LETTERS

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Long term follow up of Helicobacter pylori induced gastric diffuse large B cell MALT lymphoma following eradication treatment alone

I was interested to read the article by Alsolaiman and colleagues on the long term follow up of gastric diffuse large B cell lymphoma after eradication of Helicobacter pylori (Gut 2003; 52: 507–9).

Gastric lymphomas represent approximately 5% of all gastric malignancies and are frequently due to mucosa associated lymphoid tissue (MALT) B cell gastric lymphomas. Acquired MALT due to H pylori infection provides the tissue of origin for the B cell lymphoma. Monoclonal proliferation of B cells in the germinal centres of lymphoid tissue with epithelial invasion—“lymphoepithelial lesions”—are the histological hallmark of MALT lymphoma. H pylori induced chronic gastritis through genetic mutation of trisomy 3 and 18 leads to the chronic gastritis, as often is employed in the case of low grade gastric MALT lymphoma.1

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References

Is hepatobiliary scintigraphy insensitive for the diagnosis of sphincter of Oddi dysfunction?

I was very pleased to read the letter by Dr Madaccy in response to our article “Scintigraphy versus manometry in patients with suspected sphincter of Oddi dysfunction” (Gut 2003;52:352–7). The major criticism of our study refers to the change from the original study of Sostre and colleagues (Gut 2000;50:1385–9). We were of the opinion that the use of cholecystokinin (CCK) as a trigger for sphincter of Oddi manometry remains the only objective means of selecting patients with sphincter of Oddi dysfunction who may benefit from treatment. At present, we are developing a new catheter assembly system for manometric recording of the sphincter of Oddi, which we believe will eliminate the risk of pancreatitis. This catheter may replace the endoscopy procedure and may become the new standard while we await the development of non-invasive reproducible diagnostic tests of sphincter of Oddi dysfunction.

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References

Is hepatobiliary scintigraphy insensitive for the diagnosis of sphincter of Oddi dysfunction?

We read with interest the article by Craig and colleagues (Gut 2003;52:352–7) who reported disappointing results on the value of quantita tive hepatobiliary scintigraphy (QHBS) in patients with a suspected sphincter of Oddi dysfunction (SOD). As our paper documented, the contrary results was referred to, we must add a few words of comment.

Firstly, it should be emphasised that in patients with SOD there is an up to fivefold risk of post-endoscopic retrograde cholangiopancreatography and post-manometry pancreatitis, and therefore there is a strong need for any objective non-invasive method. Hence it is crucial to known whether QHBS can be applied to predict abnormal manometric results. Two European groups recently published concordant results1 which clearly showed abnormal results of QHBS and endoscopic sphincter of Oddi manometry (ESOM). These findings and those of Craig et al are so different that there must be some explanation. We believe this may be due to differences in study design and cholecystokinin (CCK) administration in particular.

The Australian group changed the CCK augmentation method during QHBS, as originally suggested by Sostre and colleagues; whereas Sostre’s group injected a short three minute bolus of 20 ng/kg/body weight of CCK octapeptide (CCK-OP), completed 12 minutes before injection of QHBS, Craig et al infused 20 ng/kg/body weight CCK-OP over 45 minutes, starting 15 minutes before QHBS, and continued the infusion.

Table 1 T 1/2 parameter of common bile duct emptying, measured by scintigraphy

<table>
<thead>
<tr>
<th>QHBS (min)</th>
<th>QHBS+CCK (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>43</td>
</tr>
<tr>
<td>SD</td>
<td>23</td>
</tr>
</tbody>
</table>

QHBS, quantitative hepatobiliary scintigraphy; CCK, cholecystokinin.
during the first 30 minutes of the QHBS study. The authors believe that the modification had no effect on the scan. We disagree, as from a scintigraphic methodological aspect, the first 30 minutes of QHBS after radiotracer administration is critical. In cholecystectomised subjects, most of the radiotracer has been emptied from the biliary tree into the duodenum after 30 minutes. Once the tracer is in the duodenum, no further information is available on SO function and resistance. Mandour et al. clearly reveals that CCK-OP has a relaxing effect on the SO. 2 In scintigraphic terms, transient SO relaxation means rapid tracer emptying. Moreover, a paradoxical SO response after CCK-OP is a rare phenomenon, occurring in less than 25% of all SOD patients. 3

Therefore, in most SOD patients with an elevated SO basal pressure, CCK-OP induces a significant pressure drop, as demonstrated by Hogan and Geenen. 4 CCK-OP administration during QHBS must therefore be regarded as a relaxed, tonically relaxed SO. 5

