Paris staging system for primary gastrointestinal lymphomas

Currently, much effort is put into the development of specific therapeutic approaches that are tailored to specific lymphoma entities. As a consequence, more specific information needs to be collected to support the choice of therapy. In gastric marginal zone B cell lymphoma of MALT-type, the need for specialised documentation of tumour extent in the gastrointestinal tract. Several modifications for staging of local extent of the disease. The ‘T’ part of this system pertains to the anatomical structure of the organs and sufficiently fulfills the requirements for staging of local extent of the disease.

The European Gastro-Intestinal Lymphoma Study Group (EGILS) is a multidisciplinary group of investigators, including clinical investigators such as gastroenterologists, medical oncologists, radiotherapists, and pathologists, as well as basic researchers such as cellular and molecular biologists. Several groups from the UK, France, Germany, the Netherlands, Spain, and Austria now take part and have come together regularly since 1999 to discuss and study subjects in epidemiology and molecular and cell biology of gastrointestinal lymphoma. Clinical protocols and trials have been developed and performed as a collaborative effort. As a result of discussions on staging protocols and reporting systems over the past years, we would like to propose a modified TNM staging system, named after the first venue of the group in Paris. The staging system adequately records: (1) depth of tumour infiltration; (2) extent of nodal involvement; as well as (3) specific lymphoma spreading (table 1). It is adjusted to the gastrointestinal origin of the lymphoma, considering histopathological characteristics of extranodal B and T cell lymphomas. The use of this system in future studies will permit accurate comparison of the reported cohorts and should allow rapid accumulation of good data for proper stratification of patients for risk assessment and treatment options.

Table 1 Paris staging system for primary gastrointestinal lymphomas*

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>T0</td>
<td>No evidence of lymphoma</td>
</tr>
<tr>
<td>T1</td>
<td>Lymphoma confined to the mucosa/submucosa</td>
</tr>
<tr>
<td>T1m</td>
<td>Lymphoma confined to mucosa</td>
</tr>
<tr>
<td>T1sm</td>
<td>Lymphoma confined to submucosa</td>
</tr>
<tr>
<td>T2</td>
<td>Lymphoma infiltrates muscularis propria or subserosa</td>
</tr>
<tr>
<td>T3</td>
<td>Lymphoma penetrates serosa (visceral peritoneum) without invasion of adjacent structures</td>
</tr>
<tr>
<td>T4</td>
<td>Lymphoma invades adjacent structures or organs</td>
</tr>
<tr>
<td>NX</td>
<td>Involvement of lymph nodes not assessed</td>
</tr>
<tr>
<td>NO</td>
<td>No evidence of lymph node involvement</td>
</tr>
<tr>
<td>N1</td>
<td>Involvement of regional lymph nodes</td>
</tr>
<tr>
<td>N2</td>
<td>Involvement of intra-abdominal lymph nodes beyond the regional area</td>
</tr>
<tr>
<td>N3</td>
<td>Spread to extra-abdominal lymph nodes</td>
</tr>
<tr>
<td>MX</td>
<td>Dissemination of lymphoma not assessed</td>
</tr>
<tr>
<td>MO</td>
<td>No evidence of extranodal dissemination</td>
</tr>
<tr>
<td>M1</td>
<td>Non-continuous involvement of separate site in gastrointestinal tract (e.g., stomach and rectum)</td>
</tr>
<tr>
<td>M2</td>
<td>Non-continuous involvement of other tissues (e.g., peritoneum, pleura) or organs (e.g., tonsils, parotid gland, ocular adenexa, lung, liver, spleen, kidney, breast etc.)</td>
</tr>
<tr>
<td>BX</td>
<td>Involvement of bone marrow not assessed</td>
</tr>
<tr>
<td>BO</td>
<td>No evidence of bone marrow involvement</td>
</tr>
<tr>
<td>B1</td>
<td>Lymphomatous infiltration of bone marrow</td>
</tr>
</tbody>
</table>

