LETTERS

If you have a burning desire to respond to a paper published in Gut, why not make use of our “rapid response” option?

Log onto our website (www.gutjnl.com), find the paper that interests you, and send your response via email by clicking on the “eLetters” option in the box at the top right hand corner. Points which are irrelevant or obscure, or will be posted within seven days. You can retrieve it by clicking on “read eletters” on our homepage.

The editors will decide as to whether to also publish it in a future paper issue.

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 development of MALT lymphoma.

Eradication of H pylori with triple therapy (two antibiotics and one proton pump inhibitor) is curative for low grade gastric MALT lymphoma. There are reports of long term studies in the literature from the major centres around the world7 on the efficacy and safety of this modality of treatment for low grade MALT lymphoma.

District General Hospital (DGH) experience of treating MALT lymphomas is limited due to the rarity of the disorder. However, MALT lymphoma can be managed at a DGH with long term follow up. Regular endoscopic surveillance is required following eradication of H pylori.

Primary diffuse large B cell gastric lymphoma (previously known as high grade MALT lymphoma) is not considered treatable with antimicrobial agents alone. I agree with the authors that it is important to differentiate between patients who may benefit from H pylori eradication as a single modality of treatment and patients who require conventional chemotherapy in this group. The authors have cautioned that although some patients with diffuse large B cell gastric lymphoma might benefit from eradication treatment, this should not be considered standard therapy at present.

However, it was encouraging to note that high grade gastric MALT lymphoma can be treated with a single course of antibiotics and eradication of H pylori, provided the patient is willing to undergo close observation and endoscopic surveillance. This is particularly pertinent for a DGH to heed this message as in a rare situation of being faced with a high grade gastric MALT lymphoma, one would feel confident to try antibiotic eradication of H pylori alone with careful endoscopic surveillance, as often is employed in the case of low grade gastric MALT lymphoma.

R Sinharay
Royal Gwent Hospital, Cardiff Rd, Newport, Gwent NP20 2UB, UK; ranjit.sinharay@hotmail.com

References

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

I was very pleased to read the letter by Dr Madacy in response to our article “Scintigraphy versus manometry in patients with suspected sphincter of Oddi dysfunction” (Gut 2000;52:352–7). The major criticism of our study refers to the change from the original study of Sostre and colleagues1 that we made with regards to administration of cholecystokinin octapeptide (CCK-OP): I would like to refer the reader to our manuscript (Gut 2003;52:352–7) for the explanation regarding this change, as detailed on page 353, and discussed on page 356. Previous studies have shown that a bolus injection of CCK-OP produces unpredictable results on the biliary tract. Furthermore, the half life of CCK-OP would eliminate its effect within three minutes of injection hence further complicate its reproducibility. The only means of overcoming these effects is via an infusion which has been shown to be the most reproducible means of CCK-OP administration. CCK-OP is given in this setting in order to relax the sphincter of Oddi. This is to eliminate transient spasm of the sphincter of Oddi as the cause of an abnormal scintigraphic score. To use an unpredictable means of achieving this end did not make sense to us, hence the adoption of an infusion.

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 triplane manometry and may become the new standard while we await the development of non-invasive reproducible diagnostic tests of sphincter of Oddi dysfunction.

J Toouli
Department of General and Digestive Surgery, Flinders Medical Centre, Adelaide, South Australia, Australia; jim.toouli@flinders.edu.au

References

Table 1 $T_{1/2}$ parameter of common bile duct emptying, measured by scintigraphy

<table>
<thead>
<tr>
<th></th>
<th>QHBS (min)</th>
<th>QHBS + CCK (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>43</td>
<td>18</td>
</tr>
<tr>
<td>SD</td>
<td>23</td>
<td>16</td>
</tr>
</tbody>
</table>

QHBS, quantitative hepatobiliary scintigraphy; CCK, cholecystokinin.
Table 2  Sensitivity and specificity of scintigraphic results as compared with manometry

