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 the stiffer colonoscopy 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-colonoscope mucoscopes 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-200HL (13.5 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 PCF2300 (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 this measure (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:

\[
EI = W/L^2 \times b/3
\]

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 Newtons) was selected such that the beam, of area product of the modulus of elasticity (or EI theory, the flexural rigidity is EI), is given by the second moment of area about the neutral axis of the beam section. In our own studies, the modulus of elasticity of the beam material was determined to be 200 GPa.

The now commercially available variable stiffness Olympus colonoscopes have indeed significantly altered its shaft stiffness from being almost as floppy as a paediatric endoscope to as stiff as a standard Olympus 200HL 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”.

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 GB Bell, Endoscopy Unit, Sunderland Royal Hospital, Kayll Road, Sunderland SR4 7TP, UK. duncan_bell@compuserve.com


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 possible. 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 and judged 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 for continuous data.

There were 97 abstracts from this particular BSG that were considered by the AGA. Of these, 73 were accepted for publication (23.9%) in the AGA.

The abstract and possible resultant manuscript were then examined and judged 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 for continuous data.

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.1,5 The outcome of one individual meeting may not be considered as representative of other meetings and should yield the validity of our
Figure 1  Time between abstract presentation and publication.

Leptin in the human stomach

Editor.—After the report in 1998 by Bado and colleagues describing the presence of leptin in the 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.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.4

Interestingly, Sobhani et al have also shown 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 and on the efficacy of secretin in stimulating pepsinogen secretion.5

However, by immunoelectron microscopy we observed6 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.7 Its ultrastructure corresponded to the P cell type.8

Thus secretory granules of both endocrine and chief cells contain leptin. 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 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 patients9 is in agreement with a likely functional response of human stomach leptin to food intake. The effects of cholecystokinin in the rat10 and of pentagastrin and secretin in humans11 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.12

There is a need for further investigation in humans, with difficulties arising from ethical limitations. However, taken together, both articles13 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.

S CINTI
R DE MATTEIS
E CERESI
Institute of Normal Human Morphology-Anatomy, University of Ancona, 60100 Ancona, Italy

C PICÓ
J OLIVER
P OLIVER
A PALOU
Departamento de Biología Fundamental y Ciencias de la Salud, Universitat de les Illes Balears, Ctra. Valldemossa Km 7.5, 07071 Palma de Mallorca, Spain

A OBRADOR
Hospital Son Dureta, Palma de Mallorca, Spain

C MAFFEI
Department of Pathiatrics, University Hospital, Verona University, Verona, Italy

Correspondence to: Professor A Palou. dbfallpo@ps.us.es

This work was supported by European Commission COST 918 Action from DG XII 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, Secretaría de Estado de Educación, Universidades, Investigación y Desarrollo, Spain (grant PM97-0094) to A Palou.


Histological and genetic heterogeneity in synchronous hepatocellular carcinoma

Editor.—The recent paper by Sirivatanan 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; (ii) genetic 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. Nine new nodules (eight RNs or HCCs) had identical electrophoretic

www.gutjnl.com

Gut: first published as 10.1136/gut.49.1.156 on 1 July 2001. Downloaded from http://gut.bmj.com/ on May 11, 2022 by guest. Protected by copyright.
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 were still nodules originating from the initial lesion, within the same liver segment, is a different genetic pattern. In these very large tumours, satellite nodules were similar to one of the colon lesions, 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. 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.

Corespondence to: Dr F Cetta, Institute of Surgical Clinic, University of Siena, New Poloclinico-Viale Braschi-53100 Siena, Italy. cetta@unisi.it

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;47:331) found that C282Y homozygotes detected by transferrin saturation >60% is a cost effective screening parameter for genetic haemochromatosis. This is in contrast with our study of HFE (C282Y) gene mutation and phenotypic expression in outpatients referred to a liver clinic. In this setting we found that transferrin saturation was a 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 (70%) 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.