*Valid for lymphomas originating from the gastro-oesophageal junction to the anus (as defined by identical histopathological structure).
†In case of more than one visible lesion synchronously originating in the gastrointestinal tract, give the characteristics of the more advanced lesion.
‡Anatomical designation of lymph nodes as "regional" according to site:
(a) stomach: perigastric nodes and those located along the ramifications of the coeliac artery (that is, left gastric artery, common hepatic artery, splenic artery) in accordance with compartments I and II of the Japanese Research Society for Gastric Cancer (1995);
(b) duodenum: pancreaticoduodenal, pyloric, hepatic, and superior mesenteric nodes;
(c) jejunum/ileum: mesenteric nodes and, for the terminal ileum only, the ileocolic as well as the posterior caecal nodes;
(d) colonrectum: pericolic and perirectal nodes and those located along the ileocecocolic, right, middle, and left colic, inferior mesenteric, superior rectal, and internal iliac arteries.
Case report
A 52 year old woman was referred to our unit for epigastric pain and anorexia of two months’ duration. She also complained of nausea and a 2 kg weight loss. Her past medical history was unremarkable. She was a non-smoker and denied alcohol abuse or medication intake but remarked on overconsumption of warm black tea for several months (1.5–2 litres/day). Physical examination was normal. Laboratory data demonstrated normal ranges for electrolytes, liver function tests, blood count, urea, creatinin, C reactive protein, amylase, and lipase. Fasting blood glucose, triglycerides, cholesterol, and thyroid function tests were normal. Autoantibodies were negative. Serum CA19.9 was 1432 UI/ml (normal <37), and CEA was 2 ng/ml (normal <5).

Upper endoscopy, colonoscopy, and barium meal of the small bowel showed normal results. Abdominal ultrasonography and computed tomography scan found mild enlargement of the body of the pancreas without hepatobiliary abnormalities. Endoscopic ultrasonography showed no pancreatic malignancy or biliary abnormalities. The pancreas was homogenous and mildly enlarged in the body without pathological significance.

The patient was advised to stop tea consumption. Four weeks later she became asymptomatic and gained 2 kg weight. Another serum CA19.9 assay showed a considerable drop in levels to 42 UI/ml. A rechallenge test was then attempted. The patient restarted tea consumption as previously. Four weeks later, CA19.9 increased to 745 UI/ml followed by a fall to 25 UI/ml one month after withdrawal. Follow up one year later revealed no clinical abnormalities. Abdominal and chest computed tomography scan were normal.

Discussion

References
5 Liu H, Ye H, Ruskone-Fourmestraux A, et al. (111 In) is a marker for all stage gastric MALT lymphomas that will not respond to H pylori eradication. Gastroenterology 2002;202:1286–94.

A new cause for CA19.9 elevation: heavy tea consumption
Serum carbohydrate associated antigen (CA19.9) is a reliable tumour marker of biliarypancreatic malignancies. A number of benign diseases are also known to be related to CA19.9 elevation. Here we report a case of markedly raised levels of CA19.9 associated with heavy tea consumption.

Case report
A 52 year old woman was referred to our unit for epigastric pain and anorexia of two months’ duration. She also complained of nausea and a 2 kg weight loss. Her past medical history was unremarkable. She was a non-smoker and denied alcohol abuse or medication intake but remarked on overconsumption of warm black tea for several months (1.5–2 litres/day). Physical examination was normal. Laboratory data demonstrated normal ranges for electrolytes, liver function tests,
Non-alcoholic steatohepatitis and hepatic steatosis in patients with adult onset growth hormone deficiency

Adult onset growth hormone (GH) deficiency closely resembles syndrome X. Patients with syndrome X often suffer from obesity, dyslipidemia, insulin resistance, and hypertension, and hepatic steatosis and non-alcoholic steatohepatitis (NASH) have also been characterised as symptoms of syndrome X. The relationship of GH deficiency to hepatic steatosis and NASH remains unclear.