We administered CCK during QHBS, 60 minutes after radiotracer administration, to demonstrate the reversibility of SO obstruction and to visualise baseline SO function before CCK-OP. 6 We thereby proved significant acceleration of transpapillary bile flow by QHBS after CCK-OP as compared with the baseline study in 37 patients with suspected SOD, as demonstrated in table 1. 7

In common with the study of Craig et al., we recently compared our scintigraphic (without CCK-OP) and manometric results. 3 Compari- son of our results with those of Craig et al reveals that a continuous CCK-OP infusion during QHBS might uniformly accelerate transpapillary bile flow, thus masking basal bile flow differences in SOD patients. As a net result, Craig et al achieved very high specificity at a cost of a low sensitivity as compared with our levels (table 2). Therefore, instead of continued debate in this field with results of small studies in different centres with different study designs, we suggest initiation of a large multicentre study for the non-invasive diagnosis of SOD as compared with manometry.

Table 2 Sensitivity and specificity of scintigraphic results as compared with manometry

<table>
<thead>
<tr>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>PPV (%)</th>
<th>NPV (%)</th>
<th>Accuracy (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>QHBS positive</td>
<td>79</td>
<td>71</td>
<td>88</td>
<td>55</td>
</tr>
<tr>
<td>CBD&gt;12 mm</td>
<td>42</td>
<td>86</td>
<td>89</td>
<td>35</td>
</tr>
<tr>
<td>Abnormal LFT</td>
<td>26</td>
<td>71</td>
<td>71</td>
<td>23</td>
</tr>
</tbody>
</table>
| QHBS, quantitative hepatobiliary scintigraphy; CBD, common bile duct; LFT, liver function test; PPV, positive predictive value; NPV, negative predictive value.


Worsening of steatosis and fibrosis progression

We read with great interest the article by Casterra and colleagues (Gut 2003;52:288–92) and acknowledge that worsening of steatosis in chronic hepatitis C is associated with fibrosis progression. However, in our view there are no data supporting a causal role for this correlation or any specific relation of this finding to chronic hepatitis C.

Firstly, the authors provide no explanation as to why steatosis worsened in patients under consideration. Overweight, diabetes, and alcohol consumption are the main causes of steatosis in Western countries and major causes of fibrotic liver disease. There are no data throughout the study indicating whether patients in whom steatosis worsened simply gained weight or developed any of the complications associated with insulin resistance. The latter can develop within the course of liver injury well before cirrhosis is present1 or be epidemiologically linked to infection by hepatitis C virus (HCV) for reasons that have yet to be determined. 1 High serum glucose, as well as diabetes, 2 are associated with liver fibrosis progression 3 and might contribute to enhanced fibrogenesis. 4 For alcohol consumption, a thorough evaluation is needed before ruling out the possibility of even slight increases in daily alcohol consumption trans- lating, over the course of several years, into enhanced steatosis. There is a theoretical possibility that progression of steatosis reflects the natural course of HCV infection if steatosis were to occur later than the necroinflammatory lesions defining chronic hepatitis. However, as current knowledge stands, this is purely speculative and also, there is no indication in this study that patients in whom steatosis progressed had a longer duration of infection than those in whom it did not. Hence there appears to be no data in this study suggesting that progression of steatosis in chronic hepatitis C is HCV-related or that confounding prostea- togenic factors have been ruled out.

The second issue is that it has not been made entirely clear what “worsening” of steatosis means. This was defined as an increase of at least one point on a grading scale that is not evenly distributed (0%; 1–10%; 10–30%; >30%). Since many patients had no steatosis on the first biopsy, such a definition would mean that in most cases an increase from 0% to 5% would be qualified as “worsening” of steatosis. This may explain the authors’ state- ment that “there were less patients with pro- gression of steatosis than patients with steatosis appearance between the two biop- sies”. In any event, the biological relevance of minor increases in the amount of steatosis appears highly improbable, especially if the total amount of steatosis that was worsened was not associated with the amount of fibro- sis, as noted in this study. This biological relevance could have been strengthened had the authors provided quantitative data on a correlation between progression of steatosis and progression of fibrosis.

