LETTERS TO THE EDITOR

Endoscopic findings and clinical patterns are not useful for distinguishing low from high grade gastric MALT lymphoma

EDITOR,—Taal *et al* showed recently (*Gut* 1996; 39: 556-61) that both clinical parameters and endoscopic findings differ significantly between low and high grade gastric mucosa associated lymphoid tissue (MALT) lymphoma. In fact, low grade MALT lymphoma has often been interpreted endoscopically as a benign condition, whereas high grade MALT lymphoma often has neoplastic-like features. Weight loss and older age are more frequent in high grade MALT lymphoma.

In our experience the endoscopic findings and clinical parameters differ from the data reported by Taal *et al*.

Between 1993 and 1996 we diagnosed one high grade and three low grade MALT lymphomas. In the low grade MALT lymphomas (one 70 year old man and two women, one 65 and the other 56 years old), the endoscopic findings were a gastric ulcer in the male patient, and a large antral mass with multiple ulcerations in the first and a large polypoid mass with pyloric obstruction in the second female patient. All of them had lost weight (6-10 kg). The male patient and the female patient with multiple antral ulcerations were *Helicobacter pylori* positive. These patients were treated with an *H. pylori* eradication regimen, and the *H. pylori* negative patient was treated with polichemotherapy. In all cases the tumour regressed and the endoscopic pattern resolved. In the 67 year old woman with high grade MALT lymphoma the endoscopy showed a pin-point stenosis of the pilorus, with a large mass ulcerated at the angulus and weight loss (6 kg in two months). Although *H. pylori* was present, the patient was treated successfully with surgery (gastrectomy), followed by polichemotherapy. These data show that the clinical and endoscopic patterns are very similar in low and high grade MALT lymphoma, and that is very difficult to determine the grade of the MALT lymphoma on the basis of the endoscopic or clinical, or both, patterns.

At the initial gastroscopy the male patient with the low grade MALT lymphoma had two adjacent ulcers on the posterior wall of the gastric corpus, was *H. pylori* positive, and had histological and immunohistochemical evidence of low grade MALT lymphoma only in gastric mucosal specimens obtained from the ulcer margins. Anti-*H. pylori* treatment resulted both in the eradication of the bacterium and regression of the neoplasia, with resolution of the ulcers.¹ After 18 months, the patient was reinfected by *H. pylori* and endoscopy showed a diffuse thickening of gastric mucosal folds in all gastric sites, mimicking linitis plastica. Histological and immunohistochemical analyses showed a low grade MALT lymphoma.¹ After the failure of two consecutive courses of anti-*H. pylori* treatment, the patient was treated surgically due

to progressive weight loss. Histological examination of the surgical specimens confirmed the diagnosis of low grade MALT lymphoma, stage E11.²

This case shows clearly that the endoscopic pattern of low grade MALT lymphoma can change greatly even in a single patient, making it impossible to use the endoscopic or clinical pattern to distinguish between low and high grade MALT lymphoma.

ANTONIO TURSI
GIOVANNI CAMMAROTA
ALFREDO PAPA
GIUSEPPE FEDELI
GIOVANNI GASBARRINI
*Istituto di Medicina Interna e Geriatria-Divisione di Gastroenterologia,
Policlinico A. Gemelli,
Università Cattolica del Sacro Cuore,
Largo A. Gemelli,
8 00168-Roma,
Italy*

Correspondence to: Dr Antonio Tursi.

- 1 Cammarota G, Tursi A, Montalto M, Papa A, Branca G, Vecchio FM, *et al*. Prevention and treatment of low-grade B-cell primary gastric lymphoma by anti-*H. pylori* therapy. *J Clin Gastroenterol* 1995; 21: 118-22.
- 2 Cammarota G, Tursi A, Papa A, Voso MT, Serra F, Chiusolo P, *et al*. The growth of primary low-grade B-cell gastric lymphoma is sustained by *Helicobacter pylori*. *Scand J Gastroenterol* 1997; 32: 285-7.

