Severe polyneuropathy complicating active Crohn’s disease: rapid response to Infliximab

Treatment with a chimeric antiumour necro- sis factor (TNF) antibody (Infliximab) has been shown to be highly efficient for patients with steroid refractory Crohn’s disease (CD). However, the mechanism of action remains largely unknown. Recently, a favourable response to Infliximab treatment was demonstrated in some diseases complicating active CD such as acute idiopathic pancreatitis. We report a case of a middle aged female with CD that developed an aggressive form of polyneuropathy resistant to corticosteroids. A 55 year old White female, weighting 68 kg, presented with exacerbation of CD (CDAI activity index (CDAI) >450) associated with potentially life threatening side effects. As a result, patients must be treated to be safe for both mother and foetus, we would like to highlight concerns that it is considered as case finding and clearly it is the patient who has initiated the consultation and in some sense is consenting for investigation. Conversely, individuals (who are not patients) found to have CD through screening programs, may have considered themselves as “well” and it is the physician or healthcare system that is identifying them as potentially ill.

We recently performed a primary care based cross sectional study using immunoglobulins, IgA/IgG antigliadin antibodies and endomy- sel antibodies to initially recognise CD. 1200 samples. The prevalence of CD in this primary care population sample is 1% (95% CI 0.4–1.3%). In this screening study, 9/12 diagnosed cases of CD ultimately had symptoms which could be attributed to CD (for example, anaemia or subtle gastro- intestinal symptoms). We, and others have demonstrated a delay in the diagnosis of CD—surely the important change in our clinical practice (both in primary and secondary care) is to have a low threshold for case finding. If you look for CD you will find it.

Coeliac disease: is case finding the correct ethical and logistical approach?

I read with interest the debate pertaining to screening for coeliac disease (CD). Although one can argue that CD fulfils the tenets of any screening programme, however, we do not know the natural history of screen detected patients with CD. Logistically when would we decide to screen—at what age and how often there after? Serological markers may be highly sen- sitive and specific but the value of these tests decrease when they are used in the general population. Although the investigational process for population screening and case finding may be the same, there is an important ethical differ- ence between them. If a patient seeks medical help then the physician is attempting to diag- nose the underlying condition (for example: patients with CD who present with symptoms of irritable bowel syndrome). This would be classified as case finding and clearly it is the patient who has initiated the consultation and in some sense is consenting for investigation.

References

LETTERS

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

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We recently performed a primary care based cross sectional study using immunoglobulins, IgA/IgG antigliadin antibodies and endomysial antibodies to initially recognise CD. 1200 volunteers were recruited from January 1999 to June 2001 from 5 general practices in South Yorkshire, UK. Any participant with a positive IgA antigliadin antibody, positive endomysial antibody or only IgG antigliadin antibody in the presence of IgA deficiency was offered a small bowel biopsy to confirm the diagnosis of CD. Twelve new cases of CD were diagnosed from 1200 samples. The prevalence of CD in this primary care population sample is 1% (95% CI 0.4–1.3%). In this screening study, 9/12 diagnosed cases of CD ultimately had symptoms which could be attributed to CD (for example, anaemia or subtle gastrointestinal symptoms). We, and others have demonstrated a delay in the diagnosis of CD—surely the important change in our clinical practice (both in primary and secondary care) is to have a low threshold for case finding. If you look for CD you will find it.

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References

Caution with the use of cyclosporin in pregnancy

We read the article by Alstead and Nelson-Piercy with great interest.

We report a case (submitted for publication) of a woman with fulminant ulcerative colitis in the 29th week of pregnancy. Her dis- ease was refractory to steroids, but she refused to have cyclosporin whilst pregnant. She therefore underwent an emergency Caesarean section and was given intravenous cyclosporin post-operatively. After 48 hours of treatment she developed severe hypertension with hypertensive encephalopathy and sei- zures. Although cyclosporin has been consid- ered to be safe for both mother and foetus, we would like to highlight concerns that it is associated with potentially life threatening side effects. As a result, patients must be counselled thoroughly about the potential morbidity associated with this treatment and monitored closely. We agree with Dr Alstead that cyclosporin should be used with extreme caution in pregnancy and the postna- tural period.

