LETTERS TO
THE EDITOR

Measurement of the stiffness of
dendoscopes—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 loop but makes passage through an angulated sigmoid more difficult and causes more stretching and hence pain when loops do occur. Conversely, the more flexible colonoscope shaft is 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-200HL (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 workers’ 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 stiffness (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’ 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:

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EI = W/L^3
\]

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 (mean of 10 readings) was less than 0.5% of the length of the 20 cm “beam”. An example of the 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 cm²) are compared for (a) an Olympus PCF 240L (11.2 mm diameter) instrument, (b) a fiberoptic Olympus CF200HL (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 curve is 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.

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.

G D BELL
Sunderland University Medical Sciences Faculty, University of Sunderland, Sunderland, UK

K BURN
School of Computing, Engineering and Technology, University of Sunderland, Sunderland, UK

Correspondence to: Professor GD Bell, Endoscopy Unit, Sunderland Royal Hospital, Kayll Road, Sunderland SR4 7TP, UK. duncan_bell@compuserve.com


Research outcomes in British gastroentrology: 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 (=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 manuscripts 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 contribute to 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 χ² 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 0.0–68). Of the abstracts, 34 (23.9%) were in high impact factor journals (arbitrarily designated ≥4). The mean impact factor was 2.5 (median 2.9).

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 correspondence of two independent review systems often reflects the papers of greatest scientific merit. 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.1,3,4

The outcome of one individual meeting may not be considered as representative of other specialities, but this study supports the validity of our
Leptin in the human stomach

Editor.—After the report in 1998 by Bado and colleagues1 describing the presence of leptin in the 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 in chief cells but also in endocrine cells indicating a possible presence of secretin receptors on these cells1 and on the efficacy of secretin in stimulating pepsinogen secretion.1

However, by immunoelectron microscopy we observed2 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.2

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 observation2 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 postprandial conditions compared with five fasted patients3 is in agreement with a likely functional response of human stomach leptin to food intake. The effects of cholecystokinin in the rat4 and of pentagastrin and secretin in humans5 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.6

There is a need for further investigation in humans, with difficulties arising from ethical limitations. However, taken together, both articles1,2 on leptin in the human stomach and the previous report in rats,7 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 Sirivatanaukorn et al1 (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 (ii) genetic changes in 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) to compare the DNA fingerprint of HCCs and regenerative nodules (RNs) removed from cirrhotic explant livers. They found considerable genomic heterogeneity in 54 HCCs and 31 RNs that were microdissected. The two nodules (either RNs or HCCs) had identical electrophoretic patterns.
patterns. Contrary to expectation, even “satellite” nodules in close proximity with 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 possess distinct genomic patterns. Contrary to expectation, even “satellite” nodules were similar to one another, whereas right lobe involvement occurred in 66 of 104 patients with cirrhosis (88.2%) had a unique macroscopic involvement occurred in 66 of 104 patients with cirrhosis (88.2%) had a unique macroscopic involvement occurred in 66 of 104 patients with cirrhosis (88.2%) had a unique macroscopic involvement occurred in 66 of 104 patients with cirrhosis (88.2%) had a unique macroscopic involvement occurred in 66 of 104 patients with cirrhosis (88.2%) had a unique macroscopic involvement occurred in 66 of 104 patients with cirrhosis (88.2%) had a unique macroscopic involvement occurred in 66 of 104 patients with cirrhosis (88.2%) had a unique macroscopic involvement occurred in 66 of 104 patients with cirrhosis (88.2%) had a unique macroscopic involvement occurred in 66 of 104 patients with cirrhosis (88.2%) had a unique macroscopic involvement occurred in 66 of 104 patients with cirrhosis (88.2%) had a unique 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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 use of the “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 that is more 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 our 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 raised by Professor MacDonald is also as to the specificity of the Th2 response for H felis (as opposed to H polygyrus) and this 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 inflated 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 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 metaplasia, 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 decrease in the 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 have suggested 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, should be considered to account for this variation in patterns of immune response to H pylori, as well as the differing rates of gastric cancer induction.

J FG OX
Division of Comparative Medicine, Massachusetts Institute of Technology, 77 Massachusetts Ave, Bldg. 16-825G, Cambridge, MA 02139, USA

T C WANG
Division of Gastroenterology, University of Massachusetts Medical Center, Biotech 2, Suite 202, 373 Plantation St, Worcester, MA 01605-2177, USA

C NAGLER-ANDERSON
Massachusetts Institute of Technology Laboratory, Massachusetts General Hospital East, Building 149, 11th Street, Charlestown, MA 02129, USA

Correspondence to: JG Fox. jgfox@mit.edu


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 (e.g. eight times more frequent than biopsies in the series reported by Piccinino and colleagues’ 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.

I M MURRAY-LYON
Chelsea and Westminster Hospital
369 Fulham Road, London SW10 9NH, UK

R de WILDE QC
199 The Strand, London WC2R 1DB, UK


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 the 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 worse.

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 redress 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 “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.

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 with whom I have discussed this with, not a single one has opted for the blind approach if both were available.

R J PLAYFORD
Gastroenterology Section, Imperial College School of Medicine, Hammersmith Campus, London, UK
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 chronic inflammation.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

O J OLOBO
Department of Medicine for the Elderly, Tameside General Hospital, Ashton under Lyne OL6 9RW, UK andrea.hunt.exchange@ttc-tt.nhs.uk


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 8; 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/her 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 Diarrhela Diseases and Nutrition Research, All India Institute of Medical Sciences, New Delhi, Tel: +91 11 6963822; fax: +91 11 6862662; email: asccom2001@rediffmail.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 Derfel 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@dfe.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 Príncep Felipe, Ciutat de les Arts i les Ciències, Avda. Instituto Obiero, 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.

IGGH-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/iggh. 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 Straw, Symposium Co-ordinator. Tel: +44 (0)131 225 7324; fax: +44 (0)131 439 4393; email: e.straw@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 Conseild, 6 bis rue des Cendriers, 75020 Paris, France. Tel: +33 1 44 62 68 80; fax: +33 1 44 69 68 58; email: mail@m-centonze-conseil.com