Reply

EDITOR,—The endoscopic pattern of gastric non-Hodgkin's lymphoma (NHL) has been described by several authors.^{1,2} There are three main patterns: a tumour-like appearance with a polypoid mass (exophytic type); ulceration or multiple small erosions (infiltrative type); or thickened, giant folds such as those seen in linitis plastica. Unfortunately, these descriptions are not specific for gastric NHL. As gastric lymphoma is a rather uncommon disease, one might imagine that the endoscopist is not always aware of the possibility of a lymphoma rather than a carcinoma or even the more frequently present gastritis.

In our series of 114 patients with primary gastric lymphoma we evaluated the initial endoscopic diagnosis in relation to clinical findings and grade of malignancy: the endoscopic appearance was interpreted as benign disease in 52% of 51 patients with low grade MALT lymphoma. In most patients (71% of 63) the high grade lymphomas were classified as malignant and in only 13% a correct diagnosis of NHL was made by the endoscopist. The pathologist, however, was able to give an accurate diagnosis at initial biopsy in 77% and in 91% after repeat endoscopy with multiple biopsies, when the diagnosis was not conclusive.³

So, we are not at all surprised by the findings reported by Dr Tursi and colleagues. They describe four cases: three of them with a low grade gastric NHL associated with a polypoid tumour mass in two (compared with 13 of 51 patients with low grade lymphoma in our series) and the third with an ulcer. The fourth case with high grade lymphoma suffered from significant weight loss and a tumour mass as is often found in this group. Thus, in summary the findings were consistent with the general pattern in two of the four cases. This supports our conclusion that the endoscopic recognition of gastric NHL is difficult;

a high index of suspicion may help, but we feel that it is of utmost importance to take routine biopsy specimens in as many patients as possible, preferably all patients seen in the GI endoscopy unit. So we do not agree with Tursi *et al* who reject our conclusions (based on the experience in 114 patients) with their report of four cases only, which cannot be considered as an impressive number of cases or even a series of patients.

The issue of treatment of gastric NHL by eradication of *H. pylori* was not the subject of our study. Information on this topic is increasing and eradication treatment may induce complete regression in non-bulky, low grade MALT lymphoma stage I in approximately 70% of cases.^{2,4} The value of *H. pylori* eradication in bulky disease as mentioned by Tursi *et al* is still a matter of much debate.

In general, it is important to detect gastric lymphoma at an early stage when treatment might be less toxic and most effective. Therefore, we would like to stress that a diagnosis of low grade gastric NHL should be kept in mind when encountering benign gastritis, and that high grade NHL expresses more carcinoma-like features. Multiple biopsy specimens are an absolute requirement for an optimal diagnosis and treatment choice.

B G TAAL
H BOOT
D DE JONG
*Netherlands Cancer Institute/Antoni van Leeuwenhoekhuis,
Plesmanlaan 121,
1066 CX Amsterdam,
The Netherlands*

- 1 Seifert E, Schulte F, Weismuller J, de Mas CR, Stolte M. Endoscopic and biopsic diagnosis of malignant non-Hodgkin's lymphoma of the stomach. *Endoscopy* 1993; 25: 497-501.
- 2 Bayerdörfer E, Neubauer A, Rudolph B, Thiede C, Lehn N, Eidt S, *et al*. Regression of primary gastric lymphoma of mucosa associated lymphoid tissue type after cure of *Helicobacter pylori* infection. *Lancet* 1995; 345: 1591-4.
- 3 Taal BG, den Hartog Jager FCA, Burgers JMV, van Heerde P, Tio L. Primary non-Hodgkin's lymphoma of the stomach: changing aspects and therapeutic choices. *Eur J Cancer Clin Oncol* 1989; 25: 439-50.
- 4 Boot H, de Jong D, van Heerde P, Taal BG. Role of *Helicobacter pylori* eradication in high grade MALT lymphoma. *Lancet* 1995; 346: 448-9.