Although the idea that steatosis progresses rather than the amount of steatosis associated with fibrosis progression warrants further study, it is hard to reconcile with lessons from non-alcoholic fatty liver disease where patients with massive steatosis do not develop liver fibrosis whereas they obviously experienced steatosis progression. This argues against a simple and direct link between stea- tosis and fibrosis. We propose an alternate view in which both steatosis and fibrosis are the result of a common underlying condition, insulin resistance, which operates through proinflammatory mediators to enhance fibro- genic pathways and through alterations in metabolic pathways to promote steatosis.

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References


6 Fehrenbach H, Weiskirchen R, Kasper M, et al. Up-regulated expression of the receptor for advanced glycation end products in cultured rat hepatic stellate cells during post-
Colorectal screening guidelines in acromegaly

We write with concern regarding the recent “screening guidelines for colorectal cancer and polyps in patients with acromegaly” (Gut 2002;51(suppl V):V13–14). While there is little doubt that patients with acromegaly have an increased risk of developing colorectal cancer, the exact nature of this risk is far from clear. The endocrine literature has witnessed a significant debate, polarising two separate views. Jenkins and Fairclough advocate screening, while Rennahan et al suggest that the risk of colorectal cancer formation does not warrant screening or surveillance. The recommendations by Jenkins and Fairclough for a five yearly interval, to correspond to the 13–14-fold increase in the risk of colorectal cancer in acromegics relative to the general population, This is at odds with larger studies (e.g., ACC, n = 1041, n = 1634) which overall indicate an increased colorectal cancer risk of 2.5–3-fold. Jenkins and Fairclough advocate an intensive screening protocol beginning at 40 years of age (a case occurring at 39 years although mean age of the 10 patients with cancer was 67 years). They advocate repeat colonoscopy at five years, or three yearly if at increased risk (as determined by atrophy at initial colonoscopy or increased IGFI–1 levels). Rennahan et al however conclude that there is no increased incidence of colorectal adenomas compared with a normal control population (generated from postmortem and colonoscopy data). These data may differ because of the colonoscopy rate and this probably also explains their variance with the larger studies. Based on the current literature, an independent view is that patients might benefit from a single sigmoidoscopy or colonoscopy at approximately 55 years of age. However, no study suggests a risk of proximal neoplasia that warrants the risks and difficulties of a colonoscopy. Guidelines for familial colorectal cancer screening suggest that colonoscopic surveillance is only warranted for a lifetime risk of 1 in 10 or greater.

It is therefore both worrying and disappointing that the published guidelines reflect only one point of view of a very polarised debate. This may reflect the process of guideline formation in which experts were required to submit guidelines. At present there is insufficient data to advocate an intensive colorectal cancer screening programme for patients with acromegaly. The increased risk of colorectal cancer is modest and the potential risk of colorectal cancer in acromegalic patients is considerable. These guidelines need to be challenged before gastroenterologists are forced into a practice which is not evidence based and may be detrimental to patient well being.

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References

Authors’ reply
We thank Drs Perry and Kane and Professor Stewart for their contribution to the debate on this topic. The correspondents acknowledge that patients with acromegaly have an increased risk of developing colorectal cancer; the question really concerns the magnitude of the cancer risk and the relative risk of colonoscopy.

As stated in the guidelines, our recommendations were based on aggregated data from a total of 13 prospective colonoscopic studies involving almost 700 patients with acromegaly. The relative risk was derived from the prevalence of colorectal cancer in patients with acromegaly compared with the asymptomatic matched control populations in the same studies. On this basis, there is also a clear increase in the risk of tubular adenomas. We believe this to be the best quality data on which to base recommendations. We have previously given the reasons why we think that control data generated from the postmortem studies referred to by Rennahan et al are of poorer quality.

One of the major aims of a screening programme is to prevent the development of colorectal cancer by detection and removal of adenomas. We therefore feel that it is sensible to begin this preventative screening at the age of 40 years in this group of patients who seem to be at relatively high risk. The recommendations will probably change in the light of further information about the pathophysiology and behaviour of colorectal neoplasia in acromegaly and could prove to have been over cautious. However, until then, a policy of early screening examinations and data collection seems prudent.

In addition to our own observations, several other studies have reported an increased prevalence of right sided colonic neoplasia in acromegaly. We therefore believe it would be unwise to accept the suggestion that a single sigmoidoscopy is a sufficient screening procedure. At this stage, full colonoscopy is warranted, especially in the case of the individual and for the sake of providing a firm basis on which to make recommendations. Furthermore, on the rare occasions on which colonoscopy is not completed, either a barium enema or a virtual colonoscopy should be performed. The risks of colonoscopy in good hands are minimal. We have not encountered a single complication in over 500 procedures in patients with acromegaly but clearly these are not procedures to be undertaken by the inexperienced. In this context the innumerable reports have detected 10 asymptomatic cancers, as well as numerous adenomas. Thus based on the literature and on our own experience, the risk of undetected colorectal cancer far outweighs the theoretical risks of colonoscopy.

