

# PostScript

## LETTERS

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### *H pylori* infection and reflux oesophagitis

We read with considerable interest the paper by Kuipers *et al* (*Gut* 2004;53:12-20) which found no significant adverse impact on the severity of reflux disease or its control after two years of omeprazole therapy following *H pylori* eradication, during which time gastritis largely healed. In the commentary by McColl (*Gut* 2004;53:5-7), it is stated that although published data are conflicting, Schwizer *et al* reported improvement in reflux symptoms following *H pylori* treatment.<sup>1</sup> Our preliminary published data,<sup>2</sup> which are in accordance with those of Schwizer and colleagues,<sup>1</sup> consisted of a small cohort of 69 patients with gastro-oesophageal reflux disease (GORD) and irritable bowel syndrome (IBS). Forty patients were treated with omeprazole (20 mg/day) plus trimebutine (600 mg/day) for three months (group A) and 29 were treated with omeprazole (20 mg/day) as monotherapy for an equal period of time (group B). Inclusion and exclusion criteria matched those of Kuipers *et al*. Upper and lower gastrointestinal endoscopic, histological, and clinical evaluations were made at baseline. Furthermore, upper gastrointestinal evaluation was repeated three

months post-treatment. At baseline, oesophagitis, confirmed by histology, and the histological presence of *H pylori* were observed in 67.5% and 62.1% and in 80% and 82.8% of groups A and B of patients, respectively. All *H pylori* positive patients received eradication treatment, as analysed in Kuipers *et al*'s paper. The eradication rate was observed in 84% of *H pylori*/positive patients in group A and in 83% of *H pylori*/positive patients in group B. Three months post-treatment, there was a significant improvement in GORD ( $p=0.003$ ), IBS symptoms ( $p<0.0001$ ), and oesophagitis ( $p=0.029$ ) in group A compared with group B. At baseline, all 24 *H pylori*/positive patients who received omeprazole and the eradication regimen had GORD symptoms and 15 (62.5%) had histologically proven oesophagitis. Three months post-treatment, GORD was present in 12 (50%) patients ( $p<0.0001$ ) and oesophagitis in nine (37.5%) ( $p>0.05$ ). As improvement in oesophagitis did not reach a statistically significant level, our study was continued and results are shown in table 1 (unpublished data). All 45 *H pylori*/positive patients who received omeprazole and the eradication regimen had GORD and 29 (64.4%) had oesophagitis at baseline. Three months post-treatment, 22 (48.9%) had GORD symptoms ( $p<0.00001$ ) and 18 (40%) had oesophagitis ( $p=0.03$ ) (Wilcoxon's rank sum test, two tailed  $p$  values; data not shown in table 1). There was a statistically better response in patients who also received trimebutine.

In our preliminary study, Barrett's oesophagus was observed in eight (20%) of 40 and in five (17.2%) of 29 patients in groups A and B, respectively, similar to that (24.3%) observed by Kuipers *et al*. As there is an increased prevalence of IBS in patients with GORD,<sup>3</sup> it would be interesting to know how many of the patients in Kuipers *et al*'s study had symptoms suggestive of IBS, and if their regimen had results similar to ours.

Our data show that *H pylori* is frequent in GORD and may contribute to the pathogenesis of GORD by several mechanisms.<sup>3</sup> Also, we propose that the increasing prevalence of GORD may be partially explained not just by the decrease in prevalence of *H pylori*

infection, as suggested by McColl (*Gut* 2004;53:5-7), but rather by healing of *H pylori* associated peptic ulcer disease, which coexists with GORD.<sup>4,5</sup> Thus eliminating peptic ulcer disease unmasks GORD.<sup>7</sup>

In our latest unpublished data, 18 (48.6%) of 37 patients, in whom *H pylori* was eradicated, had reflux symptoms on omeprazole compared with four (50%) of eight patients in whom *H pylori* was not eradicated. Although the latter group was too small to draw definite conclusions, it seems that eradicating *H pylori* did not make GORD more difficult to control. While the editorial advocates that *H pylori* eradication makes it more difficult to achieve long term control of GORD with omeprazole therapy, we suggest that *H pylori* eradication leads to better control of GORD symptoms and improves oesophagitis.