Natural history of polypoid lesions of the gall bladder

EDITOR,—Moriguchi *et al* (*Gut* 1996; 39: 860-2) report that of 4343 patients, 109 had gall bladder polyps detected on ultrasound, most of which were benign. They follow this with an interesting brief review of the literature. From their comments and my own reading of the subject, the overwhelming majority of gall bladder polyps are benign, although one of the 109 patients was found to have gall bladder cancer histologically, and this ties in with previous reports.¹ Koga *et al*¹ found carcinoma of the gall bladder presenting as polypoid lesions in three of four cases on ultrasound in their study and, surprisingly, by gall bladder wall thickening in only one of the four cases. Although the numbers are small and gall bladder cancer is relatively rare, it still comprises 3% of all gastrointestinal malignancies and causes 6500 deaths annually in the USA.² In addition, the spread and size of tumour are correlated.^{3,4} The patients who survive more than five years are those in whom the cancer is diagnosed histo-

logically at cholecystectomy for presumed benign disease—that is early, with disease confined to the mucosa or submucosa. Disease beyond these limits is accompanied by a drastic drop in survival rates at five years from 64 to 44% and the overall five year prognosis is quoted as less than 5%.⁴ Gallstones have also been found commonly in association with both gall bladder polyps and gall bladder malignancy.^{2-4,6} In the light of these findings, early cholecystectomy in selected patients could reduce long term mortality. Preliminary histology at surgery could permit dissection of portal hepatic nodes, excision of the gall bladder bed, and right hepatic lobectomy at initial cholecystectomy, thus improving survival² and reducing the costs of repeat operation.

What remains controversial and undecided, however, is how to select those patients who should have early cholecystectomy from those who should have six monthly follow up ultrasound. Certainly, rapidly enlarging lesions or those with areas of gall bladder wall thickening should be removed, including benign lesions in women, especially those over 60,³ and in patients with gallstones, even asymptomatic ones.⁷ In the study by Moriguchi *et al* the size of the polyps was not related to whether malignancy developed, extrapolating from the adenoma–carcinoma sequence in which gall bladder adenomas are premalignant lesions.⁶ Indeed in Moriguchi *et al*'s study it is interesting that the larger polyps were found in the younger age group.

Clearly, logistic reasons dictate that not every patient with polyps should have an instant cholecystectomy if asymptomatic, and there does have to be a process of sifting and selection, but, in my view, this should err towards the side of more frequent surgery in the fit patient for the reasons discussed above.

E KYRIACOU
London

- 1 Koga A, Yamauchi S, Izumi Y, Hamanaka N. Ultrasonographic detection of early and curable carcinoma of the gallbladder. *Br J Surg* 1985; 72: 728–30.
- 2 Bivins BA, Meeker WR, Weiss DL, Griffen WO. Carcinoma in situ of the gallbladder: a dilemma. *South Med J* 1975; 68: 297–300.
- 3 Koga A, Watanabe K, Fukuyama T, Takiguchi S, Nakayama F. Diagnosis and operative indications for polypoid lesions in the gallbladder. *Arch Surg* 1988; 123: 26–9.
- 4 Bergdahl L. Gallbladder carcinoma first diagnosed at microscopic examination of gallbladders removed for presumed benign disease. *Ann Surg* 1980; 191: 20–2.
- 5 Sawyer KC. The unrecognized significance of papillomas, polyps, and adenomas of the gallbladder. *Am J Surg* 1970; 120: 570–8.
- 6 Aldridge MC, Bismuth H. Gallbladder cancer: the polyp–cancer sequence. *Br J Surg* 1990; 77: 363–4.
- 7 Jensen KH, Jorgensen T. Incidence of gallstones in a Danish population. *Gastroenterology* 1991; 100: 790–4.

Reply

EDITOR,—We are grateful to Dr Kyriacou for her interest in our recent study. We agree with him that there is a small number of patients who are diagnosed with gall bladder carcinoma and that early detection of carcinoma would improve their prognosis. The study by Koga *et al* cited by Dr Kyriacou, however, describes the prevalence of gall bladder carcinoma in patients who had undergone cholecystectomy. Therefore, it is difficult to draw

any conclusion from this study in terms of the prognosis of polypoid lesions of the gall bladder.

