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 “letters” option in the box at the top right hand corner. Points of view or information included in your letter will be acknowledged. If we publish it, you will be invited to make use of our

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 5 and 18 leads to the development of MALT lymphoma.

Eradication of H pylori with triple therapy (two antibiotics and a 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 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 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 lymphomas.

R Sinharay
Royal Gwent Hospital, Cardiff Rd, Newport, Gwent NP20 2UB, UK; ranjitsinharay@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 Madaczy in response to our article “Scintigraphy versus manometry in patients with suspected sphincter of Oddi dysfunction” (Gut 2003;52:352–7). This letter raises valid concerns regarding our methodology, and I would like to present the following points.

1. 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 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.

2. The Australian group changed the CCK augmentation method during QHBS, as originally suggested by Sostre and colleagues1; 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: T1/2 parameter of common bile duct emptying, measured by scintigraphy

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

QHBS, quantitative hepatobiliary scintigraphy; CCK, cholecystokinin.

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

References
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 cholecystectomy subjects, most of the radiotracer has been emptied from the biliary tree into the duodenum after 30 minutes. Once the tracer is in the duodenum, no further information is available on SO function and resistance. Manometry clearly reveals that CCK-OP has a relaxing effect on the SO. 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.

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

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. 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.

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

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+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 their concern that worsening of steatosis in chronic hepatitis C is associated with fibrosis progression. However, in our view there are no data supporting a causal 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 these complications associated with insulin resistance. The latter can develop within the course of liver injury 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. 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 C. 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 additional 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 the results have 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 regression 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 that may be measured 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 liver fat is 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’Hepato-gastroenterologie Groupe, Hopitalier Pine-Salpetriere, 75651 Paris, Cedex 13, France
Correspondence to: Dr V Ratziu; vratziu@heather.com

References

6 Fehrenbach H, Weiskirchen R, Kasper M, et al. Up-regulated expression of the receptor for advanced glycation end products in cultured rat hepatic stellate cells during...
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(supp VI):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 Renenh et al. suggest that the risk of colorectal cancer formation does not warrant screening or surveillance. The recommendations by Jenkins and Fairclough for a lifetime 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 an 15–14-fold increase in the risk of colorectal cancer in acromegolics relative to the general population. This is at odds with larger studies (6, 102, 11, 1041, n = 1634) which overall indicate an increased colorectal cancer risk of 2.5–3-fold. Jenkins and Fairclough advocate an intensive screening protocol beginning at 40 years of age. However, for the same case occurring at 39 years although mean age of the 10 patients with cancer was 67 years. They advocate repeat colonoscopy at five years, or three yearly if at increased risk (determined by adenoma at initial colonoscopy or increased IgF-1 levels). Renenh et al. however conclude that there is no increased incidence of colorectal neoplasms compared with a normal control population (generated from postmortem and colonoscopy data). These data may differ because of the control population and also probably also explain their variance with the larger studies. Based on the current literature, an independent view is that patients might benefit from a single sigmoidoscopy or colonoscopy at approximately 55 years of age. However, no study suggests a risk of proximal neoplasia that warrants the risks and difficulties of a colonoscopy in acromegalic patients. We read with interest the paper by Walker et al. (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 which the pathologists in recognising gastric atrophy, and in Walker’s words, “where to biopsy?” and, are we might add, “how extensively to biopsy?” to correctly detect 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. 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) in which the pathologists might recommend 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 “Western system” 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. 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 curve 4–6 cm proximal to the angularis and one from the greater curve 4–6 cm distal to the angularis); (ii) antral mucosa (one biopsy each from the greater and lesser curves, 3–5 cm proximal to the pyloric ring), and (iii) only one additional biopsy from the pyloric antrum. Histological categories included the basic distinction between non-atrophic and atrophic gastritis. Two pathologists independently

References


Authors’ reply

We thank Drs Perry and Kane and Professor Stewart for their contribution to the debate on this topic. The correspondents acknowledge that patients with acromegaly may 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 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 overly 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 interest 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 context, the incidence of asymptomatic tumours, as well as numerous adenomas. Thus based both on the literature and on our own experience, the single risk of undetected colon cancer far outweighs the theoretical risks of colonoscopy.

P J Jenkins, P D Fairclough
Barts and The London NHS Trust, St Bartholomew’s Hospital, West Smithfield, London EC1A 7BE

Correspondence to P Jenkins; p.jenkins@mdx.ac.uk

Atrophic gastritis: pathology and endoscopy in the reversibility assessment

We read with interest the paper by Walker et al. (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 which the pathologists in recognising gastric atrophy, and in Walker’s words, “where to biopsy?” and, we might add, “how extensively to biopsy?” to correctly detect 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. 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) in which the pathologists might recommend 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 “Western system” 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. 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 curve 4–6 cm proximal to the angularis and one from the greater curve 4–6 cm distal to the angularis); (ii) antral mucosa (one biopsy each from the greater and lesser curves, 3–5 cm proximal to the pyloric ring), and (iii) only one additional biopsy from the pyloric antrum. Histological categories included the basic distinction between non-atrophic and atrophic gastritis. Two pathologists independently

The results of this study were discussed with Pelayo Offerhaus (Fleiss’ value = 0.91). Table 1 shows the atrophy prevalence according to biopsy location.

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, it is far higher than the cost of histological examination, and the price of an angular biopsy is emphasised by the fact that the incisura angularis is a vulnerable point for mucosal tears. 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), 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.

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.

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

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

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 simply balanced by the benefit of an appropriate assessment of gastric disease and a suitable evaluation of the patient’s cancer risk.

M Rugge, M Cassaroo, G Pennelli
Dipartimento di Scienze Oncologiche and Chirurgiche, Università degli Studi di Padova, Italia
G Leandro
Ospedale G De Bellis, Castellana Grotte-Bari, Italia
F Di Mario
Dipartimento di Gastroenterologia, Università di Parma, Italia
F Farinati
Dipartimento di Scienze Chirurgiche and Gastroenterologiche, Università degli Studi di Padova, Italia

Correspondence to: M Rugge; massimo.rugge@unipd.it

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

References

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.

PostScript

Y Komuro, T Watanabe, K Hata, H Nagawa
Department of Surgical Oncology, Faculty of Medicine, University of Tokyo, Tokyo, Japan

Correspondence to: Dr Y Komuro, Department of Surgical Oncology, Faculty of Medicine, University of Tokyo, 7-3-1 Hongo, Bunkyo-ku, Tokyo 113–8655, Japan; komuro@k.t.u-tokyo.ac.jp

References

BOOK REVIEWS

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


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 and autoimmune 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 overlap syndromes are also extensively reviewed. This section of the book demonstrates particularly well how an immunological understanding can provide direct insights into a 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 immunologists 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


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 in 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 gastroenterologists and provides much needed background information about aetiology, screening, pathogenesis, and current treatments. There is no doubt that the book is much more than a textbook on “diagnosis and treatment”. However, overall this is a useful book and a good introduction to liver immunology.

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 collaborative and critical approach that he believes will 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

www.gutjnl.com

1389

Downloaded from http://gut.bmj.com/ on June 28, 2017 - Published by group.bmj.com
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 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
Long term follow up of *Helicobacter pylori* induced gastric diffuse large B cell MALT lymphoma following eradication treatment alone

R Sinharay

Gut 2003 52: 1385
doi: 10.1136/gut.52.9.1385

Updated information and services can be found at:
http://gut.bmj.com/content/52/9/1385.1

These include:

**References**

This article cites 5 articles, 1 of which you can access for free at:
http://gut.bmj.com/content/52/9/1385.1#BIBL

**Email alerting service**

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/