We looked for the presence of hepatic steatosis using computer tomography (CT) in a study of 18 patients with adult onset anterior lobe pituitary hormone deficiency, with or without GH deficiency. We also performed a liver biopsy in one patient with adult onset GH deficiency. None of the patients was positive for hepatitis B surface antigen, hepatitis C virus antibody, antinuclear antibody, or antimitochondrial antibody. The criterion used for diagnosing hepatic steatosis was a ratio for the liver/spleen CT value of less than 0.9. In 13 patients with GH deficiency, seven showed hepatic steatosis (53.8%) while in five patients without GH deficiency no hepatic steatosis was present (p=0.035). Body mass index (BMI kg/m²), serum triglyceride level (TG mg/dl), and serum total cholesterol (T-ch mg/dl) were similar in patients with or without GH deficiency. Of the 18 patients, 11 had GH deficiency and seven did not. The patient did not have a history of alcohol abuse but was slightly obese (BMI 30 kg/m²). Liver biopsy was performed when the patient was 72 years old, and this showed the presence of NASH (fig 1).

We have demonstrated that hepatic steatosis is more frequently observed in patients with GH deficiency than in those without GH deficiency. Furthermore, by liver biopsy, we have shown that a patient with GH deficiency also had NASH. These results indicate that adult onset GH deficiency is a possible risk factor for hepatic steatosis and NASH.

T Ichikawa, K Hamsasaki, H Ishikawa, E Ejima, K Eguchi
First Department of Internal Medicine, Nagasaki University School of Medicine, Nagasaki, Japan

References


Expression of thiopeurine methyltransferase in South Asians

Azathioprine and its metabolite 6-mercaptopurine (6-MP) are widely used in the treatment of Crohn’s disease, ulcerative colitis, autoimmune hepatitis, and a range of other inflammatory and autoimmune diseases, as well as for prevention of transplant rejection. Thiopeurine methyltransferase (TPMT) converts 6-MP to 6-methylIMPDH, which is responsible for the expression of the enzyme. TPMT is genetically polymorphic and treated patients to bone marrow depression; it may also enhance the efficacy of thiopeurines by increasing their metabolism to thioguanine nucleotides. Previous reports in Caucasians have shown that approximately 0.3% are homozygous and 10% heterozygously deficient in TPMT.

Expression of thiopeurine methyltransferase in South Asians

Azathioprine and its metabolite 6-mercaptopurine (6-MP) are widely used in the treatment of Crohn’s disease, ulcerative colitis, autoimmune hepatitis, and a range of other inflammatory and autoimmune diseases, as well as for prevention of transplant rejection. Thiopeurine methyltransferase (TPMT) converts 6-MP to 6-methylIMPDH, which is responsible for the expression of the enzyme. TPMT is genetically polymorphic and treated patients to bone marrow depression; it may also enhance the efficacy of thiopeurines by increasing their metabolism to thioguanine nucleotides. Previous reports in Caucasians have shown that approximately 0.3% are homozygous and 10% heterozygously deficient in TPMT.

One study
suggests that the frequency of individuals with the mutant TPMT allele, TPMT*3A, is lower in Southwest Asians than in Caucasians, allele causes reduced enzyme activity in vitro.1 However, there are no data on the phenotypic expression of TPMT in South Asian populations.

We have compared expression of TPMT in South Asian and Caucasian patients attending gastroenterology, renal, rheumatology, and dermatology clinics who were being considered for treatment with thiopurines. TPMT activity was assayed by tandem mass spectrometry for 6-methylMP (assays kindly provided by Purine Research Laboratory, Guy's Hospital, London SE1 9RT, UK). Homozygous deficiency of TPMT was defined as a level <10, heterozygous as 10–25, and normal as > 25 pmol/h/mg haemoglobin.