Prospective studies are needed to determine whether polypoid lesions develop into gall bladder carcinoma. Progression from adenoma to carcinoma takes time, and therefore these patients should be followed long term.

CHIFUMI SATO
Division of Health Science,
Faculty of Medicine,
Tokyo Medical and Dental University,
1-5-45 Yushima, Bunkyo-ku,
Tokyo 113, Japan

- 1 Koga A, Watanabe K, Fukuyama T, Takiguchi S, Nakayama F. Diagnosis and operative indications for polypoid lesions of the gallbladder. *Arch Surg* 1988; 123: 26–9.

Polypoid lesions of the gall bladder

EDITOR,—The paper by Moriguchi *et al* (*Gut* 1996; 39: 860–2) raises a number of questions. The authors state that this is the first study to describe the natural history of these lesions, but their solitary conclusion, that most polypoid lesions of the gall bladder detected by ultrasonography are benign, is not new. Indeed, they seem to underestimate the risk of malignancy compared with other larger series.

Yang *et al*¹ studied 182 patients who underwent cholecystectomy and who had an ultrasonographic or pathological diagnosis of polypoid lesions of the gall bladder. There were 10 false positive ultrasound diagnoses. All the lesions less than 1 cm in diameter were benign. There were 13 malignant lesions, 11 of which were greater than 1.5 cm in diameter. All the malignant lesions were solitary. All but two of the 182 patients underwent cholecystectomy and the calculated sensitivity of ultrasonography was 90.1% and specificity was 93.9%.

These findings were in patients with right upper quadrant symptoms. Moriguchi *et al* do not indicate how their patients were selected, nor whether they were symptomatic. However, it seems likely that most were asymptomatic as only four patients underwent cholecystectomy during a five year follow up period. It may be that differences in selection explain their much lower incidence of polyps greater than 1 cm (6.4% *v* 19.8%) and of malignant tumours (1.8% *v* 7.6%).

While most gall bladder polypoid lesions may indeed be benign, those which exceed 1 cm in diameter and are single have a high risk of malignancy and should be removed surgically, especially if the patient is over 50 years old.¹ Unfortunately, Moriguchi *et al*'s study does not give us any information about the relationship between symptoms and gall bladder polypoid lesions. It seems reasonable to recommend, however, that cholecystectomy be performed in symptomatic patients, particularly if gallstones are also present.

C D JOHNSON
University Surgical Unit,
Southampton General Hospital,
Southampton, UK

- 1 Yang HL, Sun YG, and Wang Z. Polypoid lesions of the gallbladder: diagnosis and indications for surgery. *Br J Surg* 1992; 79: 227–9.

Reply

EDITOR,—We are grateful to Dr Johnson for his interest in our recent paper. As we described in the title, we studied the natural history of polypoid lesions in the gall bladder. The study by Yang *et al* cited by Dr Johnson, however, showed the prevalence of gall bladder carcinoma in patients who had undergone cholecystectomy. In their paper, the prognosis of polypoid lesions of the gall bladder is not described. To our knowledge, there are no papers that describe the natural history of polypoid lesions in the gall bladder in the literature. Therefore, it is difficult to say whether our study underestimates the risk of malignancy. A large study does not necessarily result in the correct conclusion, but we do feel that our study would have benefitted from a larger series of patients.

All of the patients presented to the outpatient with heterogeneous abdominal complaints, but were asymptomatic during follow up, differing in this regard from the patients studied by Yang *et al*. In our opinion patients with right upper quadrant symptoms should not be followed for five years without treatment.

Finally, we agree with Dr Johnson that patients with gallstones should be monitored carefully. However, none of our patients had gallstones despite the close association between gall bladder carcinoma and gallstones.