<table>
<thead>
<tr>
<th></th>
<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>
<td>77</td>
</tr>
<tr>
<td>CBD&gt;12 mm</td>
<td>42</td>
<td>86</td>
<td>89</td>
<td>35</td>
<td>54</td>
</tr>
<tr>
<td>Abnormal LFT</td>
<td>26</td>
<td>71</td>
<td>71</td>
<td>23</td>
<td>38</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 Castera 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 metabolic association 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 present or be epidemiologically linked to infection by hepatitis C virus (HCV) for reasons that have yet to be determined. High serum glucose, as well as diabetes, are associated with liver fibrosis progression and might contribute to enhanced fibrogenesis. Although for alcohol consumption, a thorough evaluation is needed before ruling out the possibility of even slight increases in daily alcohol consumption transgressing 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 necro-inflammatory 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 is HCV-related or that confounding protease-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%–10%–30%–30%–50%–70%–100%–>100%). 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’ statement that “there were less patients with progression of steatosis than patients with steatosis appearing between the two biopsies”. In any event, the biological relevance of minor increases in the amount of steatosis appears highly improbable, especially if the total amount of steatosis in patients who worsened was not associated with the amount of fibrosis, 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 minimal steatosis do not develop liver fibrosis although they obviously experienced steatosis progression. This argues against a simple and direct link between steatosis 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 fibrogenesis and through alterations in metabolic pathways to promote steatosis.

V Ratziu, M Saboury, T Paynord
Service d’Hépatogastroentérologie Groupe, Hôpitalier P inev-Sophial, 75651 Paris, Cedex 13, France
Correspondence to: Dr V Ratziu; vratziu@heas.fr

References


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 Renenah et al suggest that the risk of colorectal cancer formation does not warrant screening or surveillance. The recommendations by Jenkins and Fairclough for a surveillance programme is a sufficient screening procedure based on aggregated data from a number of studies. One of the major aims of a screening programme is to detect an initial cancerous focus; colorectal cancer is the fourth most common cause of death and an early detection could be made for the patient’s benefit. However, the risk of colorectal cancer in acromegaly is not substantially increased compared to the normal control population. In the main, the proposed guidelines are 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 visceral adenomas. We believe this to be the best quality data on which to base recommendations. We have previously reviewed the reasons why we think that control data generated from the postmortem and colonoscopy data. These data may differ because of the 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 reviewed the reasons why we think that control data generated from the postmortem and colonoscopy data. These data may differ because of the increased risk of developing colorectal cancer; the question really concerns the magnitude of the cancer risk and the relative risk of colonoscopy.

One of the major aims of a screening programme is to prevent the development of colorectal cancer by detecting colorectal adenomas. We therefore believe it would be reasonable to biopsy any presence of colorectal cancer in acromegaly. Concerning the first point on the classification of atrophic gastritis, the current literature is largely based on the histological criteria used to categorise atrophic gastritis. To amalgamate the different viewpoints and also test interobserver agreement in atrophy classification, 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, indefinite for atrophy), which is intended to replace 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 Western system seem a suitable compromise between the excessive pathological 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.

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 antrum and one from the greater curvature 4–6 cm distal to the incisura angularis); (ii) antral mucosa (one biopsy each from the greater and lesser curvatures, 3–5 cm proximal to the pyloric ring), and (iii) only one additional biopsy from the incisura angularis. Histological categories included the basic distinction between non-atrophic and atrophic gastritis. Two pathologists independently
assessed the biopsies with a 93% consistency (Fleiss’ K value = 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 would have been missed if sampling had not included the angular mucosa. The NND percentage of atrophic gastritis (46%) that would have been missed if sampling had not included the angular mucosa. The NND would have been missed if sampling had not included the angular mucosa. The NND

<table>
<thead>
<tr>
<th>By site prevalence of gastric atrophy (504 H pylori positive consecutive patients)</th>
<th>Gastric atrophy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>IM absent</td>
</tr>
<tr>
<td>Corpus only</td>
<td>0</td>
</tr>
<tr>
<td>Antrum only</td>
<td>3</td>
</tr>
<tr>
<td>Antrum and incisura angularis</td>
<td>6</td>
</tr>
<tr>
<td>Incisura angularis only</td>
<td>9</td>
</tr>
<tr>
<td>Total</td>
<td>18</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 insufflation

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 insufflation 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), haematoctrit 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

<table>
<thead>
<tr>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 Genta RM. “We used the Sydney System”… Am J Gastroenterol 1997;92:1960–1.</td>
</tr>
</tbody>
</table>

Figure 1 Endoscopic insufflation 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.