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Atrophic gastritis: pathology and endoscopy in the reversibility assessment

We read with interest the paper by Walker (Gut 2003;52:1–4). We agree that histology remains the most suitable test for both detecting and assessing reversion of atrophic gastritis. Such a view elicits two basic questions, however: (1) are pathologists in recognising gastric atrophy? and (2) in Walker’s words, “where to biopsy?” and, we might add, “how extensively to biopsy?” to correctly categorise any presence/ regression of gastric atrophy?

Concerning the first point on the classification of atrophic gastritis, the current literature is largely biased by the inconsistency of the histological criteria used to categorise atrophy.7 To amalgamate the different viewpoints and also test interobserver agreement in atrophy classification, we formed an international group of pathologists recently published an extensive description of the different Phenotypes of gastric atrophy. By merging Western and Eastern experiences, the new proposal extensively describes the diagnostic categories that should be adopted to enable acceptable comparisons between clinicopathological studies involving gastric atrophy (both non-metaplastic and metaplastic). The proposed classification also introduces a new diagnostic category (that is, indeterminate for atrophy) to allow the evaluation of atrophy when high grade inflammation—mostly related to Helicobacter pylori infection—interferes with a reliable assessment of the “loss of appropriate glands.”

As for the number and location of biopsies for atrophy assessment, the recommendations of the “22 guidelines” seem a suitable compromise between the excessive pathologists’ demands and the operating limits of routine practice.

The question of “where to biopsy” is more intriguing. No doubt both the oxyntic and antral mucosa need to be tested, but endoscopists too often neglect the recommendation to take an additional angular sample.8 We studied 504 consecutive H pylori positive patients who underwent gastroscopy for untreated non-ulcer dyspepsia. In all patients, biopsies were obtained (Pentax, Japan: KW2415S) from: (i) oxyntic mucosa (one biopsy from the lesser curvature 4–6 cm proximal to the anulus and one from the greater curvature 4–6 cm) and (ii) antral mucosa (one biopsy each from the greater and lesser curvers, 3–5 cm proximal to the pyloric ring), and (iii) only one additional biopsy from the antrum. Histological categories included the basic distinction between non-atrophic and atrophic gastritis. Two pathologists independently

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Correa; the authors thank Professor Correa for his.

The results of this study were discussed with Pelayo.

Acknowledgements

The results of this study were discussed with Pelayo Correa; the authors thank Professor Correa for his valuable advice.

References

2 Gent MA. “We used the Sydney System...”. Am J Gastroenterol 1997;92:1960–1.

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Assessed the biopsies with a 93% consistency (Fleiss’ $K = 0.91$). Table 1 shows the atrophy prevalence according to biopsy location.

In this series, the importance of sampling the incisura angularis is emphasised by the percentage of atrophic gastritis (46%) that would have been missed if sampling had not included the angular mucosa. The NND (number needed to diagnose) values in detecting atrophy calculated for incisura, antrum, and corpus sampling were 2.17, 4.85, and 63.29, respectively.

Because of different pricing policies in different countries, it is difficult to estimate the additional cost of processing and interpreting the extra biopsy sample taken from the incisura angularis. In most countries, however, the cost of the endoscopy procedure is far higher than the cost of histological examination, and the price of an angular biopsy seems amply balanced by the benefit of an appropriate assessment of gastric disease and a suitable evaluation of the patient’s cancer risk.

Table 1 By site prevalence of atrophic gastritis (distinguishing between atrophy with and without IM) in 504 consecutive Helicobacter pylori positive patients. Patients with phenotype “indefinite for atrophy” and/or “foveolar restricted IM” were included among cases of non-atrophic gastritis.

<table>
<thead>
<tr>
<th>By site prevalence of gastric atrophy (504 H pylori positive consecutive patients)</th>
<th>Gastric atrophy</th>
<th>IM absent</th>
<th>IM present</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Corpus only</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Antrum only</td>
<td>3</td>
<td>10</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>Antrum and incisura angularis</td>
<td>6</td>
<td>14</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>Incisura angularis only</td>
<td>9</td>
<td>20</td>
<td>29</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>18</td>
<td>45</td>
<td>63</td>
<td></td>
</tr>
</tbody>
</table>

IM, intestinal metaplasia
Incisura angularis, number of patients whose gastric atrophy (with or without IM) was detected only in the incisura biopsy samples.