CHIFUMI SATO
Division of Health Science,
Faculty of Medicine,
Tokyo Medical and Dental University,
1-5-45 Yushima, Bunkyo-ku,
Tokyo 113, Japan

Bone mineral density in Crohn's disease

EDITOR,—We were interested to read the recent article by Jahnsen *et al* (*Gut* 1997; 40: 313–9). We have just published a similar study investigating the bone mineral content (BMC) of children with inflammatory bowel disease (IBD),¹ and have also found that BMC was reduced in those with Crohn's disease but not in those with ulcerative colitis. No relation could be found between BMC and disease duration, disease activity, or biochemical markers of bone metabolism. There was, however, a significant relation between reduction in bone mineralisation and steroid usage, as also noted by Jahnsen *et al*. Although BMC was significantly lower in the children treated with steroids, there was no association with magnitude of steroid use in either dosage or duration. We postulated that the steroid effect was an all or nothing effect, or that the use of steroids acts as a marker for other variables, such as worsening disease activity. We agree that screening of bone mineral status in an important aspect of continuing care in patients with IBD and would like to emphasise that children are not immune to the osteopenic side effects of IBD.

F J COWAN
J W GREGORY
H R JENKINS
University Hospital of Wales,
Heath Park,
Cardiff CF4 4XW, UK

Correspondence to: Dr Cowan.

- 1 Cowan FJ, Warner JT, Dunstan FDJ, Evans WD, Gregory JW, Jenkins HR. Inflammatory bowel disease and predisposition to osteopenia. *Arch Dis Child* 1997; **76**: 325-9.

Reply

EDITOR,—We are grateful to Drs Cowan, Gregory and Jenkins for their interest and comments on our study. Crohn's disease is obviously a more severe systemic disease than ulcerative colitis and surprisingly most studies have failed to demonstrate a difference in bone mineral density between the two patient groups.

The adverse effects of corticosteroids on bone density are well recognised. However, published studies provide conflicting results on the association between steroid treatment and bone mineral density in patients with IBD.^{1,2} The cumulative steroid dose is usually calculated retrospectively from medical records and indeed this method is uncertain as treatment is intermittent and of varying length and dose. Inaccurate estimation of the total steroid dose may have contributed to the conflicting results reported in the literature on this issue.

We fully agree that steroid use (but also total lifetime dose) might reflect other variables connected to the intestinal illness which impact on bone mineralisation, such as disease activity.

J JAHNSEN
J A FALCH
E AADLAND
Medical Department,
Aker University Hospital,
Oslo, Norway

- 1 Compston JE, Judd D, Crawley EO, Evans WD, Evans C, Church HA, *et al*. Osteoporosis in patients with inflammatory bowel disease. *Gut* 1987; **28**: 410-5.
2 Bjarnason I, Macpherson A, Macintosh C, Buxton-Thomas M, Forgacs I, Moniz C. Reduced bone density in patients with inflammatory bowel disease. *Gut* 1997; **40**: 228-33.

Incidence of persistent symptoms after laparoscopic cholecystectomy

EDITOR,—I read with great interest the article by Luman *et al* (*Gut* 1996; **39**: 863-6) on the incidence of persistent symptoms after laparoscopic cholecystectomy.

I would like to point out that their statement that no study has analysed prospectively symptoms before and after laparoscopic cholecystectomy is not quite true. Although in a much smaller patient population, we published a paper on the same subject in 1995.¹ In our study, we found that cholecystectomy significantly improved quality of life, and cured nausea, fatty food upset, abdominal distension, and biliary pain. We also found that laparoscopic cholecystectomy improved quality of life and symptomatology at an earlier stage than conventional cholecystectomy.

Furthermore, I would like to stress that, although Luman *et al*'s study provides us with lots of interesting data and recommendations, it also leaves us with many unanswered questions. How many patients were excluded from the study because of planned open cholecystectomy and inability to answer the questionnaire? What were the reasons for

treating patients by open cholecystectomy? How many patients underwent preoperative endoscopic retrograde cholangiopancreatography (ERCP) and did all of them undergo sphincterotomy? Were there any differences in symptoms between patients with or without sphincterotomy? Why is ERCP not mentioned in the analysis of preoperative investigations (Table IV)? Was there symptomatic relief of heartburn in patients from the uncomplicated group? I would be grateful if the authors would answer some of these questions.

