LETTERS TO THE EDITOR

Measurement of the stiffness of endoscopes—a plea for commonality

EDITOR,—In a previous issue (*Gut* 2000;**46**:801–8), Brooker and colleagues described their experience with an exciting new variable stiffness colonoscope. They made the point that a stiffer colonoscope shaft reduces recurrent looping but makes passage through an angulated sigmoid more difficult and causes more stretching and hence pain when loops do occur. Conversely, the more flexible thinner paediatric instruments are better for negotiating a fixed or narrow sigmoid colon but then tend to allow recurrent loop formation later in the procedure. Their randomised trial using either a standard Olympus CF200HL (13.3 mm shaft diameter) or a prototype (Olympus XCF-SH230L—12.9 mm shaft diameter) variable stiffness colonoscope looked very promising although in one case a paediatric Olympus PCF230I (11.3 mm shaft diameter) was required to get past a fixed sigmoid secondary to diverticular disease.

In addition to Brooker *et al*, there are a number of research workers[1–4] and endoscope manufacturers interested in colonoscope/flexible sigmoidoscope shaft stiffness and its relation to patient discomfort/procedure time, yet sadly there is no agreement as to the best way to express (and thus directly compare) results. The beam deflection technique adopted by Brooker *et al* appeared to us to be an entirely arbitrary one involving a strain gauge, 5 cm shaft deflection, and just three duplicate measurements every 10 cm along the three instruments.

We agree with Wehrmeyer and colleagues[1] that flexural rigidity is a more precise, accurate, and reproducible engineering parameter to measure when trying to compare endoscope shaft stiffness. In beam bending theory, the flexural rigidity is *EI*, which is the product of the modulus of elasticity (or Young's modulus) *E* and the second moment of area *I* of the beam cross section about an axis through the centroid perpendicular to the plane of bending. *EI* is given by the following expression:

$EI = WL^3/192\delta$

where *W* is the load applied at the centre of the beam, *L* is the length of the beam, and δ is the deflection at the centre. In our own studies, the value of *W* (typically either 0.5 to 1 Newtons) was selected such that δ (mean of 10 readings) was less than 0.5% of the length of the 20 cm "beam". An example of the results obtained is shown in fig 1 in which mean (SD) flexural rigidity values (in N cm^2) are compared for (a) an Olympus PCF 240I (11.2 mm diameter) instrument, (b) a fibreoptic Olympus CF20HL (13.3 mm diameter) endoscope, and (c) an Olympus CF-240AL (12 mm diameter) variable stiffness colonoscope. These three instruments were taken as being the nearest we had available in our own unit to those employed in the study of Brooker *et al*. Although our results are expressed in different units, the shape of the curves are remarkably similar to those published by Brooker *et al*. We confirm that the now commercially available variable stiffness Olympus colonoscope can indeed significantly alter its shaft stiffness from being almost as floppy as a paediatric endoscope to as stiff as a standard Olympus 20HL near its most proximal end.

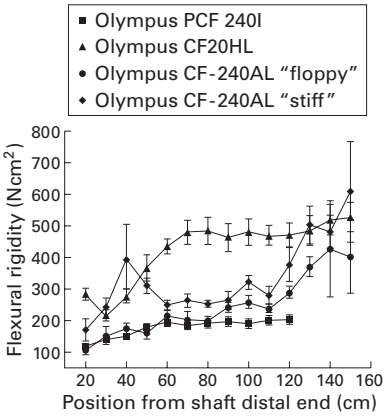

Figure 1 Mean (SD) flexural rigidity measurements (N cm^2) of three different colonoscopes: (a) Olympus PCF 240I, (b) Olympus CF20HL, and (c) Olympus variable stiffness CF-240AL instrument in its "floppy" and "stiff" modes.

We agree with Brooker *et al* that modifications that may enhance the efficacy of a variable stiffness colonoscope might include "more floppiness in the paediatric setting and greater stiffness at the maximum stiffness setting" .

We welcome debate and discussion on how best to measure endoscope shaft stiffness. In the meantime, until a better way of expressing the results is suggested, it would seem to us that some form of simple beam displacement methodology to determine flexural rigidity has the advantage of at least being relatively easy, reproducible, and inexpensive to perform.