Antrum, number of patients whose gastric atrophy (with or without IM) was detected only in the antral biopsy samples.

Antrum and incisura angularis, number of patients whose gastric atrophy (with or without IM) was detected in both the antral and the incisura angularis biopsy samples.

Corpus, number of patients whose gastric atrophy (with IM) was detected only in the corpus biopsy samples.

Diversion colitis with a mucosal tear on endoscopic insulation

I read with interest the report of Cruz-Correa and colleagues (Gut 2002;51:600). They described three cases of collagenous colitis with mucosal tears on endoscopic insufflation and stated that as far as they were aware there were no reports in other gastrointestinal diseases. We would like to present the case of a similar mucosal tear on endoscopic insufflation in a patient with diversion colitis.

A 46 year old Japanese man presented with an acute abdomen caused by ascending colon diverticular perforation. He underwent drainage of the abdominal cavity with loop colostomy. He had been suffering from systemic lupus erythematosus and chronic renal failure for 25 years. He had received more than 90 g of oral steroid at the time of referral and was taking 10 mg/day. After operation, he was free from symptoms and gave no history of haematemesis or blood in stools. On surveillance colonoscopy, the dysfunctional colon mucosa, which was 10 cm away from the loop colostomy, was torn with slight bleeding, and the muscularis mucosa was exposed on endoscopic insulation with air (fig 1). The lumen of the colon was narrowed and the remaining colon mucosa showed mild colitis with a decreased vascular pattern and oedema. The post endoscopic course was uneventful without any treatment. Routine laboratory investigations revealed: white blood cell count 10600/µl (normal range 4000–9000/µl), haemoglobin 12.2 g/dl (normal range 14–18 g/dl), haematocrit 36.1% (normal range 40–48%), blood urea nitrogen 76.4 mg/dl (normal range 9–21 mg/dl), and serum creatinine 4.29 mg/dl (normal range 0.6–1.2 mg/dl). Cultures for stool pathogens were negative.

Diversion colitis may occur in a part of the bowel that was previously healthy and which has been placed outside the faecal stream because of a proximal stoma. The mechanism of diversion colitis remains unclear but may be associated with changes in the intestinal bacterial flora, absence of essential nutrients, or intestinal toxins. In most cases, there are no symptoms, as in our case. Frisbie et al reported that mucosal erythema and friability were seen in most patients who had undergone diverting colostomy for neuropathic large bowel. Continuous high doses of steroids make human tissue fragile, including the colon mucosa. Taken together, these results

Figure 1 Endoscopic insulation of a diverted colon resulted in a mucosal tear.
suggest that the mucosal tear in our case may have been attributable to diversion colitis with fragile mucosa.

When performing surveillance colonoscopy for patients with a stoma, the dysfunctional colorectum must be surveyed with great care.

Viral hepatitis accounts for a substantial part of the observed liver diseases, especially viral hepatitis and autoimmune diseases. They have made a credited attempt to define distinct pathophysiological entities and produce drugs to correct them. The real world is not like this: pure, typical, or textbook IBS is unusual. Patients present with a variety of symptoms, originating from many systems, some of which may have features of IBS. Clinicians reading this book might, quite reasonably, ask themselves whether they can apply all of this IBS information to the patients they see in clinics. I suspect that the underlying issues are similar, regardless of the underlying symptoms, but this remains to be proven.

The synopsis claims this text to be an “updated and comprehensive description of the most relevant features of colorectal cancer ...” — I beg to differ.

In the preface, Dr de Leon states that he hopes the volume captures his spirit of the multiplicity of approaches and critical assessment. It is not surprising that viral hepatitis and autoimmune diseases have featured in many of the other chapters. Excellent stuff.