P W PLAISIER
Department of General Surgery,
St Clara Hospital,
Olympiaweg 350,
NL-3078 HT Rotterdam,
The Netherlands

- 1 Plaisier PW, van der Hul RL, Nijs HGT, den Toom R, Terpstra OT, Bruining HA. Quality of life and the course of biliary and gastrointestinal symptoms after laparoscopic and conventional cholecystectomy. *Dig Surg* 1995; **12**: 87-91.

Reply

EDITOR,—We thank Dr Plaisier for the interest he has expressed in our paper and for his thoughtful comments. We are grateful to him for pointing out his group's published work. We agree with Dr Plaisier that laparoscopic cholecystectomy carries significantly less morbidity than open surgery.¹⁻³

Our paper assessed symptoms after laparoscopic cholecystectomy and patients undergoing open surgery were not considered. Five per cent of patients undergo open cholecystectomy in our institution each year. No patients were excluded from the study because they were unable to answer the questionnaire.

As mentioned in our paper, patients with a history of jaundice, abnormal liver function tests, dilated common bile duct, or pancreatitis underwent preoperative ERCP. Ten patients had preoperative ERCP, five of whom required sphincterotomy for removal of common bile duct stones. All of them had successful symptomatic outcome from laparoscopic cholecystectomy. ERCP was not mentioned in the analysis of preoperative investigation as it was carried out for complications of choledocholithiasis—that is, pancreatitis or obstructive jaundice, and not for elucidation of causes of abdominal pain. There were preoperative differences in symptoms in patients who had choledocholithiasis as indicated in the first sentence of this paragraph.

In Table V, we showed that heartburn was relieved by laparoscopic cholecystectomy in only 3% of patients. None of the patients developed heartburn after surgery.

W LUMAN
K R PALMER
Gastrointestinal Unit,
Western General Hospital,
Crewe Road,
Edinburgh EH4 2XU, UK

- 1 Grace PA, Qureshi A, Coleman J. Reduced postoperative hospitalization after laparoscopic cholecystectomy. *Br J Surg* 1991; **78**: 160-2.
2 Nathanson LK, Shimi S, Cushieri A. Laparoscopic cholecystectomy: the Dundee technique. *Br J Surg* 1991; **78**: 155-9.
3 Macintyre IMC, Wilson RG. Laparoscopic cholecystectomy. *Br J Surg* 1993; **80**: 552-9.

An exceptional high concentration of serum CA 19.9 in a patient with alcoholic liver disease

EDITOR,—Since the discovery by Koprowski and co-workers in 1979, raised serum concentrations of carbohydrate antigenic determinant, CA 19.9, has been used as an additional diagnostic test for adenocarcinoma of the upper gastrointestinal tract.¹⁻³ According to Benamouzig *et al*,³ at the usual 37 U/ml cut off value, CA 19.9 had a sensitivity of 90% and a specificity of 45% for malignant biliary obstruction due to pancreatic or biliary neoplasm. For a more increased cut off value of 200 U/ml, its sensitivity was 65% and specificity was 91%. If higher cut off values are used, specificity rises so that at levels greater than 1000 U/ml, it approaches 100%.⁴ A very high concentration of serum CA 19.9 is therefore currently the "gold standard" marker for malignant biliary obstruction resulting from pancreatic cancer.⁴

Here, we report an exceptional high concentration of serum CA 19.9 in a patient with alcoholic liver disease. When the patient ceased drinking, the CA 19.9 serum concentration returned to normal within few months.

CASE REPORT

A 58 year man was admitted to our hospital because of jaundice and weight loss. He also complained of fatigue and pruritus of several weeks duration. His medical history was, except for psoriasis, unremarkable. However, he took acetylcysteine, eprazinohydrochloride, and prior to admission clarithromycin for a respiratory infection and obstructive lung disease. The patient smoked 15 cigarettes a day. For his ischiatic pain, he also took naloxonhydrochloride and tramadolhydrochloride. In addition he consumed approximately 10 pints of beer a day for many years and for the past two years also a quarter bottle of gin a day.

Physical examination showed jaundice, firm hepatomegaly and psoriatic skin lesions.