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Author’s reply

If I interpret Dr Sanders’ position correctly, he favours population screening, provided that a clear finding approach is applied. His letter gives me the opportunity to expand further on my opinion on the appropriateness for coeliac disease (CD) population screening. As I mentioned in my final remarks and found in the summary of my debate, while CD fulfils the criteria for mass screening, currently we lack the evidence based elements to justify a universal screening in European and North American populations. Therefore, my current position does not diverge substantially from that of Dr Sanders. I firmly believe that an “open-minded approach”, in which increased awareness and low threshold are applied to populations at risk, is ethical, logical, and socially acceptable. This attitude was extremely effective in the USA, where the healthcare community had the perception that CD was extremely rare. We have recently subverted this wisdom by showing that the overall prevalence of CD in the USA is 1:133 in not-at-risk groups and between 2%–9% in at-risk groups, so proving that this disease was historically overlooked in the USA. If some reports in the literature indicating that prolonged gluten exposure can lead to increased morbidity and mortality are confirmed, we should be ready to change our attitude and embrace new guidelines for CD mass screening.

Epidemiological data published worldwide suggest that CD is one of the most frequent genetically based chronic diseases of mankind. Therefore, there has been no better time to establish the appropriateness for CD mass screening by performing well designed studies, rather than look in the opposite direction and ignore the problem. If we are not humble enough to embrace this approach to resolve this problem, we will not only be ethically and logistically incorrect, but also morally responsible for a poor outcome of our medical mission.

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References

Multiple focal nodular hyperplasia of the liver in a patient with prostatic cancer

We read with interest the study of Luciani and colleagues (Gut 2002;50:877–80) comparing focal nodular hyperplasia (FNH) of the liver in men and women. The major findings of this very large study conducted by an expert team in this field were that mean age at diagnosis was higher in men (p<0.01), mean FNH size smaller (p<0.001), and occurrence more often performed (p<0.001) in men (n=18) than in women (n=216). Interestingly, perhaps because of the relatively small number of men (although in very large terms important, the rare occurrence of FNH in men), no cases of multiple FNH were noted in the male population. We report here on multiple FNH in a 74 year old patient with a biopsy proven prostatic cancer. This patient had not received any treatment. He was referred to our liver unit in March 2001 for evaluation of multiple liver masses discovered on abdominal ultrasonography during the staging of his cancer. Bone scintigraphy disclosed no metastases. Liver biochemistry was normal except for a mild increase in gamma glutamyl transferase activity. Prothrombin index was 100%. Serological search for hepatitis B or hepatitis C virus infection was negative. Genetic (haemochromatosis, alpha 1 antitrypsin deficiency) and autoimmune liver diseases were carefully excluded, and alcohol consumption was below 10 g/day. Upper gastrointestinal endoscopy and colonoscopy were normal. Tumour markers of malignant primary or secondary liver lesions were within the normal range. Liver Doppler ultrasonography disclosed multiple heterogeneously hypoechoic lesions with a hypoechogenic pattern and without an arterial signal. Abdominal tomodensitometry before and after contrast enhancement revealed multiple lesions with rapid contrast enhancement during the arterial phase. The largest lesion was located between the left liver and segment IV and measured 75 mm.

Because there was no magnetic resonance imaging (MRI) in our centre, ultrasound guided liver biopsy in both tumoral and non-tumoral areas was performed. The diagnosis of typical FNH was made in several of the lesions whereas non-tumoral liver was normal. The patient received hormonal treatment from April 2001. In October 2001, MRI confirmed a diagnosis of FNH with a central stellate area in the largest lesion. In December 2002, he was in good health with unchanged ultrasonography.

This case report is unique in that there were multiple lesions in a patient who had not received any previous treatments or portal caval shunt. Although from a literature search it is difficult to determine the exact number of men with multiple FNH, the numbers are probably very low. In this report, the discrepancy between normal bone scintigraphy and multiple liver lesions and the diagnosis of liver metastasis probable. Nevertheless, histological examination of several hepatic lesions, retrospective MRI, and outcome made the diagnosis of multiple FNH certain. This report, in common with the large series of Luciani et al, indicates that FNH diagnosis may be very difficult in men.