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Research outcomes in British gastroenterology: an audit of the subsequent full publication of abstracts presented at the British Society of Gastroenterology

EDITOR,—The presentation of abstracts at scientific meetings provides an opportunity to rapidly convey the results of novel research. It also allows the researcher a chance to receive informal peer review. This may help to clarify aspects of the work, particularly in the identification and correction of potential weaknesses prior to submission for full publication. Although abstracts submitted to conferences are peer reviewed, this process may not be as rigorous as that of an indexed journal considering publication of the full manuscript.[1]

Presentation of an abstract at a prestigious meeting may suggest that full publication is probable. Certainly, acceptance as opposed to rejection increases the likelihood of subsequent publication, but this is not absolute.[2] Other medical specialities have studied their societies' publication rates and this value varies from 21% to 66%.[3 4]

There have been no studies evaluating the outcome of abstracts presented at gastroenterology meetings. Therefore, we audited the publication rate of abstracts presented at a single British Society of Gastroenterology (BSG) meeting.

All abstracts presented at the BSG meeting of March 1994 (n=255) were assessed. Two independent database searches were performed (MEDLINE and EMBASE) using cross referencing of first author, senior author, and key words from the abstract title. The abstract and possible resultant manuscript were then examined in tandem to ensure they represented the same study. Where no paper appeared to have been published, the authors were contacted to ascertain the outcome of their abstract.

Factors which may influence publication, including study type, design, category, sample size, journal of publication, impact factor, and lag time to publication were analysed. Data pertaining to submission/publication at the meeting of the American Gastroenterology Association (AGA) in the same year were also collected. Statistical analyses were performed using contingency tables and χ^2 statistics for nominal data and the Mann-Whitney U for continuous data.

There were 178 abstracts (69.8%) published from this meeting. Median lag time to full publication (fig 1) was 19 months (range 0–66). Of the abstracts published, 61 (23.9%) were in high impact factor journals (arbitrarily designated $\geq$4). The mean impact factor was 2.5 (median 2.9).

There were 96 abstracts from this particular BSG that were concordantly submitted to the AGA. Of these, 73 were accepted for presentation. Ultimately, 58 were fully published. Presentation at the AGA in the same year was the only factor that significantly increased the likelihood of publication (p=0.001; odds ratio 3.1 (95% confidence interval 1.5–6.4)). Acceptance at the AGA was a strong predictor of subsequent publication and may represent the hypothesis that concordance of two independent referee systems often reflects the papers of greatest scientific merit.[5] Alternatively, this may suggest that AGA reviewers are more stringent. This is not possible to assess with the data available.

This is the first study to assess publication rates of the BSG or indeed any specialty in the UK. We chose to study the abstracts of the 1994 BSG meeting because previous reports have suggested that the majority of abstracts are published in indexed journals within four years of presentation.[3 4 6] The outcome of one individual meeting may not be considered as representative of other meetings and could limit the validity of our

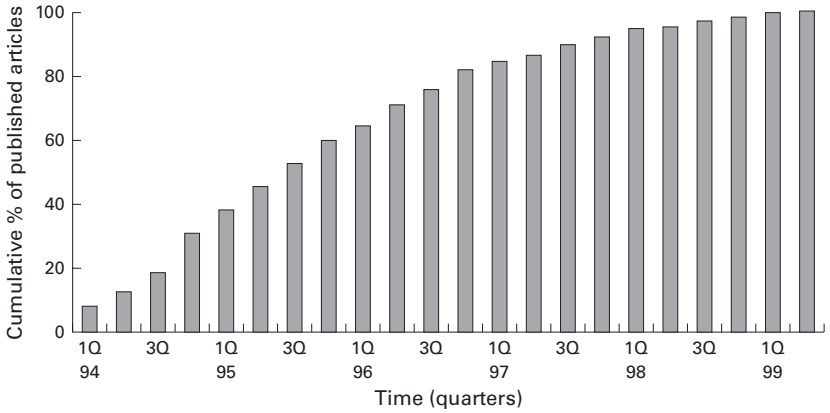

Figure 1 Time between abstract presentation and publication.

audit. However, previous similar studies from other societies have suggested that their publication rates vary by as little as 5% from year to year. Thus assessing one meeting may be adequate.[6] In conclusion, acceptance of abstracts by the BSG meeting suggests more than a 2 in 3 chance of subsequent full publication. This compares favourably with similar studies of other societies.