Laboratory investigations revealed increased concentrations of plasma bilirubin (total 6.26 mg/dl, conjugated 3.92 mg/dl, normal <1 mg/dl), alkaline phosphatase (1071 U/l, normal <240 U/l), aspartate aminotransferase (197 U/l, normal <20 U/l), alanine aminotransferase (101 U/l, normal <20 U/l), and γ -glutamyltranspeptidase (1700 U/l, normal <60 U/l). The patient also had raised concentrations of triglycerides (1398 mg/dl, normal <200 mg/dl) and cholesterol (667 mg/dl, normal <240 mg/dl). Serological markers for hepatitis A, B and C did not indicate a recent infection or active viral replication. Autoantibodies were negative. Serum CA 19.9 was extremely high at 10.981 U/ml (confirmed in a retrospective examination), while carcinoembryonic antigen (CEA; normal <5 μ g/l) was 14.4 μ g/l.

An ultrasonography and a computed tomography scan indicated hepatomegaly and fatty change without focal lesions or dilated biliary ducts. Endoscopic retrograde cholangiopancreatography showed no abnormalities, in particular no signs of primary sclerosing cholangitis or other biliary and pancreatic duct abnormalities.

A liver biopsy revealed micro- and macrovesicular steatosis, periportal fibrosis, Mallory's bodies, in addition to the presence of bilirubinostasis. Liver histology was very suggestive of alcoholic liver changes.

Although the use of medical drugs, especially clarithromycin, may contribute to this clinical syndrome, we believe that alcohol misuse was the most attributable factor. We therefore advised the patient to stop drinking. Once alcohol consumption ceased, there was a dramatic improvement in clinical and biochemical abnormalities. During a follow up visit three months after presentation, biochemistry values had almost returned to normal. The serum concentration of CA 19.9 had decreased to 96 U/ml.

We conclude, therefore, that an exceptionally high concentration of serum CA 19.9 may be found in patients with alcoholic liver disease.

E DE GOEDE
S H YAP

Department of Hepatology,
University Hospital Leuven,
Herestraat 49,
3000 Leuven, Belgium

- 1 Koprowski H, Stepkowski Z, Mitchell K, Herlyn M, Herlyn D, Fulner P. Colorectal carcinoma antigens detected by hybridoma antibodies. *Somat Cell Genet* 1979; 5: 957-72.
- 2 Ritts RE, Del Villano BC, Go VLW, Herberman RB, Klug TL, Zurawski VR. Initial clinical evaluation of an immunoradiometric assay for CA 19.9 using the NCI serum bank. *Int J Cancer* 1984; 33: 339-45.
- 3 Benamouzig R, Buffet C, Fournier C, Ink O, Moati F, Etienne JP. Serum levels of carbohydrate antigenic determinant (CA 19.9) in obstructive jaundice. *Dig Dis Sci* 1989; 34: 1640-1.
- 4 Steinberg W. The clinical utility of the CA 19.9 tumor-associated antigen. *Am J Gastroenterol* 1990; 85: 350-5.

BOOK REVIEW

Inflammatory Bowel Diseases. Third edition. Editors RN Allen, JM Rhodes, SB Hanauer, MRB Keighley, J Alexander-Williams, VW Fazio. (Pp 989; illustrated; £125.00.) Edinburgh: Churchill Livingstone, 1996. ISBN 0-443-05067-8.

The time will come, one supposes, when all the brain's mysteries will be disclosed. Then, the combined forces of information technology and neurosciences will come up with methods to introduce and store knowledge—conveniently when we are asleep—so that existing methods of learning will be so inefficient that the lecture, seminar and written word will become redundant.

What will then become a blockbuster textbook such as the present offering? Is it to be replaced by a chip downloaded directly into our brains? Maybe so, but until that time we shall just have to make do with the good, old-fashioned read—and try and retain what we can.

If we are to continue with books for the time being, then please let them be as agree-

able as the latest edition of *Inflammatory Bowel Diseases*. The greatest care has gone into the choice of chapters (112) and authors (155) to produce a definitive text for our time.

