Grading system for inflammation in ulcerative colitis

Editor,—Geboes et al described a grading system for inflammation in ulcerative colitis and carried out rigorous assessment of the reproducibility of this system (Gut 2000;47:404–9). This is a very useful study which fills a void in the histopathology assessment of ulcerative colitis. However, now that this system has been described, its use in clinical practice and clinical trials needs to be considered.

Many of the features that Geboes et al have used in their grading system are described as continuous spectra—for example, chronic inflammation assessed from no increase through to marked increase—but are divided into discrete groups (for example, mild, moderate, marked). This means that these features are ordinal categorical variables rather than continuous real numbers—that is, they have a discrete spectrum. Hence, changes in “chronic inflammation” do not have only a continuous spectrum. There are changes in subtypes of cells, and these changes show a continuous spectrum. Analysis of routinely haematoxylin and eosin stained sections is therefore obviously limited. The aim of our study was to construct and evaluate a scoring system which can be applied routinely. In this system, the distinction between the major grades (for example, structural change, chronic inflammatory infiltration, infiltration of neutrophils in the epithelium, crypt destruction, erosion and ulceration) is more important than the subgrades. The differences between these major grades are clearly defined and do not present as a continuous spectrum. A change from one grade to another is a major difference, which can indicate an important effect, while changes within a grade from mild to moderate are far less important. Furthermore, the distinction between active disease (neutrophils and epithelial damage) and inactive disease is clearly defined. For evaluation of neutrophils in the epithelium, the number of crypts involved was counted.

The results of the reproducibility study presented in table 2 as mean grades were meant to show an example of interobserver agreement. Frequency distribution histograms of the same data are available but were not included because we had to limit the data which were submitted for publication to keep the paper within a reasonable length. The score allows a good comparison for each individual patient as well as a comparison for the major grades and numbers of patients within each grade. The latter allows comparisons between patient groups. The scoring system is under prospective evaluation in clinical trials and has so far been easy to use for routine assessment of microscopic inflammation. The results will be published in due course.

We realise that the distinction between different groups within one grade is not rigorously correct but we still feel that it can be useful, especially as we decided to use the worst aspect for the grading, rather than an average aspect. The correlation between location of neutrophils in the epithelium and occurrence of crypt destruction, erosion, and ulcerations was studied using Spearman’s correlation coefficients.

In general, we agree with Dr Cross that a correct scoring system is needed. On the other hand, such a scoring system should be simple and easy to use. We have tried to find a balance between the different needs and have shown that such a system can be applied with fair interobserver agreement.

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Insulin and gall stones

Editor,—In showing for the first time that raised serum insulin is a risk factor for incident gall stones, independent of body mass index, Misciagna et al (Gut 2000;47:144–7) have made an important contribution. However, they do not seem to realise that we had similar findings in the East Bristol Gallstone Study (population based like theirs)—namely, that raised plasma insulin is a risk factor for prevalent gall stones, at least in men.1 In our study, another significant factor was abdominal fatness or central obesity, but not body mass index (as is usually the case in men), and abdominal fatness probably explained the hyperinsulinaemia as the association of insulin with gall stones disappeared when we controlled for waist-hip ratio. Abdominal fatness is a well known determinant of fasting plasma insulin and it is a pity that Misciagna et al did not include any measure of it in their study.

Should Misciagna et al continue this line of enquiry, they will be well advised to measure the insulin response to eating because in our experience, postprandial as well as fasting levels of insulin are raised in men with gall stones.1 I fully agree with Misciagna et al’s conclusion that “hyperinsulinaemia may play an important role in the aetiology of gall stones”. I also suggest that future studies of gall stone aetiology should include measurements of insulin sensitivity and of its determinants. One such determinant is physical fitness2 and this may be relevant because, in our study, there was a hint that loss of muscle bulk may be associated with gall stones in men. Men with gall stones had not gained weight during adult life more than controls, despite having more abdominal fat, suggesting they had lost more lean body mass.1

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Reply

EDITOR,—We approached with enthusiasm which have now been identified. Of the report were emphasised by the appearance of cytokines known as chemokines, over 40 of inflammatory agent. While an e other adhesion molecule of the report by Salas and colleagues (Gut 2000;14:7–7) and the insightful com-mentators’ selective invocation of potential use of heparin in tissues and on the surface of both endothelial cells and leucocytes. This interaction heightens migration along a fixed gradient, or so-called haptotaxis,5 and favours receptor binding. There is strong evidence that soluble GAGs, including heparin, prevent chemokines binding to their receptors, thus abating their chemotactic potential.