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Leptin in the human stomach

EDITOR,—After the report in 1998 by Bado and colleagues[1] describing the presence of leptin in rat stomach, we have recently reported the first evidence of leptin in the stomach mucosa of humans.[2] It was shown that the cells in the lower half of the stomach glands were clearly immunoreactive for leptin, and both leptin mRNA and leptin protein in the human gastric epithelium were detected. Western blot analysis showed the presence of a 16 kDa band corresponding to leptin and a 19 kDa band which, as suggested for rats,[1] could represent a leptin precursor. It was also shown that secretory granules of chief cells contain this hormone, suggesting that gastric leptin could function in the short term system control of feeding behaviour and that it is secreted (probably together with pepsinogen) in the stomach lumen by chief cells. Confirmation of these findings was reported by Sobhani and colleagues.[3] They also showed the presence of leptin receptor in stomach epithelium, suggesting a possible paracrine pathway for leptin. Stomach leptin levels seem to be higher in humans than in rats.[1 3]

Interestingly, Sobhani *et al* have also shown[3] that gastric leptin is simultaneously released into the blood and into the gastric juice by pentagastrin and secretin. They suggested that secretin has a direct effect on gastric chief cells, an idea based on the presence of secretin receptors on these cells[4] and on the efficacy of secretin in stimulating pepsinogen secretion.[5]

However, by immunoelectron microscopy we observed[2] the presence of leptin not only in chief cells but also in endocrine cells exhibiting a distinctive morphology in the basal portion of the gland. These cells showed secretory granules labelled with many leptin-gold particles.[2] Its ultrastructure corresponded to the P cell type.[6 7]

Thus secretory granules of both endocrine and chief cells contain leptin.[2] It is probably secreted in the stomach lumen by chief cells and into the stomach circulation by a special type of endocrine cell. The observation[3] that intravenous infusions of pentagastrin or secretin caused an increase in circulating leptin levels and leptin release into gastric juice is in keeping with both endocrine and exocrine secretory sources. They could function in the short term system to control feeding behaviour and in the gastrointestinal lumen to regulate the availability of nutrients acting in the sites where a non-degraded form of hormone would approach.

Our observation of much lower levels of leptin immunostaining in a patient under postpandrial conditions compared with five fasted patients[2] is in agreement with a likely functional response of human stomach leptin to food intake. The effects of cholecystokinin in the rat[1] and of pentagastrin and secretin in humans[3] stimulating emptying of stomach leptin are all strong arguments for a short term satiety role of leptin. There is also the observation that leptin interacts synergically with other short term satiety peptides.[8]

There is a need for further investigation in humans, with difficulties arising from ethical limitations. However, taken together, both articles[2 3] on leptin in the human stomach and the previous report in rats,[1] we can conclude that three important pathways (endocrine, exocrine, and autocrine) for the action of leptin are present in human stomach, where the main physiological role for this hormone is foreseen.

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Histological and genetic heterogeneity in synchronous hepatocellular carcinoma

EDITOR,—The recent paper by Sirivatanauksorn *et al* (*Gut* 1999;**45**:761–5) focused once again on the unresolved question as to whether (i) hepatocellular carcinoma (HCC) in human liver develops from a single clone or from multiple parallel clones and (ii) among multiple tumour nodules present in many patients, the smaller lesions represent intrahepatic metastases or "de novo" cancers. The authors correctly acknowledge that "information on the clonal origin of tumours will influence management strategies for prevention of recurrence after operation". They used arbitrarily primed polymerase chain reaction (AP-PCR)[1] to compare the DNA fingerprint of HCCs and regenerative nodules (RNs) removed from 13 cirrhotic explant livers. They found considerable genomic heterogeneity in 54 HCCs and 31 RNs that were microdissected. No two nodules (either RNs or HCCs) had identical electrophoretic

patterns. Contrary to expectation, even "satellite" nodules in close proximity within the same segment of the liver were found to have distinct genomic patterns. They concluded that their data suggest poor patient survival after surgical resection if the smaller tumours are "de novo" lesions rather than metastases.

