PostScript

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 development of MALT lymphoma.

Eradication of *H pylori* with triple therapy (two antibiotics and double dose 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 world[1–3] on the efficacy and safety of this modality of treatment for low grade MALT lymphoma.

District General Hospital (DGH) experience of treating MALT lymphoma is limited due to the rarity of the disorder. However, MALT lymphoma can be managed at a DGH with long term follow up.[4] 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 modality of antibiotic 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.[4]

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

I was very pleased to read the letter by Dr Madacsy 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[1] 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 triple lumen manometry 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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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 quantitative hepatobiliary scintigraphy (QHBS) in patients with a suspected sphincter of Oddi dysfunction (SOD). As our paper documenting contrary results was referred to,[1] 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 results[1 2] 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.

In fact, the Australian group changed the CCK augmentation method during QHBS, as originally suggested by Sostre and colleagues[3]: 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 initiation 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

	QHBS (min)	QHBS+CCK (min)
Mean	43	18
SD	23	16

QHBS, quantitative hepatobiliary scintigraphy; CCK, cholecystokinin.

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

	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Accuracy (%)
QHBS positive	79	71	88	55	77
CBD>12 mm	42	86	89	35	54
Abnormal LFT	26	71	71	23	38

QHBS, quantitative hepatobiliary scintigraphy; CBD, common bile duct; LFT, liver function test; PPV, positive predictive value; NPV, negative predictive value.

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.[4] Once the tracer is in the duodenum, no further information is available on SO function and resistance. Manometry clearly reveals that CCK-OP has a relaxing effect on the SO.[5] 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.[6]

Therefore, in most SOD patients with an elevated SO basal pressure, CCK-OP induces a significant pressure drop, as demonstrated by Hogan and Geenen.[7] CCK-OP administration during QHBS must therefore be regarded as a relaxation test of the SO.[7]

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.[1 8] 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.[8]

In common with the study of Craig *et al*, we recently compared our scintigraphic (without CCK-OP) and manometric results.[9] Comparison 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.

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Worsening of steatosis and fibrosis progression

We read with great interest the article by Castera and colleagues (*Gut* 2003;**52**:288–92) and acknowledge the finding that worsening of steatosis in chronic hepatitis C is associated with fibrosis progression. However, in our view there are no data supporting a casual role for this statistical 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[1] or be epidemiologically linked to infection by hepatitis C virus (HCV) for reasons that have yet to be determined.[2] High serum glucose,[3] as well as diabetes,[4] are associated with liver fibrosis progression[5] and might contribute to enhanced fibrogenesis.[6] As for alcohol consumption, a thorough evaluation is needed before ruling out the possibility of even slight increases in daily alcohol consumption translating, 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 is HCV-related or that confounding prosteatogenic 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' statement that "there were less patients with progression of steatosis than patients with steatosis appearance 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 on the first biopsy 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 progression rather than the amount of steatosis is 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[7] 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[8] to enhance fibrogenesis and through alterations in metabolic pathways to promote steatosis.

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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 Renehan *et al* suggest that the risk of colorectal cancer formation does not warrant screening or surveillance.[1 2] The recommendations by Jenkins and Fairclough for a national screening programme, endorsed by the BSG and ACPBG, are based largely on a series of 222 patients enrolled in a colonoscopy programme in one centre. The principal finding of this study was a 13–14-fold increase in the risk of colorectal cancer in acromegalics relative to the general population. This is at odds with larger studies (n=1362,[3] n=1041,[4] n=1634[5]) 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 (citing the youngest case in their 222 case series 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 adenoma at initial colonoscopy or increased IGF-1 levels). Renehan *et al* however conclude that there is no increased incidence of colonic adenomas compared with a normal control population (generated from postmortem and colonoscopy data).

These data may differ because of the controls used and this probably also explains their variance with the larger studies.[6] 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.[7]

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 requested to submit guidelines.[7] 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 colonoscopy 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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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 Renehan *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, both for the sake 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 complete, 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 same 500 examinations, we have detected 10 asymptomatic cancers, as well as numerous adenomas. Thus based both on the literature and on our own experience, the risk of undetected colonic 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) how consistent 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 evaluate 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.[1 2] To amalgamate the different viewpoints and also test interobserver agreement in atrophy classification/scoring, an international group of pathologists recently published an extensive description of the different phenotypes of gastric atrophy.[3] 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 suspends any 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 updated Sydney system[4] 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.[5]

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 angulus and one from the greater curvature 4–8 cm distal from cardia); (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.[3] Two pathologists independently

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

By site prevalence of gastric atrophy (504 *H pylori* positive consecutive patients)	Gastric atrophy		
	IM absent	IM present	Total
Corpus only	0	1	1
Antrum only	3	10	13
Antrum and incisura angularis	6	14	20
Incisura angularis only	9	20	29
Total	18	45	63

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.

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 (number needed to diagnose[6]) 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.

