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 loopong but makes passage through an angulated sigmoid more difficult and causes more stretching and hence pain when loops do occur. Conversely, the more flexible endoscope 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 CF-2000L (13.5 mm shaft diameter) or a prototype (Olympus XCP-SH230L—12.9 mm shaft diameter) variable stiffness colonoscope looked very promising although in one case a paediatric Olympus PCF230L (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 workers7 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 replicate measurements every 10 cm along the three instruments.

We agree with Wehrmeyer and colleagues 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: 

\[ EI = \frac{Wl^2}{192b} \]

where W is the load applied at the centre of the beam, L is the length of the beam, and \( \delta \) 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 \( \delta \) (mean of 10 readings) was less than 0.5% of the length L of the 20 cm “beam”. An example of the results obtained is shown in fig 1 in which 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 200L near its most proximal end.

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

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 \( \chi^2 \) statistics for nominal data and the Mann-Whitney U test 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 (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 considered by AGA reviewers. It is not possible to assess with the data available if AGA reviewers are more stringent. This may help to clarify 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 specialties 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 gastrointestinal 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=225) were assessed. Two independent database searches were formed (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.

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Leptin in the human stomach

Editor.—After the report in 1998 by Bado and colleagues describing the presence of leptin in rat stomach, we have recently reported the first evidence of leptin in the stomach mucosa of humans.1 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, 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.2 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.3

Interestingly, Sobhani et al have also shown4 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 cells5 and on the efficacy of secretin in stimulating pepsinogen secretion.6

However, by immunoelectron microscopy we observed7 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.8 Its ultrastructure corresponded to the P cell type.9

Thus secretory granules of both endocrine and chief cells contain leptin.10 It is probably secreted in the stomach lumen by chief cells and into the stomach circulation by a special type of endocrine cell. The observation11 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.12

Our observation of much lower levels of leptin immunostaining in a patient under postprandial conditions compared with five fasted patients13 is in agreement with a likely functional response of human stomach leptin to food intake. The effects of cholecystokinin in the rat14 and of pentagastrin and secretin in humans15 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.16

There is a need for further investigation in humans, with difficulties arising from ethical limitations. However, taken together, both articles7,10 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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This work was supported by European Commission COCOP 1998 Action from DGXII to S Cinti; by Ancona University grants to S Cinti; by project “Physiopathology, Pharmacology and Clinical Aspects of Obesity”, to S Cinti; and by DGESIC, Secretaria de Estado de Educacion, Universidades, Investigacion y Desarrollo, Spain (grant PM97-0094) to A Palou.


Histological and genetic heterogeneity in synchronous hepatocellular carcinoma

Editor.—The recent paper by Sirivathanaukornsarn 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) whether cryptic mutations in the DNA that encode the hepatitis C virus core protein and thymidylate synthetase lead to tumour heterogeneity. They used arbitrarily primed polymerase chain reaction (AP-PCR) to compare the DNA fingerprint of HCCs and regenerative nodules (RN) 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 fingerprint of HCCs and regenerative nodules.

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 included in the resection 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 virial infection. In addition, primary multifocality is supported by the high incidence of "neoplastic" lesions such as dysplastic nodules from cirrhotic inducing liver. Furthermore, a word of caution is suggested before extrapolating data and conclusions from this paper to all HCCs. In fact, there is a selection bias that could be responsible for the observed findings. Candidates 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 treated, 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). Satellite nodules were found in only four of the 49 patients of the former group (8%). Viral infection was detected in 38.7% of patients in the former group compared with 93.7% of patients with cirrhosis (p<0.001). In particular, the following findings were observed: (1) an as yet not well defined proportion of patients (10–20%) showed an unimodal HCC which were well capsulated, located in the right liver, with low tendency to vascular spread, and usually not associated with viral infection. Cytologically 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 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 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 large nodule, without left lobe involvement. 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 the 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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The study was supported in part by: the National Research Institute (CNR) (grants No 93.00239.CT04, No 94.00376.CT04, No 95.00897.CT04); Regione Toscana (grant No 358/C, 1995); MURST 40%-MURST 60%; and Telethon (grant E611).


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

EDITOR,—Distante and colleagues (Gut 2000;47:47–52) 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 71%). Indeed their strategy of performing C282Y genotyping on all acute patients admitted to hospital may identify a small group of patients with a raised transferrin saturation (>50%) who would have detection 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 patients. 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 our 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 or diabetic clinics) is likely to identify larger numbers of homozygotes than testing all comers.

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Replay

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 412 liver disease patients admitted to our hospital,14/18 (78%) liver biopsy proved haemochromatosis patients had an increased transferrin saturation >50%. In line with Cotler and colleagues,2 we also found a far higher 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.


www.gutjnl.com
With respect to the clinical implications of our study, 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 criticism that “negative points” that were not sufficiently emphasised. The main findings in our study were that _H pylori_ co-infection attenuated the degree of parietal cell loss, mucus 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 that is more relevant 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 fелиs_ 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 pylori_ infection biased the immune response to _H fелиs_ 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 fелиs_ 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 polarising responses and downregulate intestinal Th1 responses. The question that Professor MacDonald raises regarding the specificity of the Th2 response for _H fелиs_ (as opposed to _H pylori_) is an interesting one. However, the explanation that the increased Th2 cytokines in the stomach of mice infected with both _H fелиs_ and _H pylori_ are derived from “migration into the inflamed gastric mucosa of Th2 cells responding to _H pylori_ antigens” seems less likely in our opinion.

We would agree that induction of a Th2 anti- _H fелиs_ 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 _Helicobacter_ induced gastric atrophy. (Nature Med 2000;6:536-42)

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 with whom we have discussed this case, not one has opted for the blind approach if both were available.
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.2 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.”3 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 TIR-F index), which achieves an unequivocal separation between iron deficient patients with coexisting chronic inflammation compared with those with the sole diagnosis of iron deficiency.4 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.5

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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 49; 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 H3Z 1G2. Tel: +1 514 897 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 2 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 Delph 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: +39 055 417123; email: m.pinzani@delf.unifi.it

Lecture Course in Coloproctology

This course will be held on 15–17 October 2001 in Harrow, UK. Professor Russell Stitt 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.stmarkshosp- ital.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: Catedra Santiago Grisolía, Fundación Museu de les Ciències Príncipe Felipe, Ciutat de les Arts i les Ciències, Avda. Institut Obreiro, 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/ighc. 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/issg2001.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, Symposium 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 44 62 68 58; email: mail@m-centonze-conseil.com