Neither Salas and colleagues nor Perretti and Page chose to mention an anti-chemokine mechanism for the anti-leucocyte migration activity of heparin. We ignore chemokines in our paper, and thus this misses their sheer number and abundance, and the intensity of the effort being directed at discovering pharmaco-mological inhibitors of their function, highlight their critical role in inflammation.

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Management of variceal haemorrhage in cirrhotic patients

EDITOR,—We have serious concerns about several of the recent UK guidelines for the management of variceal haemorrhage in cirrhotic patients (Gut 2000;46(suppl 3 and 4):iii–iiii–115), particularly those that contra-dict current published evidence. We highlight below the ones we feel are the most important.

In the management of acute variceal bleeding, vascular surgery is the method of first choice which was given an AI recommendation. Meta-analysis of all trials of acute bleeding of banding versus injection sclerotherapy have shown no statistically sig-nificant difference between the two treat-ments for either control of bleeding or survival (data derived from 12 studies with 419 patients), with no statistical heterogen-eity.

The implication of recommending ligation for acute bleeding is that double intubation would be necessary in a patient who is actively bleeding so as to attach the ligation device after the initial diagnostic endoscopy. Although we appreciate this would make more risk to the patient; it is common sense that a single intubation would be pref-erable and would take less time. At best the recommendation should be that either endo-scopic technique could be used as first choice, dependent on operator expertise and facilities.

Secondly, there is evidence from ran-domised studies of vasoactive drug therapy combined with endoscopic techniques that combination therapy is superior in terms of control of bleeding. This is based on five ran-domised studies with 610 patients (pooled odds ratio 0.42, 95% confidence interval 0.29–0.6). Publication bias assessment has shown that 29 null or negative studies would be needed to render the results non-significant, and thus this effect is fairly robust. Moreover, in several of these studies vasoac-tive drugs were given before diagnostic endoscopy, demonstrating their utility during the period of resuscitation before endoscopy could be safely performed, which in practice may be several hours after admission. This goes against the recommendation that drugs can be used if endoscopy is not available.

As regards the prevention of rebleeding from sources due to portal hypertension, the treatment of first choice, unless there are contraindications, is either non-selective β blockers as they are equally effective with banding,1 or band ligation. No fully published randomised studies are available with regard to β blockers versus banding. If banding is not available, β blockers should be used, not sclerotherapy, as recommending contraindications or intolerance to β blockers, banding should be used. One can argue cogently that as non-selective β blockers are cheap and do not involve repeated endoscopy sessions, they always should be considered the treatment of first choice.

The recommendation of measuring hepatic venous pressure gradient (HVPG) in patients given β blockers cannot be one for current practice. Only two Spanish groups have suggested this, and it is unclear when a repeat measurement should be performed. Moreover, both a 20% reduction from baseline HVPG or an absolute reduction to less than 12 mm Hg are “protective” from rebleeding, so both end points, and not just the absolute reduction, need to be mentioned if this management strategy is used. In any case the randomised studies of endoscopic therapy used non-selective β blockers empiri-cally to the maximum tolerated by patients so that use of drugs without pressure measure-ment was effective. Lastly, if the recommen-dation of using drugs with re-measurement of pressure is taken to its logical conclusion, all patients should be tried on drugs first, as those who respond have far less rebleeding (10% or less) than patients who receive banding, and secondly, a recommendation of what to do next would need to be made for those who do not reduce their portal pressure (for which as yet there is no evidence).

Lastly, two meta-analyses comparing TIPS with endoscopic techniques have shown that TIPS did not improve survival.13 The increased encephalopathy, greatly increased cost, as well as poor availability of TIPS treatment does not make it a first choice treatment for rebleeding, even in centres with expertise such as the authors’ own, as stated in the guidelines. Thus the AI recommen-dation grading is particularly inappropriate.

With respect to primary prevention of por-tal hypertensive bleeding in cirrhosis, the
recommendation that nitrates should be used if neither β blockers nor banding are available or contraindicated is potentially dangerous. A long term randomised study has shown that at least in elderly patients, nitrates on their own decrease survival. Thus to err on the side of caution, nitrates cannot be recommended as a substitutive therapy.

Finally, the guidelines should have included some issues of general management — for example, association with fluids, easy assessment of portal vein patency, and presence of hepatocellular carcinoma — and an AI recommendation for the use of prophyactic antibiotics in acute bleeding based on the work of the authors quoted. A corrected and improved update of these guidelines is needed soon.