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Guidelines for colonoscopic screening in acromegaly are inconsistent with those for other high risk groups

We welcome the joint work of the British Society of Gastroenterology and the Association of Coloproctology of Great Britain and Ireland in commissioning guidelines for colorectal cancer screening in high risk groups (Gut 2003;50:V13–14). In the absence of direct evidence from randomised trials for most of the groups, the various authors have balanced a wealth of recent genetic and epidemiological evidence depicting an individual’s levels of elevated risk against the risks associated with screening. The end result is the recognition that within the label “high risk” there is a spectrum of risks such that colonoscopic screening and surveillance must be tailored accordingly. This avoids the ineffective, costly, and potentially harmful “blanket-type” approach, which formerly prevailed.

However, within the guideline series, there is one exception—screening and surveillance in patients with acromegaly. We and other researchers1,2 have repeatedly stated that the studies undertaken by the authors of these guidelines have overestimated the risk of colorectal cancer in this patient group. They report a 13–14-fold increase in risk based on colorectal cancer detection rates among acromegals, which are underpinned by various studies compared with cancer rates from published series of colonoscopic screening in non-acromegalic subjects (see table 3 in Jenkins and Fairclough’s paper). All of these studies were conducted in mixed race US populations and lack data to permit age-sex comparisons. This simply does not rank as a well designed controlled study and the recommendations should not receive grade B status.

In the same manner as relative risks for those with relatives with colorectal cancer are estimated from population based data (summarised in Dunlop),3 we have argued for a similar approach for patients with acromegaly. Based on three population based studies, we calculated (by fixed effects meta-analysis) a relative risk of 2.0 (95% confidence intervals 1.4–2.9) for colon and rectal cancer combined in acromegalic patients compared with cancer rates from published series of colonoscopic screening in non-acromegalic subjects (see table 3 in Jenkins and Fairclough’s paper). All of these studies were conducted in mixed race US populations and lack data to permit age-sex comparisons. This simply does not rank as a well designed controlled study and the recommendations should not receive grade B status.

In terms of absolute risk, with an approximate 2% cumulative risk of colorectal cancer by age 70 years in the general UK population, the estimated risk in acromegalic would be 4%. Jenkins and Fairclough estimate the incidence in the general US population to be 4.5% (10 of 222)–not a quarter if the estimate of a 13-fold increase was applied. Acromegalic patients thus have a modest absolute cancer risk compared with the high risk. In first degrees relatives with a strong family history (a high risk group), Dunlop recommends early colonoscopic screening and then wait until 55 years for repeat colonoscopy if initial screening is clear (the majority). How can Jenkins and Fairclough justify early screening and colonoscopy five yearly thereafter in all of their acromegalics?

Atkin and Saunders have demonstrated that colonoscopic surveillance following initial screening in the non-acromegalic population is determined primarily by clinicopathological findings. These basic guidelines must also be applied to the acromegalic patient population. Jenkins and Fairclough have stated that elevated serum IGF-1 levels may predict for recurrent adenomas in acromegalic patients and “should be offered screening at three year intervals”. This is based on data from only eight acromegalic patients with recurrent adenomas and should not replace other well recognised predictors of recurrence.

Looked at in the context of (other high risk) groups, the guidelines for colorectal cancer screening in patients with acromegaly are inconsistent. The aggressive approach to...
colonoscopic screening recommended by Jenkins and Fairclough should be seriously questioned.

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1. Renehan AG, Shalet SM. Acromegaly and colorectal cancer: risk assessment should be based on population-based studies. J Clin Endocrinol Metab 2002;87:1909

Authors’ reply

We thank Dr Renehan and colleagues for their comments on our data, which they have also made previously. We do not claim that our data are perfect in all respects but it seems to us, on the basis of the data we have collected in our own series and that of other groups, that patients with acromegaly should be regarded as having a significantly increased risk of colorectal neoplasia. The two contrary studies referred to by Renehan et al are his own and that of one other author who relied on retrospective data acquired more than 50 years ago. These data and those from the population based studies preferred by Renehan suffer from flaws of their own. The morbidity associated with acromegaly has changed in the last 25 years, probably related to the increased survival associated with aggressive and effective treatment of the cardiovascular and metabolic complications of the disease. Our data and those of others show that the prevalence of colon neoplasia in acromegaly is age dependent. Thus it is only now that patients are surviving long enough to develop this complication, and valid comparative data must therefore be acquired contemporaneously, to take account of the changing pattern of morbidity associated with increased longevity.