Of 83 Caucasian patients, one (1.3%) was homozygously deficient, 10 (12%) were heterozygous, and 72 (86.7%) had normal expression of TPMT. The corresponding values in 77 South Asian patients were 0.7 (9%), and 70 (91%), respectively.

The prevalence of deficiency of expression of TPMT in South Asian patients resembles that in Caucasians. Use and monitoring of azathioprine and 6-MP should therefore follow similar principles in both ethnic groups.

E Tsironi, M Browne, D S Rampton, A B Ballingar
Academic Department of Adult and Paediatric Gastroenterology, Barts and the London, Queen Mary's School of Medicine and Dentistry, and Department of Clinical Biochemistry, Barts and the London NHS Trust, London, UK.

Correspondence to: Dr E Tsironi, Barts and the London NHS Trust, London E1 1BB, UK. etsironi@yahoo.com

References

Fatigue is associated with high circulating leptin levels in chronic hepatitis C

We read with great interest the recent paper from Piche and colleagues (Gut 2002;51:434–49) relating serum leptin levels to fatigue in chronic hepatitis C patients. On the surface, their observation that serum leptin levels correlate with the fatigue impact scale are very intriguing. However, leptin shows a gender based observation that serum leptin levels correlate with leptin levels, and multivariate analysis the authors cannot affirm that serum leptin levels are implicated in fatigue development in patients with chronic hepatitis C.

M Romero-Gómez, D Sánchez-Muñoz
Hepatology Unit, Hospital Universitario de Valme, Seville, Spain
M Cruz
Biochemistry Unit, Hospital Universitario de Valme, Seville, Spain

Correspondence to: Dr M Romero-Gómez, C/Juan Antonio Cavastray 1 4ºC, 41003 Sevilla, Spain; mromerg@supercable.es

Mutant K-ras2 in serum

Ryan and colleagues' careful and well conducted study (Gut 2003;52:101–8) raises once again the interesting issue of whether molecular analysis and knowledge of mutations of the Kirsten ras gene in particular, have a role in the management of patients with colorectal cancer.

The two RASCAL (Kirsten Ras in Colorectal Cancer Collaborative group) studies11 which eventually enrolled data from 4266 patients from 42 centres in 21 countries showed that although the frequency of Kirsten ras mutations at codons 12 and 13 may vary a little between populations, overall they are only present in just over one third of patients. This is significantly less frequent than quoted by Ryan and colleagues.11 In addition, the RASCAL study also showed that Kirsten ras mutations are not associated with sex, age, tumour site, or Dukes' stage.

Much more importantly, however, they also showed that of the 12 different possible specific point mutations at codons 12 or 13, only a guanine to thymidine mutation, which are found in less than 10% of all patients, is an independent variable for relapse and death. Indeed, the most common mutation, guanine to adenine, exerted an effect on survival barely different from wild-type ras. Others have shown there is a reasonable biological basis for these findings.9 As a collaborative group, therefore, we felt it was generally unhelpful to consider Kirsten ras mutations collectively, rather than the effect of each individual mutation separately when considering the prognosis of patients with colorectal cancer. With this in mind, we believe that two crucial questions are left unanswered by the study of Ryan et al.11 Without definitive answers to these, it is unlikely that serum detection of mutated Kirsten ras gene will ever be clinically useful.

Firstly, firstly, of any new mutation in the serum of a patient who has had colorectal cancer raises the possibility of tumour recurrence, although the presence of a Kirsten ras mutation is not colon specific. However, more than one third of patients in Ryan's study did not clinically relapse despite the presence of a serum mutation for very long periods. Others have also detected tumour DNA in patients for long periods without evidence of overt clinical recurrence.12 Therefore, screening for the presence of Kirsten ras mutations per se is unlikely to be clinically helpful very soon.

However, it would have been very interesting to know whether the patients who did not relapse in this study were those who had the RASCAL group defined as "benign" rather than "aggressive" mutations.