Recent advances in immunology have been phenomenal and it is not surprising that viral hepatitis and the chronic inflammatory bowel syndrome (IBS) do not usually require endoscopic procedures, especially liver immunology and the molecular signals involved in its regulation are critical if we are to gain insights into the pathogenesis of many diseases and develop novel therapies. Recent advances in immunology have been phenomenal and it is not surprising that many clinicians find it difficult to integrate and understand the importance of emerging immunology research. The editors of this book are to be commended for providing a summary of our knowledge of immunology of the liver and how this informs liver diseases, especially viral hepatitis and autoimmune diseases. They have made a creditable attempt to demystify the molecular and immune complexities and to distil the field into one concise volume.

Chronic hepatitis C infection is one of the greatest challenges facing hepatologists and gastroenterologists alike in the 21st century and it is now clear that both viral and host immune factors determine the outcome of infection. It is thus not surprising that viral hepatitis accounts for a substantial part of the book, which covers issues from viral genetics and host responses to gene therapy of viral hepatitis and transgenic mouse models of viral progression and hepatocellular carcinoma. Potential mechanisms of autoimmune liver diseases and the clinical features of the various syndromes are also extensively reviewed. This section of the book demonstrates particularly well how an immunological understanding can provide direct insights into a particular disease.

The book is aimed at both clinicians and scientists, and provides much needed background reading in the rapidly evolving field of hepatology. However, it would be hard going for anyone without a background understanding of basic immunology/molecular biology given the complexity of the science involved. One problem with such a book is assessing the target audience. The rapid evolution of the immunology field means that parts of this book will be out of date by the time it is published and therefore of less relevance to people working directly in the field. It is perhaps most useful for clinicians or scientists working predominantly in other areas who need an introduction to liver immunology. In this context it would have been helpful to include more explanatory diagrams and a rather more “user friendly” style. However, overall this is a useful book and a good introduction to liver immunology.

Molecular Biology and Immunology in Hepatology. Advances in the Treatment of Intractable Liver Diseases

Edited by T Tsuji, T Higashi, M Zeniya, et al.
Amsterdam: Elsevier Science BV, 2002, b/w, pp 342. ISBN 0 444 50655 3

Dysregulated immune responses underlie the pathogenesis of many liver disorders including not only autoimmune diseases but also viral hepatitis and the chronic inflammatory responses stimulated by alcohol. Thus understanding liver immunology and the molecular signals involved in its regulation are critical if we are to gain insights into the pathogenesis of these diseases and develop novel therapies. Recent advances in immunology have been phenomenal and it is not surprising that many clinicians find it difficult to integrate and understand the importance of emerging immunology research. The editors of this book are to be commended for providing a summary of our knowledge of immunology of the liver and how this informs liver diseases, especially viral hepatitis and autoimmune diseases. They have made a creditable attempt to demystify the molecular and immune complexities and to distil the field into one concise volume.

Chronic hepatitis C infection is one of the greatest challenges facing hepatologists and gastroenterologists alike in the 21st century and it is now clear that both viral and host immune factors determine the outcome of infection. It is thus not surprising that viral hepatitis accounts for a substantial part of the book, which covers issues from viral genetics and host responses to gene therapy of viral hepatitis and transgenic mouse models of...
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 20 minute lecture at the Annual meeting of the Society in Glasgow in March 2005. 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 gastroduodenal 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@mmb.lu.se; website: www.helicobacter.org

Falk Symposium 135—Immunological Diseases of Liver and Gut

This symposium will be held on 12–13 September 2003 in Prague, Czech Republic. Further details: Falk Foundation e.V., Congress Division, PO Box 6529, Leinwenberstr. 5, 79041 Freiburg/Br, Germany. Tel: +49 761 15 140; fax: +49 761 15 14 359; email: symposia@falkfoundation.de; website: www.falkfoundation.de

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 Peeper'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

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.asmb@proximedia.be; website: www.straeb-asmb.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