We would like to make some comments. HCC arising in cirrhosis is frequently multifocal. This is supported by epidemiological studies and by the fact that a diffuse underlying viral disease facilitates multifocality, in particular when HCC is related to hepatitis C viral infection. In addition, primary multifocality is supported by the high incidence of "preneoplastic lesions" such as dysplastic nodules in the surrounding liver. However, a word of caution is suggested before extrapolating data and conclusions from this paper to all HCCs.[2 3] In fact, there is a selection bias that could be responsible for the observed findings. Cirrhotic explant livers are selected for liver transplantation because of a diffuse, usually viral related cirrhosis in the absence of clinically evident large nodules. In these cases HCC is often an incidental finding and is mostly, if not always, multifocal. On the contrary, in a consecutive surgical series of resected patients, 44 of 49 patients without cirrhosis (88.2%) had a unique macroscopic nodule restricted to the right lobe, larger than 5 cm in nine of 44, whereas right lobe involvement occurred in 66 of 104 patients with cirrhosis (63.4%; p=0.001[4 5]). Satellite nodules were found in only four of the 49 patients of the former group (8%).[5] Viral infection was detected in 38.7% of patients in the former group compared with 93.7% of patients with cirrhosis (p<0.001).[5] In particular, the following findings were observed: (1) an as yet not well defined proportion of patients (10–20%) showed uninodular HCCs which were well capsulated, located in the right liver, with low tendency to vascular spread, and usually not associated with viral infection and/or clinically evident cirrhosis. Interestingly, these lesions remained unique even when greater than 5–6 cm (up to 10 cm) without satellite lesions; (2) when larger than 10 cm, these HCCs had conspicuous histological and genomic heterogeneity within the same initial nodule, showing different histological variants (up to five), each of which had a different genetic pattern. In these very large tumours, satellite nodules were similar to one of these five variants, or even different, but were still nodules originating from the initial "mother" lesion. In fact, in most cases, they remained restricted to the right lobe, close to the larger nodule, without left lobe involvement.[5] Therefore, even if genetic analysis is a powerful tool in detecting that two samples with the same genetic fingerprints belong to the same clone, a word of caution is suggested before stating the opposite, namely that a satellite nodule in close proximity to a larger lesion, within the same liver segment, is a different tumour, a "de novo" lesion, rather than a metastasis from the original tumour, simply because of genetic heterogeneity.

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Phenotypic expression of the HFE gene mutation (C282Y) among the hospitalised population

EDITOR,—Distante and colleagues (*Gut* 2000;**47**:575–9) found that C282Y homozygotes detected by testing all patients acutely admitted to hospital for non-liver associated problems had a considerably lower transferrin saturation during the acute illness (median 27%) compared with at follow up (median 71%). Indeed their strategy of performing C282Y genotyping on all acute patients with a raised transferrin saturation (>50%) would have detected only 1/14 (8%) C282Y homozygotes. This is in contrast with our study of HFE (C282Y) gene mutation and phenotypic expression in outpatients referred to a liver clinic.[1] In this setting we found that transferrin saturation was a far more reliable predictor of genetic haemochromatosis than that reported by Distante *et al* in acutely ill inpatients. A strategy of performing C282Y genotyping on our outpatients with a raised transferrin saturation (>50%) would have detected 8/9 (89%) C282Y homozygotes in our study but only 8% of acutely ill C282Y homozygotes in the Norwegian study. In contrast, a false positive elevation of serum transferrin saturation and hepatic iron index have been reported in patients with end stage liver disease undergoing transplantation who did not have genetic haemochromatosis.[2]

Distante *et al* identified 14 homozygotes from 2027 hospitalised patients screened (0.74%), of whom 94% were Caucasian, from a high prevalence area for the HFE gene mutation. In our ethnically mixed population of 326 unselected liver outpatients from London, 79% were Caucasian. As Distante *et al* conclude, a serum transferrin saturation remains the screening test of choice for detecting haemochromatosis. However, we feel it needs emphasising, in the light of their findings, that screening or opportunistic testing for haemochromatosis is best restricted to relatively well subjects in the outpatient setting. Moreover, selecting target populations (such as liver[2] or diabetic clinics[3]) is likely to identify larger numbers of homozygotes than testing all comers.

