centre and 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.gi.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: jagelms@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: Marilyn Katz, Secretariat, GI Malignancies, Target Tours, PO Box 29041, Tel Aviv 61290, Israel. Tel: +972 3 5175150; fax: +972 3 5175155; email: gi@targetconf.com

Redefining Priorities in Gastroenterology

This congress will be held on 11-14 April 2001 in Monte Carlo, Italy. It will be chaired by Professor Massimo Crespi (Rome, Italy) and Professor Eammon Quigley (Cork,

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 "Autonomic nervous system" of the German Neurological Society, "Diabetes and Nervous System" 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 Hilz, Department of Neurology, University of Erlangen-Nuremberg, Schwabachanlage 6, D-91054 Erlangen, Germany. Tel: +49 0131 8534444; fax: +49 9131 8534328; 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 Markovič, University Medical Center Ljubljana, Division of Internal Medicine, Japljeva 2, 1525 Ljubljana, Slovenia. Tel: +386 (1) 231 6925; fax: +386 (1) 433 4190; email: sasa.markovic@kclj.si

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 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 Gionchetti, Policlinico S. Orsola - Malpighi, Dipartimento di Medicina Interna e Gastroenterologia, Via Massarenti 9, I-40138 Bologna, Italy. Tel: +39 (051) 6364 116 or 6364 122; fax: +39 (051) 392538; 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 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.