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Reply
EDITOR,—We thank Dr Burroughs and Dr Patch for their interest and helpful comments on the UK guidelines in the management of variceal bleeding. A number of the points raised by them reflects the fact that it is not always possible to directly translate the evidence that is gleaned from clinical trials into clinical practice because of the subjectivity in the definition of evidence based medicine. There is a lot of argument in the literature about what constitutes research evidence. Indeed, there is ongoing debate whether the results of a good randomised controlled trial are more reliable than a meta analysis on the same subject because the latter often suffers from problems introduced by heterogeneity between studies.

For the preparation of the present "guidelines", about 300 papers were reviewed and 208 have been referred to in the paper. It is clear that the vast majority of these studies were not adequately powered to detect differences in mortality and a number of points that have been raised by Dr Burroughs and Dr Patch represent alternative interpretation of the available data which are not necessarily in variance with the "guidelines".

Before discussing the specific points raised by them, it is important to point out that:

● Although the guidelines were written by us, they have undergone several revisions based on peer review organised by the British Society of Gastroenterology (BSG), Liver Section. This review process we believe was extensive and largely anonymous. The guidelines therefore represent the views of the BSG.
● The guidelines were first commissioned in 1996 but finalised for publication following several alterations in mid-1998. Some of the more important data were added into the text (the antibiotic prophylaxis section) during the proof stage.

With respect to the specific comments:
(a) We agree with Dr Burroughs and Dr Patch that studies have shown any significant differences between band ligation and sclerotherapy in their ability to control bleeding. Also, most patients who have had a varical bleed and are undergoing endoscopy are not bleeding actively. It is therefore relatively easy to band in these situations and a double intubation using the new multi-band ligation devices is not necessarily a problem. Studies have also shown that complications from endoscopic therapy in the form of oesophageal ulcers, mediastinitis, and pneumonia are significantly less in the group treated with band ligation compared with sclerotherapy. This is associated with reduced mortality in patients treated with band ligation. It stands to reason therefore that band ligation should be used where possible as there is no significant difference between treatments in their ability to control bleeding but the rate of complications has been shown to be significantly less in the band ligation group.
(b) Interpretation of data regarding the combination of vasoactive drugs with endoscopic therapy in the setting of acute bleeding is fraught with difficulty and there is no clear evidence that these combinations reduce mortality. This is despite a large number of trials in this area. The meta-analysis that Burroughs and Patch (published in 1999) refer to as a justification for the combination treatment shows no differences in survival between groups. The role of vasoactive drugs in the management of variceal bleeding is an area of intense research by a number of centre and data are needed before the combination treatment can be recommended in routine clinical practice.
(c) With respect to secondary prophylaxis of variceal haemorrhage, the literature suggests that combinations such as sclerotherapy, β blockers, or a combination of these are similar in the long term (reviewed by D’Amico and colleagues). Most patients that we treat in the UK with variceal bleeding have underlying alcoholic liver disease and who have a questionable compliance. The recommendation is that if only a β blocker is used we should ensure that this is having some effect on the most important parameter predictive of rebleeding, a portal pressure gradient <12 mm Hg (about 30% of patients in different studies show inadequate portal pressure response to β blocker therapy). It has been shown in a prospective study that in patients being treated with β blockers, none with a hepatic venous pressure gradient <12 mm Hg bled and only 8% of those whose hepatic venous pressure gradient fell by more than 20% on therapy bled during follow up.

However, if independent studies are included in patients being treated with β blockers, this is likely to increase both the cost and invasiveness. We do agree that we should add to the guidelines that a reduction in portal pressure gradient by 20% or more from baseline is acceptable.

(d) The guidelines clearly state what Dr Burroughs and Dr Patch suggest in their letter: “TIPSS is more effective than endoscopic treatment in reducing variceal rebleeding but does not improve survival and is associated with more encephalopathy”. Three studies have shown that TIPSS is more effective than endoscopic treatment in reducing variceal rebleeding but does not improve survival and is associated with more encephalopathy and prophylaxis. The only study that suggests that TIPSS is more expensive is an Italian study in which TIPSS was not strictly being used for secondary prophylaxis with patients being treated instead on a controlled basis six months after their initial variceal bleed. Studies that have compared TIPSS with band ligation have not shown any significant differences in encephalopathy between groups. This has, however, not been borne out in a meta analysis. But it is clear from individual trials and also from the meta-analysis that TIPSS significantly reduces the rate of rebleeding.

(e) The recommendation grade for the use of isosorbide-5-mononitrate (ISMN) in case of failure of propranolol or band ligation is grade B1 and is based on the equivalence study of ISMN and propranolol by Angelico and colleagues. The paper that Dr Burroughs and Dr Patch refer to as a meta analysis of data from a study that was first reported in 1993. A preliminary report of another study has not confirmed these findings and it is clear that more data are needed before nitrates can be considered as being dangerous in the primary prophylaxis of variceal bleeding.