Secondly, the authors do not say whether each postoperative serum mutation which they detected corresponded with the genotype of the primary tumour. While a number of studies have suggested that occasional metastases may carry a different mutation to the primary, this is very uncommon. If the mutation is not the same as the primary tumour, this raises many issues, not least that despite the very careful use of controls, nested polymerase chain reaction techniques may be too sensitive for clinical practice.

For an individual with colorectal cancer, a multitude of factors may shape the clinical course. To improve our knowledge, we must seek to study the most subtle variation in molecular profiles possible, as small differences may be very important. While rigorous small prognostic clinical studies may be intriguing, they may have significant limitations. Some of us have argued previously that there is only one way forward.2 All bodies funding prospective therapeutic studies in colorectal cancer should insist that a panel of molecular markers are measured and recorded in all patients using a standardised technique, even if they are not included as an end point, and make these results readily available for subsequent analysis. Studies of this nature which and which are performed prospectively carry the least risk of methodological flaws.

H J N Andreyev
Imperial College, London UK
R Benamouzig
Hospital Avicenne, Bobigny, France
M Beranek
Charles University Hospital, Hradec Kralove, Czech Republic
P Clarke
Institute of Cancer Research, Sutton, UK
D Cunningham, A R Norman
Royal Marsden Hospital, London UK
W Giaretti
Istituto Nazionale per la Ricerca sul Cancro, Genova, Italy
A F P M de Goeij
Maastricht University, the Netherlands
B J Jacopetta
University of Western Australia, Nedlands, Australia
E Jullian
Groupe Hospitalier Cochin-Saint Vincent de Paul, Paris, France
K Krtolica
Institute of Nuclear Sciences “Vinca”, Belgrade, Yugoslavia
J Q Lee, S T Wang
National Cheng Kung University, Taiwan
N Lees
Wythenshawe Hospital, Manchester, UK
F Al-Mulla
Kuwait University, Kuwait
O Muller
Max-Planck-Institut fur Molekulare Physiologie, Dortmund, Germany
M Pauly
Centre Universitaire de Luxembourg, Luxembourg
V Pricolo
Rhode Island Hospital, Brown University, USA
A Russo
University of Paderno, Italy
C Truong
University of Athens, Greece
Radiological Imaging of the Small Intestine


Springer Verlag are producing an extensive range of Medical Radiology books under the general editorship of Professor LB Baert, and this volume on the small intestine is the latest in the line. Professor NC Gourtsoyiannis is its distinguished editor. This is now a very well established series with topics which vary from organ based (that is, pancreas) to technique based (that is, spiral computed tomography (CT)) of the abdomen. As with all Springer publications, the illustrations are outstanding and the more than 800 in this book are no exception.

Imaging of the small intestine remains the last redoubt for the gastrointestinal radiologist due to the remorseless advance of endoscopy from both ends of the alimentary tract; a point noted in the foreword by Professor Baert. Virtual colonography has rekindled interest in the large bowel and the “knee jerk” response of biopsy is still there. Professor NC Gourtsoyiannis, however, concludes that this is primarily North American with some distinguished North American contributors. Indeed, two of the editors of Clinical Imaging are also authors of this book.

The first chapter in Clinical Imaging of the Small Intestine by Herlting, Maglinte, and Birnbaum, interestingly also published by Springer. This is the more mature book, now in its second edition, having been originally published in 1989. Its authorship is primarily North American with two European contributors whereas Radiological Imaging is primarily European with some distinguished North American contributors. Indeed, two of the editors of Clinical Imaging are authors. Both books go for the same layout—that is, chapters which are technique based follow those on pathological states. Both are totally comprehensive and although Clinical Imaging is slightly larger (576 pp versus 477 pp) it is less expensive (£164 versus £214). This price differential is most likely to be due to the inclusion of 414 beautiful colour illustrations of both endoscopic views and pathology specimens in Radiological Imaging. This is also the most recent publication (2002 versus 1999) and in areas where there is rapid technical change—typically MR imaging—the reader can appreciate the difference. It has to be said that both books fulfil their remit extremely well but your reviewer, if he had to choose, would opt for the extra Euros.