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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 mucosal 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), 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.[1] 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.[2] Continuous high doses of steroids make human tissue fragile, including the colon mucosa. Taken together, these results

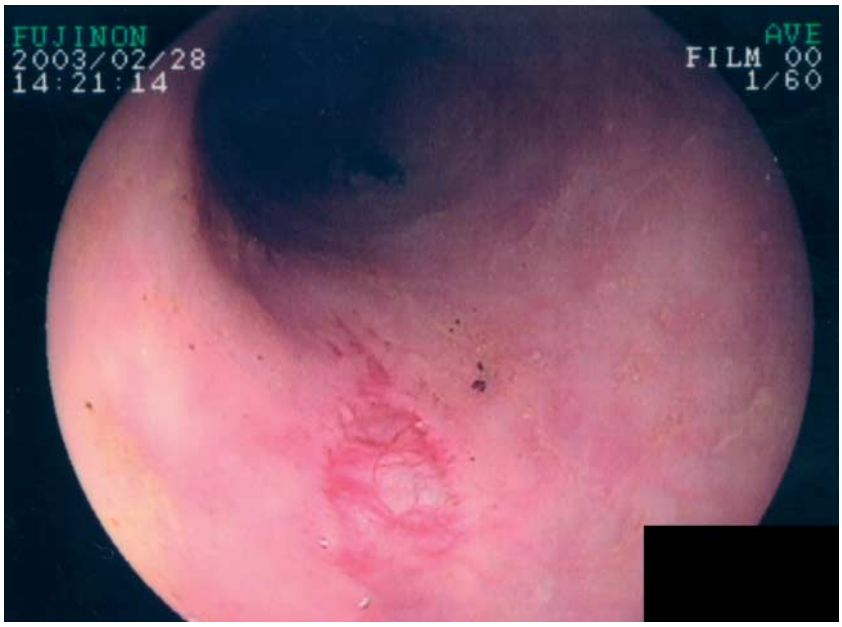

Figure 1 Endoscopic insufflation of a diverted colon resulted in a mucosal tear.

suggest that the mucosal tear in our case may have been attributable to diversion colitis with fragile mucosa.

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

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BOOK REVIEWS

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 0-444-50653-5

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 involved 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 carcinogenesis. Potential mechanisms of autoimmune liver diseases and the clinical features of the various overlap syndromes are also extensively reviewed. This section of the book demonstrates particularly well how an immunological understanding can provide direct insights into clinical disease.

The book includes chapters dedicated to the clinical management of liver disease, including viral hepatitis, hepatocellular carcinoma, acute liver failure, and living related liver transplantation. There is no doubt that the authors' extensive experience in the field of living related liver transplantation will appeal to physicians and surgeons alike but the insights brought to these areas by immunology are less clearly stated.

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.

J A Eksteen, D H Adams

Irritable Bowel Syndrome: Diagnosis and Treatment

Edited by M Camilleri, R C Spiller. Philadelphia: WB Saunders, £49.99, pp 193. ISBN 0702026557.

Gastroenterologists derive job satisfaction from performing endoscopic procedures, establishing diagnoses, and explaining and treating symptoms. Patients with irritable bowel syndrome (IBS) do not usually require endoscopic procedures, diagnosis is often uncertain, symptoms cannot easily be explained, and there is no effective treatment the health service will pay for. Its not surprising therefore that few gastroenterologists relish the prospect of seeing a patient with IBS. Can this textbook of IBS help their plight? The answer is undoubtedly yes; however, success with IBS patients depends critically on good communication, a skill that cannot be gained from reading a textbook.

The authors (both basic scientists at heart) are to be congratulated on assembling a holistic collection of contributors and paying lip service to the different profiles of IBS in varied clinical settings. Equal weight is given to psychological aspects, serotonergic receptors, physiology, causative factors, the concept of consultation behaviour, and many other factors. Thus the book is much more than a textbook on "diagnosis and treatment". However, I support the concept that this wider view of IBS is mandatory for effective diagnosis and treatment.

The book is an excellent resource for all health professionals dealing with IBS and it will be a vital starting point for those wishing to research IBS. The chapters are extensively referenced and many questions in areas of uncertainty are left refreshingly open. My favourite chapters are Grant Thompson's "A world-view of IBS" and Bennett and Kellow's "Relations between chronic stress and bowel symptoms". They place IBS in context and provide a foundation for many of the other chapters. Excellent stuff.

Inevitably, compromises are made in a multiauthor book, particularly when it covers such a difficult topic spanning so many disciplines. The editorial hand has been too light: it is a book of individual contributions rather than a cohesive text. For example, the chapter on serotonergic mechanisms by Michael Gershon (while well written and fascinating) loses the plot with a level of detail that seems misplaced among the other chapters. In other places there is unnecessary repetition. On a more practical level, there are no links to internet based resources, no contributions from patients, and little practical advice on how to structure and conduct a consultation or negotiate with a patient. There is no declaration of commercial support in the final chapter. Does this mean there was none?

Perhaps the most disappointing aspect of this book is the failure to put IBS in the context of other unexplained gastrointestinal symptoms. Grant Thompson provides a list of other functional gut disorders. This very medical approach to unexplained symptoms is driven by the need for clean entry criteria for drug trials and physiological research studies. It presumes that it must be possible 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. A chapter placing IBS in the context of other unexplained bodily sensations (UBS) will be most welcome in the next edition.

R Valori

Colorectal Cancer

M P de Leon. Berlin: Springer, 2002, £70.00, pp 303. ISBN 3-540-43047-4

My initial reaction to a single author text on colorectal cancer was that the author was either a brave or a foolish man to attempt such an onerous task single handed. New information about aetiology, screening, pathogenesis, and all aspects of treatment have changed considerably in the last 10 years and the literature abounds with new information, only some of which is important, but all of it needs sifting to distil a worthwhile and up to date text.

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 constructive and critical attitude which may help a new generation of investigators, and to some extent the volume has achieved this aim. However, he also recognises his own limitations in taking on this daunting task

The author is not explicit as to who the book is aimed at but refers to a "new generation of investigators"—if this means that the

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 colography.

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 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, Leinenweberstr. 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 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

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