We are aware of at least 12 other prospective studies evaluating colonoscopic screening in acromegaly that we did not include in our original report from St Bartholomew’s Hospital. These include one by Renehan et al in which they reported three asymptomatic patients in whom a cancer was detected. Among such studies the optimum comparison must be simultaneous screening of asymptomatic non-acromegaly patients with combined comparison of the data from all series using these control groups, none of which involved mixed race US populations, gives a relative risk of colon cancer in the range 1.4. We think it is prudent to accept the evidence of an increased risk of colon cancer, derived from these clinical observations rather than from theoretical calculations, and to screen acromegalic patients systematically until the current hypothesis is confirmed or refuted. The rarity of acromegaly means that the increase in workload for the majority of individuals with endoscopy units is likely to be minimal.

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New imaging techniques: promise or passe

I read with great interest the article by Egger and colleagues (Gut 2003;52:18–23) evaluating laser induced fluorescence endoscopy (LIFE) and methylene blue (MB) directed biopsies for detection of dysplasia in Barrett’s oesophagus.

As the authors point out, there have been no fully published studies to date on this much talked about procedure. The authors found that LIFE and MB had limited accuracy, as did standard random biopsy. Although LIFE and MB detected a total of five cases of high grade dysplasia and 11 cases of low grade not seen on four quadrant random biopsy (4QB), they concluded that these methods are “not capable of increasing the diagnostic accuracy or replacing standard four quadrant biopsies”.

How could these data lead to this conclusion? The authors discount all but one high grade and seven low grade lesions detected by LIFE or MB because they were “within the 4QB protocol”. It was assumed by the authors that these sites would have been biopsied by random technique and had not already been sampled with AF or MB. Given that the biopsies were standard 7 mm forceps, that dysplasia can be very focally distributed, and the area included within the 4QB covers two linear centimetres, it is difficult to assume that this exact site would have been biopsied with a random technique. This assumption, if incorrect, would result in underestimation of the value of LIFE or MB.

In addition, the authors further discounted the one remaining high grade dysplasia site and four more low grade sites because they occurred in patients with known cancer who presumably would be treated for the cancer regardless. There is little doubt that detection of low or even high grade dysplasia has little relevance if a cancer is already known. The main group of patients where LIFE, MB, and other advanced techniques should be applied are those with endoscopically evident tumours and cancer. Discounting LIFE and MB for this reason may further underestimate its value.

If we do not discount these cases then LIFE and MB appear to complement 4QB for the detection of dysplasia, with each technique markedly detecting dysplastic sites that the other missed.

I agree that LIFE and MB remain controversial and applaud the authors for publication of these findings. Given the limitations of the study however, it may be premature to proclaim these techniques incapable. More well conducted studies are clearly needed. The fast field of imaging technologies is also evolving rapidly and new and better techniques are constantly on the near horizon.

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Transient ischaemic colitis following an aeroplane flight

We read with interest the report of Butcher and colleagues (Gut 2002;51:746–7) of two cases of transient ischaemic colitis following an aeroplane flight. This report represents more evidence supporting the suggestion of a possible important role of acquired and hereditary thrombotic risk factors in the pathogenesis of ischaemic colitis.1 However, the largest study to date concerning these factors in patients with ischaemic colitis was not mentioned in the study.2 Moreover, the reported patients may also have other acquired or inherited thrombophilic disorders which were not evaluated. Lipoprotein (a), anticardiolipin antibodies, the C677T methylentetrahydrofolate reductase polymorphism, and the G20210A prothrombin gene mutation were not studied in both patients whereas in the second case even the very important factor V Leiden mutation as well as lupus anticoagulant and homocysteine levels were not evaluated. Although the aeroplane flight could be the most important risk factor in these cases, the rather incomplete thrombophilic screening does not permit us to conclude that it was “the only potential risk factor”.