In the London outpatient group, 1 transferrin saturation >50% detected 89% of C282Y homozygotes. In the series of 412 liver disease patients admitted at 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 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.

S DISTANTE
H BELL
Hepatology Unit, Department of Medicine,
Aber University Hospital, Oslo 0154, Norway
Correspondence to: Dr S Distante.
Sonia.distante@iolos.uio.no

1 Moodie S, Ang L, Leen GV, et al. Restricting HFE genotyping to patients with a transferrin saturation >50% is a cost effective screening strategy in liver clinic patients. J Hepatol 2000;32(suppl 2):133.

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.

1 Moodie S, Ang L, Leen GV, et al. Restricting HFE genotyping to patients with a transferrin saturation >50% is a cost effective screening strategy in liver clinic patients. J Hepatol 2000;32(suppl 2):133.

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.

1 Moodie S, Ang L, Leen GV, et al. Restricting HFE genotyping to patients with a transferrin saturation >50% is a cost effective screening strategy in liver clinic patients. J Hepatol 2000;32(suppl 2):133.

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.

1 Moodie S, Ang L, Leen GV, et al. Restricting HFE genotyping to patients with a transferrin saturation >50% is a cost effective screening strategy in liver clinic patients. J Hepatol 2000;32(suppl 2):133.
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[`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, 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 Th1 helper type of immune response that is important 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 pylori* 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 polarisation of Th1 responses and downregulate intestinal Th2 responses. The question raised by Professor MacDonald that Professor MacDonald raises regarding the specificity of the Th2 response for *H felis* (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 felis* and *H pylori* are derived from “migration into the inflamed gastric mucosa of Th2 cells responding to *H pylori* antigen” 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 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 *Helicobacter* 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 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 hosts genetics, should be considered to account for the 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. 16-825C, Cambridge, MA 02139, USA

T C WANG
Division of Gastroenterology, University of Massachusetts Medical Center, Biostch 2, Suite 202, 373 Plantation St, Worcester, MA 01605-2377, USA

C NAGLER-ANDERSON
Massachusetts Institute of Technology Laboratory, Massachusetts General Hospital East, Building 149, 1st Street, Charlestown, MA 02129, USA

Correspondence to: JG Fox, jfox@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 (eight times in 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 ultrasound guidance; the patient did not do well. 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.

M Murray-Lyon
Chelse and Westminster Hospital
369 Fulham Road, London SW10 9NW, UK

R de Wilde QC
199 The Strand, London WC2R 1DR, 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 alternatives. It is in this area of practice that the various practices that have been advocated in the UK are an example of the practice of medicine rather than the practice of science.

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, and 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 risk to the patient. In layman’s terms, Lindor et al reported a two-fold higher risk of bleeding using the “blind” approach with the probability of this occurring by chance being one in 14. 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 expert at percutaneous liver biopsy is rare 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, and 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.

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 our case, only 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.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 iron deficiency.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

O J OLORIBE
Department of Medicine for the Elderly, Tameside General Hospital, Fountain St, Ashton under Lyne OL6 9RW, UK
andrea.hirst@exchange.tgcps-tr.nwest.nhs.uk


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., Department of Gastroenterology, Leicestershire General Hospital, Leicester, UK. Correspondence to: R Playford. r.playford@ic.ac.uk


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/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: ascod2001@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 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 125 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: +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@ac.uk; website: www.stmarkshosp. potal.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 Príncep Felipe, Ciutat de les Arts i les Ciències, Avda. Instituto Obreiro, s/n, 46013 Valencia, Spain. Tel: +34 96 197 44 66; fax: +34 96 197 44 70; email: catedrags@cacs.es. Deadline for receipt of abstracts is 15 July 2001.

IGCCG-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 14119, Iran. Tel: +98 911 717 3966; fax: +98 21 225 3635; email: merat@ams.ac.ir; website: www.ams.ac.ir/igcc. 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 125 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, 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 de l’Erdre, 37000 Tours, France. Tel: +33 4 79 58 77 31; email: mail@m-centonze-conseil.com