Professor Gourtsoyiannis and his team have to be congratulated on producing a superb book that graces the swelling ranks of medical radiology. A must for every radiology department and a continuing source of information for any radiologist with an interest in the gastrointestinal system.

A H Freeman

NOTICES

New In Vivo Imaging Modalities for Molecular Biology, Cell Biology and Physiology

This Jacques Monod conference will be held on 31 May–4 June 2003 in Roscoff, France. Further information: Bertrand Tavitian, IN- SERM M150103, Service Hospitalier Frédéric Joliot, CEA Direction des Sciences du Vivant, Direction de la Recherche Médicale, 4 place du Général Leclerc, 91401 Orsay Cedex, France. Tel: +33 169 867 779; fax: +33 169 867 739; email: tavitian@slhj.cea.fr

Prague Hepatology Meeting

To be held on 5–7 June 2003 in Prague, Czech Republic. Leading speakers from Europe and the USA will present new ideas and suggestions on pathophysiology, diagnostics, and therapy of liver diseases in ten programmes. Further details: Ms Veronika Revicka. Tel: +420 241 445 759; fax: +420 241 445 806; email: veronika@congressprague.cz

8th International Liver Symposium

This symposium will be held on 13 June 2003 in Manchester, UK. Leading speakers from the USA and Europe will present state of the art reviews on recent advances in hepatology. Further details: Professor TW Warnes, Department of Gastroenterology, Manchester Royal Infirmary, Oxford Road, Manchester M3 9WL, UK. Tel: +44 (0)161 276 4316; fax: +44 (0)161 276 8779; email: judith.harrop@cmic.nhs.uk

Gastroenterology and Endotherapy: XXIst European Workshop

This will be held on 16–18 June 2003 in Brussels, Belgium. Further details: Nancy Beauprez, Administrative Secretariat of the Workshop, Gastroenterology Department, Erasme Hospital, Route de Lennik 808, B-1070 Brussels, Belgium. Tel: +32 (0)2 555 49 00; fax: +32 (0)2 555 49 01; email: beauprez@ulb.ac.be

The Association of Coloproctology of Great Britain & Ireland

This annual meeting will be held on 7–10 July 2003 in Edinburgh, UK. Further details: Conference Secretariat, The AGCBI at the Royal College of Surgeons of England, 35–43 Lincoln’s Inn Fields, London WC2A 3PE. Tel: +44 (0)20 7973 0307; fax: +44 (0)20 7430 9235; email: acgbi@asgb.org.uk; website: www.acgbi.org.uk

European Helicobacter Study Group (EHSG)

This meeting, on Helicobacter infections and gastrroduodenal pathology, will be held on 3–6 September 2003 in Stockholm, Sweden. Further details: Professor Torkel Wadstrom, President- EHSG, Lund University, Department of Infectious Diseases & Medical Microbiology, Division of Bacteriology, Solvegatan 23, SE-223 62 Lund, Sweden. Tel: +46 46 173 241; fax: +46 46 152 564; email: torkel.wadstrom@mnbb.lu.se; website: www.helicobacter.org

BOOK REVIEW

Radiological Imaging of the Small Intestine


Springer Verlag are producing an extensive range of Medical Radiology books under the general editorship of Professor LB Baert, and this volume on the small intestine is the latest in the line. Professor NC Gourtsoyiannis is its distinguished editor. This is now a very well established series with topics which vary from organ based (that is, pancreas) to technique based (that is, spiral computed tomography (CT)) of the abdomen. As with all Springer publications, the illustrations are outstanding and the more than 800 in this book are no exception.