It is known that deep vein thrombosis (mainly symptomless) may occur in up to 10% of long haul airline travellers.1 In contrast, the hypercoagulable state is a rare cause of ischaemic colitis, leading to the development of thrombotic occlusion of the small vessels supplying the colon. In a recent study on IMTT thrombophilic screening in patients with an established diagnosis of ischaemic colitis, we found the prevalence of acquired and hereditary thrombotic risk factors significantly higher compared with the prevalence of these factors in matched inflammatory and healthy controls.3 A thrombophilic tendency was demonstrated in the majority of patients and the most significant associations were with antiphospholipid antibodies and with the factor V Leiden mutation. Moreover, we recently found a high frequency of protein Z deficiency in patients with ischaemic colitis (unpublished data). Based on the recent data of the association of protein Z deficiency mainly with arterial thrombosis,4 protein Z deficiency may be involved in the development of the disease in a subgroup of patients by causing thrombosis in the small mesenteric arteries. The role of protein Z deficiency is considered the result of localised non-occlusive ischaemia of the small arteries. In contrast, the presence of hypercoagulable states suggests a possible role of
venous obstruction. It is possible that future identification of subgroups in ischaemic coli-
tis patients with sophisticated imaging tech-
niques could diminish these cases with arterial or venous ischaemia.

In conclusion, we suggest that the mech-
amism of ischaemic colitis is multifactorial. Acquired and genetic factors may interact leading to disease manifestation. Arteriomegaly and embolic conditions, oral contraceptives, and other medications, as well long haul flights probably play a role in genetically pre-
disposed individuals in the disease pathogen-
esis.

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References

BOOK REVIEWS

Surgery of the Liver, Bile Ducts and Pancreas in Children


This excellent volume builds on the success of the first edition published a decade ago by the senior editor (ERH). Inevitably it is bigger, and, yes, better too. The contributors, drawn from across the world, have been chosen for their expertise and this is apparent in their respective chapters. A major beneficial inno-
vation is inclusion of an editor and other con-
tributors from the USA; sadly, for an inher-
ently English book, English spelling has been sacrificed in the process.

All chapters have been rewritten in re-
response to the significant developments in hepatobiliary surgery over the past decade—
for example, the cytoarchitecture of the anatomy of the liver and in liver transplanta-
tion (now up from 14 to 98 pages). The chap-
ter on “portosystemic shunt surgery” has
given way to “surgery for portal hyper-
tension”, reflecting the development of alter-
native strategies for this condition. As before, the detailed and informative chapters on anat-
omy, physiology, and preoperative manage-
ment complement the core “surgical” chap-
ters, and there are new chapters on embryology, anaesthesia and perioperative care, nutrition, and the psychosocial aspects of childhood liver disease, which will be of interest to gastroenterologists and surgeons alike. Another innovation is the inclusion of a section on “surgical” paediatric pancreatic disorders, including chapters on embryology and anatomy; this is a logical development that enhances the value of this volume.

The scientific standard is high throughout the book. Each of the chapters has been well researched by its author and (with only one exception), chapters are well referenced. Of course one can find omissions but these are minor and very infrequent. The text is easy to read and the editors have ensured that the content is clinically relevant. Each chapter concludes with a useful table of annotated key references, and in some chapters a recom-
mended management strategy is summarised in a box. The book is liberally illustrated with line drawings and both black and white and colour figures, which generally are informa-
tive and clearly reproduced. The index is good; I was able, without difficulty, to locate a vari-
ety of topics. Overall, the quality of the production is excellent, with very few typo-
graphical errors. Although this is a compre-
hensive book, it is not a large tome, but is comfortable and easy to read.

This book will serve as an invaluable resource for gastroenterologists, paediatricians, surgeons, nurses, and others involved in the surgical care of children with liver, biliary, or pancreatic disorders, whether at a specialist children’s centre or a district hospital, and will appeal to trainees, whose exposure these days to complex hepatobiliary surgery is limited. In addition, gastroenterologists and surgeons treating the increasing number of adults with disorders of childhood will find this book a most helpful companion.

The Handbook of Clinical Trials and Other Research

A Earl-Slater. Abingdon, UK: Radcliffe Medi-

Clinicians are notorious for embarking upon research without a full understanding of methodology. Perhaps in the past clinical journals were guilty of publishing papers without being sufficiently critical. No doubt this was a byproduct of well meaning referring by clinicians who were themselves hamstrung methodologically, and lacking insight.