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Reply

EDITOR,—We thank Drs Moodie and Maxwell for their comments on our paper.

Indeed, it ought to be stressed that desaturation of transferrin occurs in inflammatory states. This should be taken into account to avoid false negative results when screening for haemochromatosis both in the general population and in target groups (such as in liver or diabetic clinics).

In the London outpatient group,[1] transferrin saturation >50% detected 89% of C282Y homozygotes. In the series of 312 liver disease patients admitted at our hospital,[2] 14/18 (78%) liver biopsy proved haemochromatosis patients had an increased transferrin saturation >50%. In line with Cotler and colleagues,[3] we also found a false positive elevation of transferrin saturation in 27/105 (26%) patients with alcoholic liver disease and 11/132 (8.3%) with chronic non-alcoholic liver disease.

We agree with Drs Moodie and Maxwell's suggestion that transferrin saturation screening for haemochromatosis should be performed in relatively well subjects. Both their comments and our findings highlight some of the limitations of transferrin saturation as a screening parameter for genetic haemochromatosis.

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The African enigma: the parasite's perspective

EDITOR,—We would like to thank Professor MacDonald for his interest in our work, and for his recent comment on our paper (*Gut* 2001;**48**:10–11). However, we must take issue with a number of his comments.

With respect to the clinical implications of our study,[1] we have not advocated treatment of *Helicobacter pylori* infected patients with intestinal parasites for the purpose of inducing a shift in the Th1/Th2 immune response, a possibility raised by Professor MacDonald. As Professor MacDonald is well aware, this approach has been suggested for a number of chronic diseases such as inflammatory bowel disease. However, in the case of chronic *H pylori* infection, antibiotic therapy has proved effective and in our opinion is a much safer and more palatable approach for most patients.

We appreciate Professor MacDonald's careful analysis of our histopathological results. However, we are confused regarding his comments of "negative points" that were not sufficiently emphasised. The main findings in our study were that *H polygyrus* coinfection attenuated the degree of parietal cell loss, mucous cell hyperplasia, and metaplasia, and resulted in an increase in bacterial colonisation. The changes in inflammation, as graded histologically, were clearly less marked. These latter findings simply underscore the notion that it is the T helper type of immune response rather than the overall severity or histological grade of inflammation that determines eventual epithelial injury. In addition, it is generally recognised that in the *H felis* mouse model, there is some degree of variability with respect to the degree of inflammatory response, which is well illustrated by our data. It is puzzling that Professor MacDonald would equate this biological variation with a problem regarding the "quality of the data".

Further questions were raised regarding the conclusion that *H polygyrus* infection biased the immune response to *H felis* along the same pathway. Our data clearly show that Th1 immune responses were decreased and Th2 immune responses were increased, and this was supported not only by cytokine profiles but also by *H felis* specific humoral responses. We agree that this immunomodulatory effect may not be true for every intestinal parasite. However, as pointed out in our paper, other parasites such as *Schistosoma mansoni* have also been shown to induce polarised Th2 responses and downregulate intestinal Th1 responses. The question that Professor MacDonald raises regarding the specificity of the Th2 response for *H felis* (as opposed to *H polygyrus*) is an interesting one. However, the explanation that the increased Th2 cytokines in the stomach of mice infected with both *H felis* and *H polygyrus* are derived from "migration into the inflamed gastric mucosa of Th2 cells responding to *H polygyrus* antigens" seems less likely in our opinion.

We would agree that induction of a Th2 anti-*H felis* response may not be desirable in every instance. However, this comment again seems to miss the point. Data from numerous animal models have shown that a reduction in mucosal Th1 response together with upregulation of a mucosal Th2 response is associated with decreased progression to gastric atrophy and preneoplasia, and taken in this context, a Th2 polarised response is clearly a desirable outcome. Data from a number of laboratories have suggested that increased bacterial colonisation by itself does not lead to adverse consequences; in fact, in most mouse models the level of bacterial colonisation was inversely related to the degree of atrophy and metaplasia. It is our contention that a decreased Th1 response associated with a high rate of gastric colonisation is highly preferable to a strong Th1 response associated with decreased *Helicobacter* colonisation.