(f) Our brief was to develop guidelines about the management of variceal bleeding and not about the detailed intensive care management. We have however included some pointers in the guidelines which we thought were likely to be useful. We accept that the use of prophylactic antibiotics should be a grade 1A recommendation. This section on the use of antibiotics following a varical bleed was added during the proof stage following the availability of the meta-analysis by Bernard et al in 1996.

We do agree with Dr Burroughs and Dr Patch that the treatment options in portal hypertension are continuously evolving and with the emergence of new data, “guidelines” should be revised to incorporate the advances that have occurred in that time.

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11 Cello JP, Ring EJ, Olcott EW, et al. Received lymphoblastoid IFN at a dose of 7.5 MU/m2 three times a week for 12 weeks and 30 cases received IFN at the same dose but preceded by a four week course of prednisolone. Response to treatment was defined as loss of hepatitis B surface antigen (anti-HBs) occurred in seven (18.4%) patients who responded during therapy and all but one lost HBsAg. After HBsAg clearance, anti-HBc seroconversion and loss of HBV DNA was observed in all patients. Alanine aminotransferase values normalised in 98.3% of patients. None had biochemical or serological relapse within the follow up period.

12 Renal sodium handling in preascitic cirrhosis

EDITOR,—We read with interest the commentaries by Claría and Rodés (Gut 1999;45:639) on our paper published in Gut which re-examined the mechanisms of renal sodium retention in patients with preascitic cirrhosis.1 In summary, in our patients we observed indirect evidence of expanded central vascular fluid volume compared with healthy controls and thought this physiopathological alteration was due to slight reduced value of glomerular filtration rate (measured as creatinine clearance) and, mainly, to increased distal tubular reabsorption of sodium when expressed as a fraction of the filtered sodium load that is reabsorbed by the distal nephron (26.9 (6.7) to 12.5 (3.4)% respectively; p<0.05).2 Claría and Rodés advanced two criticisms and affirmed that our results, obtained by means of the lithium clearance and fractional excretion technique, may be influenced by two fundamental flaws. Firstly, the reliability of lithium clearance as a marker of distal fluid delivery in clinical conditions characterised by low fractional sodium excretion (about 0.40%) has not been proved due to possible lithium reabsorption in the distal nephron.3 Secondly, in Claría and Rodés’s opinion, our observation of more avid fractional sodium reabsorption by the distal nephron in compensated cirrhosis merely reflects diminished delivery of fluid and sodium to the distal segments (due to reduced glomerular filtration) rather than increased distal tubular sodium reabsorption.
Correspondence to:
EDITOR,—In their letter, Sansoè and Ferrari
ing the already demonstrated increase in cen-
inappropriate avidity of sodium reabsorption
However, we consider that our results on
filtration rate using creatinine clearance.
produced in studies assessing renal function in
sodium to the distal nephron were not lower
than in healthy subjects (30.7 (9.3)
but somewhat higher, even if not significantly,
sodium excretion below which li-
thium clearance is a useful marker of
fractional sodium excretion of 1% has been
proposed as a safer limit by Koomans and
we consider that the value of
fractional sodium excretion below
which lithium clearance is disquali-
that lithium is actively reabsorbed along the
distal tubule in condi-
tions characterised by low fractional sodium
reabsorption. In preliminary studies, the esti-
mated limit of fractional sodium excretion below
which this problem arises has been
established as 0.02%. Conversely, comprehensive
studies of micropuncture have revealed that
this value may vary from 0.8% to 0.65% in
sodium depleting states. Finally, a value of
fractional sodium excretion of 1% has been
proposed as a safer limit by Koomans and
which lithium clearance remains unresolved
in cirrhosis, data derived from this method in
cirrhotic patients should be interpreted with
certainty.
We should also point out that preascitic cirrhotic patients included in Sansoè et al’s study (Gut 1999;45:750–5) had significantly lower values than controls for glomerular fil-
tion rate, as determined by creatinine
filtration rate, as determined by creatinine
clearance. Those findings are not consistent with
those previously reported in compen-
sated cirrhotics using more sensitive clear-
ance techniques such as inulin clearance.
In summary, it is gratifying to see that
Lithium clearance is a useful marker of
proximal sodium delivery remains unresolved
in cirrhosis; data derived from this method in
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index and I personally do not like the idea of paginating in sections and chapters. With a book of this length it is surely easier to simply number the pages. However, these are minor complaints and on the whole I would recommend this book to anyone interested in liver disease and particularly to trainees in gastroenterology, or hepatobiliary surgery who will come back to this book again and again.