This third edition, appearing some seven years after its predecessor, reflects the steady progress that has been made in our knowledge of this most enigmatic of areas within clinical gastroenterology. The addition of three new editors has led to the production of a volume that is, in many ways, very much more than just an update of the previous version. There are nearly 200 pages on pathogenesis which at least enable the clinician to feel a little more confident about answering patients' almost invariable question, at some time or other, as to the cause of their condition. Knowledge may provide comfort even if we all know the best short answer to that highly pertinent query! The clinical section offers comprehensive coverage of diagnosis (incorporating new imaging modalities), the treatment section has been updated and, in the case of surgery, the text has been more than minimally invaded by completely new material.

This book is 1000 pages long, and one perhaps should be a little wary of highlighting omissions. The field is fairly fast-moving, but this reviewer would like to have seen a little more about host-bacterial interaction in the gut. Presumably, the deadline was a little too late to cover all of the important recent progress in genetics. Similarly, targeted therapy has not received the attention that it might do in, for example, an annual update.

It is somewhat of an old canard to say that textbooks are out-of-date as soon as they hit the bookshelves. I certainly heard that said when I was a medical student some 25 years ago, but surely it must be ever-truer today and in the future as online journals become the norm.

I think this book could have done with a little excision. There is relatively little repetition, but I really wondered whether we needed to have 100 pages on inflammation in the gut which is not inflammatory bowel disease. I was unconvinced that we needed a chapter on '*Incisions and surgical approaches in surgery for inflammatory bowel disease*'.

Any reader mulling over the contents list will have no doubt that he is in for a pretty authoritative and substantial work when he finds out that the titles of chapters 4, 5 and 6 respectively are: *Epidemiological overview of inflammatory bowel disease*, *Epidemiology of inflammatory bowel disease*, and *Epidemiology of smoking and oral contraception*. There is patchy imbalance elsewhere. The histopathology of ulcerative colitis gets 20 pages whereas that of Crohn's disease gets just 6.

Overall though this is a very considerable volume indeed. Most of the chapters on therapy just exude with the authority of their authors. The fact that one of the authors of both the medical and surgical sections comes from the USA may even mean that this book will sell a few copies in the New World. It certainly deserves to.

ROSE MECHAN

NOTES

Sir Francis Avery Jones BSG Research Award 1998

Applications are invited by the Education Committee of the British Society of Gastroenterology who will recommend to Council the recipient of the 1998 Award. Applications (TWENTY COPIES) should include:

- A manuscript (2 A4 pages ONLY) describing the work conducted
- A bibliography of relevant personal publications
- An outline of the proposed content of the lecture, including title
- A written statement confirming that all or a substantial part of the work has been personally conducted in the UK or Eire.

Entrants must be 40 years or less on 31 December 1997 but need not be a member of the BSG. The recipient will be required to deliver a 40 minute lecture at the Annual Meeting of the Society in March 1998. Applications (TWENTY COPIES) should be made to the Honorary Secretary, BSG, 3 St Andrews Place, London NW1 4LB, by 1 December 1997.

Course in Postgraduate Gastroenterology

A Course in Postgraduate Gastroenterology will be held in Oxford, UK, on 4-7 January 1998. This course has been designed for consultants and registrars, including those who do not specialise in gastroenterology. Topics will include:

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Course fee £200 (\$330). Board and accommodation are available at Wadham College at extra cost. Six bursaries will be available for applicants training in gastroenterology or in research posts at British hospitals. Further information from: Dr DP Jewell, Gastroenterology Unit, Radcliffe Infirmary, Woodstock Road, Oxford OX2 6HE.

9th International Symposium on Cells of the Hepatic Sinusoid

The 9th International Symposium on Cells of the Hepatic Sinusoid will be held in Christchurch, New Zealand, from 27 September to 1 October 1998. Further information from: Professor Robin Fraser, I.S.C.H.S., Christchurch School of Medicine, PO Box 4345, Christchurch 8001, New Zealand. Tel: +64 3 3640 587; Fax: +64 3 3640 593; email: grogers@chmcs.ac.nz.