Finally, we concluded from our study that the ability of a concurrent helminth infection to ameliorate *Helicobacter* spp. induced gastric disease might partially explain the African enigma. It is clear that intestinal helminth infections are common in Africa, and also that there is marked variation in the pattern of helminth infections from continent to continent. The review by Professor MacDonald, aside from contributing insightful commentary on the topography of Africa and South America, has not provided additional information regarding the variable patterns and types of helminth infections in the two continents. Recent data from Mitchell and colleagues[2] have supported the hypothesis that a Th2 polarised response to *H pylori* is more common in Africa while a Th1 polarised response is more common in Europe and Australia. We would suggest that further investigations of intestinal helminths, as well as host genetics,[3] should be considered to account for this different pattern of immune response to *H pylori*, as well as the differing rates of gastric cancer induction.

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Liver biopsy: "blind" or under ultrasound control

EDITOR,—We wish to comment on the paper by Shah *et al* (*Gut* 1999;**45**:628–9) and subsequent correspondence (*Gut* 2000;**47**:455) from Aspinall.

The case for the superior safety of ultrasound guided liver biopsy has not yet been proved and the British Society of Gastroenterology Guidelines (1999) do not advocate this as routine best practice. Perforation of the gall bladder is a very rare complication (eight times in 68 276 biopsies in the series reported by Piccinino and colleagues[1]) and one of us has seen the records of a case in which the gall bladder was punctured at liver biopsy done under real time ultrasound guidance. No technique is entirely safe. Until such time as the evidence clearly supports a change in standard practice, a bid for legal redress by patients who suffer a complication of "blind" liver biopsy is unlikely to succeed, assuming that the indication for the biopsy was sound, the usual precautions were observed, that detailed informed consent was obtained setting out the nature of the risks and their frequency, and that the operator had sufficient experience or supervision.

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199 The Strand, London WC2R 1DR, UK

1 Piccinino F, Sagnelli E, Pasquale G, *et al.* Complications following percutaneous liver biopsy. A multicentre retrospective study on 68,276 biopsies. *J Hepatol* 1986;**2**:165–73.

Reply

EDITOR,—We agree with Dr Murray-Lyon and Mr de Wilde that patients should be properly informed about how a procedure is performed, the complications and risks, as well as alternatives. It is in this area of alternatives that many "prudent patients" will choose to have an ultrasound guided biopsy. In the era of the Bristol Enquiry into deaths associated with cardiac surgery, any "reasonable doctor" can do no less.

It is important that members of the medical and legal profession appreciate the difference between an "act of god" and an "accident". Everybody appreciates that complications arising from these procedures are not deliberate. Settlements for "acts of god" are unlikely to be successful whereas "accidents" (be it car, train, or medical) are considered appropriate to seek recompense if they could have potentially been avoided or the risks reduced.

We agree that death and/or gall bladder perforation is rare following liver biopsy. This does not remove the requirement however for best practice with the lowest possible risk to the patient. In layman's terms, Lindor *et al* reported a twofold higher risk of bleeding using the "blind" approach with the probability of this occurring by chance being one in 14.[1] We very much hope that practitioners such as Dr Murray-Lyon are advising their patients of figures such as these as part of their consent procedure so that the patient can make an informed decision as to which method they opt for.

An additional point of our original article, which has not been alluded to by Dr Murray-Lyon and Mr de Wilde, is that many centres no longer use the "blind approach". The training available for registrars to become proficient in this approach is therefore declining. As stated in previous correspondence, the culture of "see one, do one, teach one" is no longer acceptable.

It is unfortunate that this discussion is unlikely to be settled until a legal action takes place. The scenario is becoming clearer: a civil court action (where most likely probability is the burden of proof) with a patient who has suffered a complication following a "blind" procedure. The case will be decided on the perceived competence and training of the doctor involved and on the details of the information provided to the patient and the consent obtained. What is clear however is that the unfortunate patient is unlikely to be a gastroenterologist or radiologist as, of the numerous colleagues we have asked, not a single one has opted for the blind approach if both were available.