D H ADAMS


“A picture is worth a thousand words” is as applicable to the teaching of gastroenterology as in any other context now that gastroenterology has become a visual science. Any atlas must stand or fall on the quality of the photographs and here the reader will not be disappointed as the vast majority are of excellent clarity and content. The second edition of this Atlas of Gastroenterology provides the most comprehensive visual images in gastroenterology this reviewer has seen, covering the broad spectrum of gastroenterology—histology, endoscopic images, CT scans, radiouclide imaging, and magnetic resonance imaging, including MR cholangiopancreatography. However, there are no “virtual endoscopic” images, which is a surprise and disappointment. The atlas has a user friendly format setting pictures in their clinical context making perfect sense and easy access. There is a series of chapters entitled “Approaches to common gastrointestinal problems” beginning with a brief review of the clinical problem followed by a range of images used in establishing diagnosis, thus putting the image in context with the clinical findings at the appropriate point in the management pathway. There are also chapters on particular gastrointestinal diseases and a series of chapters illustrating diagnostic and therapeutic techniques, all written and compiled by acknowledged experts in their field. Reference lists are suitably brief and up to date.

The atlas seeks to provide more than a picture of gastroenterology but perhaps goes rather too far by providing information that would normally be within a textbook of gastroenterology. For example, there is a chapter entitled “Advice to travellers” that gives information about required vaccinations in various parts of the world and drug treatment for traveller’s diarrhoea. There are also several chapters with extensive clinical information that is more than just an accompaniment to the images. In one chapter, there is a long list of drugs likely to induce liver disease—appropriate for a textbook but not for an atlas, particularly when this atlas is designed for use with its partner the Textbook of Gastroenterology by the same editors.

This atlas provides the most up to date high quality illustrative review of gastroenterology and could perhaps only be improved by the addition of a slide or CD version. Access to the images via the Internet will probably be the next step but I for one would miss the pleasure of leafing through a book.


This is a small book which looks at specific aspects of gastric surgery from a laparoscopic approach. The overall format is attractive in that a chapter on physiology precedes the section on laparoscopic surgery. It does, however, in view of the rather concise nature, fall between two stools in that it is a specialist book and therefore does not necessarily appeal to the general trainee, but it is too short and the referencing is too limited to be a definitive text. The book succeeds on the basis that the laparoscopic approach is correct and there is very little discussion on non-laparoscopic and open surgery. This may well be appropriate in the form of laparoscopic antireflux surgery and cardiomotomy but is certainly not in the form of antiobesity surgery or surgery for cancer. The impression that the laparoscopic approach is well established is inaccurate for these latter conditions and malignancy, where open surgery holds sway. The discussion on laparoscopic antireflux surgery is limited to the 360° Nissen loose floppy wrap. The operation is described nicely with clear photographs which is a characteristic of the entire text. However, there is no discussion on the alternatives to a 360° wrap, namely a toupee 180° procedure or even the more modern partial anterior fundoplications. The various merits of these procedures would be an addition to the text as well as the role of the laparoscope in revisional surgery, and some comparison with open operations. Similarly, for cardiomotomy for achalasia, a success rate related to open cardiomotomy would be beneficial. Preceding these two operative sections however are two good chapters on the pathophysiology of reflux and achalasia. It is a pity in laparoscopic antireflux surgery that more comment is not made on the significant increase in the incidence of such surgery with the advent of the laparoscope. Is this a good thing or not? The pros and cons of treatment could be better discussed. With regard to open surgery, this really has to be emphasised as being experimental. Comparison with these success rates versus those of open surgery and a reflection on the reality of the situation, as seen in Western Europe where the disease presents at a more advanced stage, and the role of other modalities such as chemo/radiotherapy, would benefit the textbook and would expand it into a more comprehensive text. On the plus side however, the illustrations are superb and the intraoperative photographs explain the laparoscopic nodal dissection extremely clearly. It is not however a textbook of operative surgery. This book will appeal to the more specialist clinicians in upper gastrointestinal surgery and provides a cheaper and smaller alternative to the more weighty texts.

R C MASON

CORRECTION

An error occurred in the abstracts supplement Gut 2000;48(suppl I):A68. For abstract 254, PC Hayes was the senior author.

S CAIRNS
Long term follow up of interferon responder children with chronic hepatitis B

N KOÇAK, I N SALTIK, H ÖZEN, F GÜRAKAN and A YÜCE

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