Imaging of the small intestine remains the last redoubt for the gastrointestinal radiologist due to the remorseless advance of endoscopy from both ends of the alimentary tract; a point noted in the foreword by Professor Baert. Virtual colonography has rekindled interest in the large bowel and the “knee jerk” response of biopsy is still there. Professor NC Gourtsoyiannis, however, concludes that this is primarily North American with some distinguished North American contributors. Indeed, two of the editors of Clinical Imaging are authors. Both books go for the same layout—that is, chapters which are technique based follow those on pathological states. Both are totally comprehensive and although Clinical Imaging is slightly larger (576 pp versus 477 pp) it is less expensive (£164 versus £214). This price differential is most likely to be due to the inclusion of 414 beautiful colour illustrations of both endoscopic views and pathology specimens in Radiological Imaging. This is also the most recent publication (2002 versus 1999) and in areas where there is rapid technical change—typically MR imaging—the reader can appreciate the difference. It has to be said that both books fulfil their remit extremely well but your reviewer, if he had to choose, would opt for the extra Euros.

Professor Gourtsoyiannis and his team have to be congratulated on producing a superb book that graces the swelling ranks of medical radiology. A must for every radiology department and a continuing source of information for any radiologist with an interest in the gastrointestinal system.

A H Freeman

NOTICES

New In Vivo Imaging Modalities for Molecular Biology, Cell Biology and Physiology

This Jacques Monod conference will be held on 31 May–4 June 2003 in Roscoff, France. Further information: Bertrand Tavitian, INSERM M150103, Service Hospitalier Frédéric Joliot, CEA Direction des Sciences du Vivant, Direction de la Recherche Médicale, 4 place du Général Leclerc, 91401 Orsay Cedex, France. Tel: +33 169 867 779; fax: +33 169 867 739; email: tavitian@slhj.cea.fr

Prague Hepatology Meeting

To be held on 5–7 June 2003 in Prague, Czech Republic. Leading speakers from Europe and the USA will present new ideas and suggestions on pathophysiology, diagnostics, and therapy of liver diseases in ten programmes. Further details: Ms Veronika Revicka. Tel: +420 241 445 759; fax: +420 241 445 806; email: veronika@congressprague.cz

8th International Liver Symposium

This symposium will be held on 13 June 2003 in Manchester, UK. Leading speakers from the USA and Europe will present state of the art reviews on recent advances in hepatology. Further details: Professor TW Warnes, Department of Gastroenterology, Manchester Royal Infirmary, Oxford Road, Manchester M3 9WL, UK. Tel: +44 (0)161 276 4316; fax: +44 (0)161 276 8779; email: judith.harrop@cmic.nhs.uk

Gastroenterology and Endotherapy: XXIst European Workshop

This will be held on 16–18 June 2003 in Brussels, Belgium. Further details: Nancy Beauprez, Administrative Secretariat of the Workshop, Gastroenterology Department, Erasme Hospital, Route de Lennik 808, B-1070 Brussels, Belgium. Tel: +32 (0)2 555 49 00; fax: +32 (0)2 555 49 01; email: beauprez@ulb.ac.be

The Association of Coloproctology of Great Britain & Ireland

This annual meeting will be held on 7–10 July 2003 in Edinburgh, UK. Further details: Conference Secretariat, The AGCBI at the Royal College of Surgeons of England, 35–43 Lincoln’s Inn Fields, London WC2A 3PE. Tel: +44 (0)20 7973 0307; fax: +44 (0)20 7430 9235; email: acgbi@asgb.org.uk; website: www.acgbi.org.uk

European Helicobacter Study Group (EHSG)

This meeting, on Helicobacter infections and gastrroduodenal pathology, will be held on 3–6 September 2003 in Stockholm, Sweden. Further details: Professor Torkel Wadstrom, President- EHSG, Lund University, Department of Infectious Diseases & Medical Microbiology, Division of Bacteriology, Solvegatan 23, SE-223 62 Lund, Sweden. Tel: +46 46 173 241; fax: +46 46 152 564; email: torkel.wadstrom@mnbb.lu.se; website: www.helicobacter.org