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1 Lindor KD, Bru C, Jorgensen RA, *et al*. The role of ultrasonography and automatic-needle biopsy in outpatient percutaneous liver biopsy. *Hepatology* 1996;**23**:1079–83.

Guidelines for the management of iron deficiency anaemia

EDITOR,—In reply to Dr Scott's letter (*Gut* 2001;**48**:284), I would add that when iron deficiency coexists with the anaemia of chronic disorders (ACD) such as rheumatoid arthritis, a low transferrin saturation loses its diagnostic specificity due to the fact that comparable degrees of transferrin saturation occur in patients with the sole diagnosis of ACD.[1] The corollary, in this context, is that the behaviour of ferritin as an acute phase reactant negates the expected fall in serum ferritin, with consequent loss of sensitivity in this parameter.[1] Even so, in a comparison of bone marrow findings with tests such as transferrin saturation and serum ferritin in a study comprising patients with a variety of haematological disorders, "the most useful single variable to discriminate patients with iron deficiency from all other patients was serum ferritin".[2] Since then, the most promising test for identifying iron deficiency when it coexists with chronic inflammation has been the ratio of serum transferrin receptor/log serum ferritin (so-called TfR-F index), which achieves an unequivocal separation between iron deficient patients with coexisting chronic inflammation compared with those with the sole diagnosis of chronic inflammation.[3] Even in that study, the receiver operating characteristic curve for serum ferritin, on its own, was diagnostically superior to the one generated by transferrin saturation.[3]

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1 O'Connor NTJ, Hoffbrand AV. Anaemia of chronic disease. In: Delamore IW, Liu Yin JA, eds. *Haematological aspects of systemic disease*. London: Bailliere Tindall, 1990:52–7.
2 Witte DL, Kraemer DF, Johnson GF, *et al.* Prediction of bone marrow iron findings from tests performed on peripheral blood. *Am J Clin Pathol* 1986;**85**:202–6.
3 Punnonen K, Irjala K, Rajamaki A. Serum transferrin receptor and its ratio to serum ferritin in the diagnosis of iron deficiency. *Blood* 1997;**89**:1052–7.

NOTES

Falk Symposium No 123: VI International Symposium on Inflammatory Bowel Diseases

This Falk Symposium will be held on 3–5 September 2001 in Istanbul, Turkey. Further information: Falk Foundation e.V. - Congress Division, Leinenweberstr. 5, PO Box 6529, D-79041 Freiburg, Germany. Tel: +49 761 15 14 0; fax: +49 761 15 14 359; email: symposia@falkfoundation.de

Falk Symposium No 124: Medical Imaging in Gastroenterology and Hepatology

This Falk Symposium will be held on 28–29 September 2001 in Hannover, Germany. Further information: see Falk Symposium No 123 above.

9th Asian Conference on Diarrheal Diseases and Nutrition

This meeting will be held on 28–30 September 2001 in New Delhi, India. The organisers hope the meeting will promote meaningful and effective collaboration among individuals/institutions towards control of the major health problems in Asia, particularly those affecting women and children. Further information: Professor M K Bhan, Coordinator, Centre for Diarrheal Disease and Nutrition Research, All India Institute of Medical Sciences, New Delhi. Tel: +91 11 6963822; fax: +91 11 6862662; email: ascodd2001@rediffmail.com

VI Congress of the International Xenotransplantation Association

This congress will be held on 29 September to 3 October 2001 in Chicago, USA. Further information: Felicissimo & Associates Inc., 205 Viger Avenue West, Suite 201, Montreal, Quebec, Canada H2Z 1G2. Tel: +1 514 874 1998; fax: +1 514 874 1580; email: info@ixa2001chicago.com; website: www.ixa2001chicago.com

Falk Symposium No 125: Cytokines in Liver Injury and Repair

This Falk Symposium will be held on 30 September to 1 October 2001 in Hannover, Germany. Further information: see Falk Symposium No 123 above.