In the new world of publications, the research design has to be explicit, well laid out, and sufficiently robust to support the research reported. Most doctors have had little or no training in research methods, despite having completed an MD. This might be one reason why it is becoming increasingly difficult for even research experienced clini-
cians to initiate new projects. Indeed, there is a question mark as to whether research can now be done by service based clinicians or whether, because of the newer strictures and disciplines, this should be left to the profes-
sional researcher. Perhaps the answer is that clinicians ought to have access to experts who can not only guide them but see them through the entire project. Some have questioned whether clinical research will proceed. Perhaps the answer, even if only in hope, is yes, but this will require a greater familiarity with research methodology, the patience to plan thought-
fully and with experienced help.

This handbook provides definitions and contemporary examples. It provides recent references from major journals and is well illustrated. It contains material beyond expla-
nations of research terminology and method-
ology including the new requirements for the Research Ethics Committees, and the EU Clinical Trials directive. Perhaps the most obvious is that research is fast moving; in the UK there is a single electronic ethics application form now with new rules regarding consent for multicentre

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Hepatology Principles and Practice

E Kuntz, H-D Kuntz. Berlin: Springer Verlag, colour, pp 825. ISBN 3540 42161 0

“You have a very large parcel”, Zeinab, my secretary said breathlessly as she struggled up from the post room with a copy of this enormous book. It seemed all the heavier as I lugged it around the London bus network from the wilds of East Acton on my way home and then back to work several times. Contained in a reinforced Harrod’s bag, which was the only thing I could find that was strong enough to hold it while I elbowed my way through the myriad of commuters that were forced to travel by bus in lieu of the non-existent Central line, I felt my back pain had returned with a vengeance and did not know whether to take up weight training (where the book would come in handy) or admit defeat and sue the authors for damage. However, sanity soon prevailed and I soon became engrossed in this weighty tome.

At first glance, one could say that another comprehensive book on hepatology is really not needed, given all the other titles on the market but, it turns out that this 2002 English edition of the original German “Praktische Hepatologie”, published in 1998, has been updated and was a labour of love by Erwin Kuntz whose son, Hans-Dieter, died before the book could be finished. My schoolboy German is not up to the original edition but this has become a bible in Germany and the current version is welcome, despite the fact that a lot of the English is somewhat awkward with curious turns of phrase. Examples of this include “MRT”, rather than MRI, which is curious turns of phrase. Examples of this include “MRT”, rather than MRI, which is commonly seen, and on MRI (“MRT”) is almost non-existent by comparison. It is a shame that the chapter on cognitive testing and the investigations of the neurological sequela of liver disease does not contain detail on newer psychometric tests and technology such as MR spectroscopy, given the wealth of expertise on hepatic encephalopathy that currently exists in Germany. Chapters on the complications of chronic liver disease are well set out and those on liver abscesses, bacterial, parasitic, and fungal (“mycotic”) liver disease are useful.

The question of who may actually buy this book looms large. It is too big and too costly to use and is a useful repository of terms and other sources of information. I am glad I have my copy but can you really teach old dogs new tricks?

P Hungin

NOTICES

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 ACGBI 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 9233; email: acgbi@acgbi.org.uk; website: www.acgbi.org.uk

European Helicobacter Study Group (EHSG)

This meeting, on Helicobacter infections and gastroduodenal pathologies, 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, Sölvegatan 23, SE-223 62 Lund, Sweden. Tel: +46 46 173 241; fax: +46 46 152 564; email: Torkel.Wadstrom@mbi.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 6329, Leinonenstr. 5, 79041 Freiburg/B, Germany. Tel: +49 761 15 14 190; 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 Popper’s 100th birthday, will be held on 15–22 October 2003 in Freiburg, Germany. Further details - see Falk Symposia above.

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, Bruxelles 1000, Belgium. Tel: +32 (0)2 648 07 60; fax: +32 (0)1 647 86 94; email: straeb.asmb@proximedia.be; website: www.straeb-asmb.com
Multiple focal nodular hyperplasia of the liver in a patient with prostatic cancer

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