In the preface, Dr de Leon states that he hopes the volume captures his spirit of the multiplicity of approaches and critical assessment. It is not surprising that viral hepatitis and autoimmune diseases have featured in many of the other chapters. Excellent stuff.
book is aimed at giving an overview of colorectal cancer to people working in basic science on colorectal cancer then the book is short enough to be digestible. As an overview of colorectal cancer in the 21st century, a single author could not be expected to do justice to the whole topic and this text is not a comprehensive overview of colorectal cancer. The best sections of the book are not surprisingly those areas which Dr de Leon has written and published on himself, namely the genetics of colorectal cancer and chemoprevention of colorectal cancer. In many respects the excellent description of the state of the art in these areas highlights the inadequacies in other areas such as pathology, surgical technique, mesorectal excision, adjuvant chemotherapy, and the role of radiotherapy, which are covered in a superficial manner. With the exception of the genetics of colorectal cancer, the reviews of the literature are brief and highly selected. The section on adjuvant chemotherapy and the data presented on faecal occult blood screening are far too brief to do them justice given the current interest worldwide in these aspects of the disease. The section on screening by endoscopic means makes no mention of the potential complications of this modality, and surely deserves at least a mention. Unfortunately, there are also some inaccuracies in the book—for example, the section on screening by CT colonography.

The book is written in a very readable style but with very few illustrations and the quality of the illustrations included is adequate. I found the lack of detail and lack of inclusion of some of the most relevant literature (the last five years) irritating and frustrating. Given the size of the task, I imagine such a book was several years in gestation and this may account for some recent important publications being omitted. It is certainly not a reference book but might provide useful background reading for investigators who are new to the area.

J H Scholefield

NOTICES

Sir Francis Avery Jones British Society of Gastroenterology Research Award 2004

Applications are invited by the Education Committee of the British Society of Gastroenterology who will recommend to Council the recipient of the 2004 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 old or less on 31 December 2004 but need not be a member of the Society. The recipient will be required to deliver a 30 minute lecture at the Annual meeting of the Society in Glasgow in March 2004. Applications (TWENTY COPIES) should be made to the Honorary Secretary, British Society of Gastroenterology, 3 St Andrews Place, London NW1 4LB by 1 December 2003.

British Society of Gastroenterology Hopkins Endoscopy Prize 2004

Applications are invited by the Endoscopy Committee of the British Society of Gastroenterology who will recommend to the Council the recipient of the 2004 Award. Applications (TEN 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.

An applicant need not be a member of the Society. The recipient will be required to deliver a 20 minute lecture at the Annual meeting of the Society in Glasgow in March 2004. Applications (TEN COPIES) should be made to the Endoscopy Section Secretary, British Society of Gastroenterology, 3 St Andrews Place, London NW1 4LB by 1 December 2003.

European Helicobacter Study Group (EHSG)

This meeting, on Helicobacter infections and gastroesophageal 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@mmh.lu.se; website: www.helicobacter.org

European Course on Laparoscopic Endoscopy

This course will be held on 18–21 November 2003 in Brussels, Belgium. Further details: Secretary to Professor Cadière, Service de Chirurgie Digestive, Rue Haute 322, Brussels 1000, Belgium. Tel: +32 (0)2 648 07 60; fax: +32 (0)2 647 86 94; email: straeb.asm@proximedia.be; website: www.straeb-asm.com

Hong Kong-Shanghai International Liver Congress 2004

This conference will be held on 14–17 February 2004 in Hong Kong. The topic of the conference is “Liver Diseases in the Post-Genomic Era”. Further details: Ms Kristie Leung, Room 102–105 School of General Nursing, Queen Mary Hospital, 102 Pokfulam Road, Hong Kong. Tel: +852 2818 4300/8101 2442; fax: +852 2818 4030; email: kristieleung@hepa2004.org; website: www.hepa2004.org

The European Society of Parenteral and Enteral Nutrition (ESPEN)

ESPEN will celebrate its silver anniversary at the time of the annual congress, which is to be held on 20–23 September 2003 in Cannes, France. Further details: www.espen.org

XII Falk Liver Week

The XII Falk Liver Week, in honour of Hans Popper’s 100th birthday, will be held on 15–22 October 2003 in Freiburg, Germany. Further details - see Falk Symposia above.

3rd Congress of the European Chapter of the American College of Nutrition

This meeting will be held on 14–15 November 2003 in Göttingen, Germany. Abstract deadline: 01 October 2003. Main topics: Metabolic Syndrome, Plant-genomics, Treatment of Obesity, Hormonal Regulation of the Body Weight, Pediatric Nutrition, Malnutrition, Food-induced Diseases, Food and Allergy. Further details: G Schickedanz, Congress Secretary, Department of Gastroenterology and Endocrinology, University of Göttingen, Robert-Koch-Str. 40, 37075 Göttingen, Germany. Tel: +49 551 396326; fax: +49 551 3919125; email: nutrition2003@med.uni-goettingen.de; website: www.nutrition-europe.org

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Is hepatobiliary scintigraphy insensitive for the diagnosis of sphincter of Oddi dysfunction?

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