Falk Symposium No 126: Hepatocyte Transplantation

This Falk Symposium will be held on 2–3 October 2001 in Hannover, Germany. Further information: see Falk Symposium No 123 above.

EASL Single Topic Conference

The EASL Single Topic Conference "Liver fibrosis: from basic science to clinical targets" will be held on 12–13 October 2001 in Florence, Italy. Organisers: Massimo Pinzani (University of Florence) and Detlef Schuppan (University of Erlangen-Nuernberg). The aim of the conference is to provide the latest information on this key area of hepatology and to translate the current knowledge into clinical terms. It is directed at both the expert in the field and the general hepatologist. Further information: Massimo Pinzani, Dipartimento di Medicina Interna, Università degli Studi di Firenze, Viale GB Morgagni, 85, I-50134 Firenze, Italy. Tel: +39 055 4277845; fax: +44 39 055 417123; email: m.pinzani@dfc.unifi.it

Lecture Course in Coloproctology

This course will be held on 15–17 October 2001 in Harrow, UK. Professor Russell Stitz from Australia will be the Sir Alan Parks Visiting Professor and, for the first time, there will be a Sir Francis Avery Jones Visiting Professor, which will be Professor Paul Rutgeerts from Belgium. Further information: The Administrator, St Mark's Academic Institute, St Mark's Hospital, Northwick Park, Harrow, Middx, HA1 3UJ, UK. Tel: +44 (0)20 8235 4046/8; fax: +44 (0)20 8235 4039; email: stmarks@ic.ac.uk; website: www.stmarkshospital.org.uk

International Symposium on Hyperammonemia, Liver Failure and Hepatic Encephalopathy

This symposium will be held on 20–22 October 2001 in Valencia, Spain. Further information: Cátedra Santiago Grisolía, Fundación Museu de les Ciències Príncipe Felipe, Ciutat de les Arts i les Ciències, Avda. Instituto Obrero, s/n, 46013 Valencia, Spain. Tel: +34 96 197 44 66; fax: +34 96 197 44 70; email: catedrasg@cac.es. **Deadline for receipt of abstracts is 15 July 2001.**

ICGH-2: The Second Iranian Congress of Gastroenterology and Hepatology

The main Iranian meeting of gastroenterologists and researchers in this field will be held on 27 October to 1 November 2001 in Tehran, Iran. Further information: Dr Shahin Merat, Digestive Diseases Research Center, Shariati Hospital, N. Kargar Street, Tehran 14114, Iran. Tel: +98 911 717 3966; fax: +98 21 225 3635; email: merat@ams.ac.ir; website: www.ams.ac.ir/icgh. **Deadline for submission of abstracts is 31 May 2001.**

Falk Symposium No 127: Autoimmune Diseases in Pediatric Gastroenterology

This Falk Symposium will be held on 8–9 November 2001 in Basel, Switzerland. Further information: see Falk Symposium No 123 above.

42nd Annual Conference of the Indian Society of Gastroenterology

This conference will be held on 23–29 November 2001 in Lucknow, India. The programme includes two pre-conference symposia (on gastrointestinal motility and scientific communication, on 23 November), a one day postgraduate course or CME (24 November), and an endoscopy workshop (28–29 November). Further information: Dr S R Naik, Department of Gastroenterology, SGPGI, Lucknow 226014, India. Tel: +91 522 440700 or 440800, ext 2400; fax: +91 522 440078 or 440017; website: www.sgpgi.ac.in/conf/isg2001.html

41st St Andrew's Day Festival Symposium on Therapeutics

This will be held on 6–7 December 2001 in Edinburgh, UK. Further information: Ms Eileen Strawn, Syposium Co-ordinator. Tel: +44 (0)131 225 7324; fax: +44 (0)131 220 4393; email: e.strawn@rcpe.ac.uk; website: www.rcpe.ac.uk

14th Intensive European Course of Digestive Endoscopy

This course will be held on 17–18 December 2001 in Strasbourg, France. Further information: Michele Centonze Conseil, 6 bis rue des Cendriers, 75020 Paris, France. Tel: +33 1 44 62 68 80; fax: +33 1 43 49 68 58; email: mail@m-centonze-conseil.com