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**Table 1** Patient profile prior to and after the therapeutic regimens and comparisons between the two groups

	Group A (n=92) (omeprazole plus trimebutine)					Group B (n=56) (omeprazole)					Significance (p)			
	Prior		After 3 months			Prior		After 3 months			A v B		A1b v B1b	A2 v B2
	A1 (n=73)	A2 (n=19)	A1a (n=12)	A1b (n=61)	A2 (n=19)	B1 (n=45)	B2 (n=11)	B1a (n=8)	B1b (n=37)	B2 (n=11)	Prior	After	After	After
GORD symptoms	73	19	5	10	3	45	11	4	18	7	NS	<0.001	<0.005	<0.025
Oesophagitis	51	12	5	10	3	29	7	2	16	5	NS	<0.01	<0.01	NS
IBS symptoms	73	19	2	7	3	45	11	4	23	7	NS	<0.001	<0.001	<0.025

$p$  values between the two groups were calculated using the  $\chi^2$  test with Yates' correction (two tailed  $p$  values).

Group A, patients were assigned to three months of omeprazole plus trimebutine treatment; group B, patients were assigned to three months of omeprazole monotherapy; groups A1, B1, *H pylori* positive patients prior to treatment assigned to omeprazole plus trimebutine or omeprazole alone, respectively; groups A1a, B1a, patients from groups A1 and B1, respectively, who remained *H pylori* positive after the eradication regimen; groups A1b, B1b, patients from groups A1 and B1, respectively, in whom *H pylori* infection was eradicated; groups A2, B2, *H pylori* negative patients prior to and after treatment with omeprazole plus trimebutine or omeprazole alone, respectively

## Research trends in British gastroenterology: publication rates in newly appointed NHS consultants over a nine year period

It has been suggested that medical research within the UK may be in decline.<sup>1,2</sup> Possible explanations for this could be the shortened training scheme created by the Calman Specialist Registrar (SpR) post, reduced availability of research funding, or the progressive expansion of the consultant body (as a government imperative to provide a consultant delivered service).<sup>1-4</sup> Although this may be the perception, there has previously been no published evidence to demonstrate a reduction in research output. In this study, we wished to observe any overall trend in the number of publications and higher degrees that trainees (in gastroenterology) have at the time of their National Health Service (NHS) consultant appointments over a nine year period.

### Participants, methods, and results

All consultant appointments and place of training were noted over a fixed period from February 1993 to April 2001 (courtesy of the British Society of Gastroenterology (BSG)). The BSG is considered to have a comprehensive list of its trainees and consultants.<sup>4</sup> This has been used as a source for the prediction of manpower needs in gastroenterology.<sup>4</sup> This model has subsequently been applied to other medical subspecialties.<sup>4</sup> We cross referenced this source with the Medical Directory and BSG members' handbook. We performed a PubMed and Embase search noting the number and type of publications of each consultant. This search included a lag time of up to 19 months post-appointment. This period has previously been described as the mean time from submission to publication.<sup>5</sup> Higher degrees held by each consultant were also noted.

Over the nine years, 349 appointments were made. We excluded consultant to consultant transfers and appointments to or from academic posts ( $n = 52$ ). Also excluded were trainees who had subsequently left the UK or the medical register, as well as individuals where data were difficult to obtain due to name and centre similarities ( $n = 50$ ).

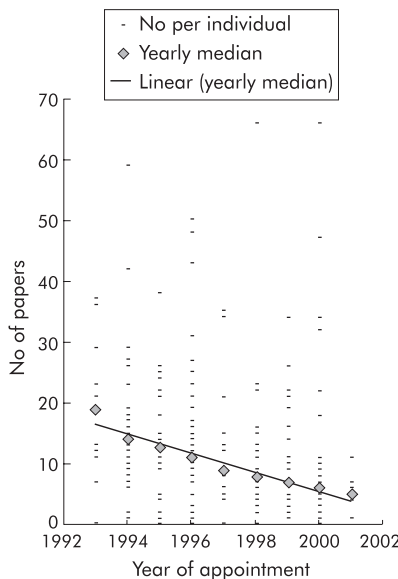
Statistical analysis was performed using Microsoft Excel to produce a linear regression line and correlation coefficient ( $r$ ) to show the trend and strength of any relationship of the median publication rate over the time sampled. The nine years of medians gave 7 degrees of freedom ( $df = 7$ ). We used  $\chi^2$  distribution to show any significant difference between the groups.

There was a year by year significant decreasing trend in the median number of publications by SpRs prior to their NHS consultant appointment, from 19 in 1993 to five in 2001 (fig 1).

Specifically assessing first author publications revealed a similar downward trend ( $r = 0.836$   $df = 7$ ;  $p < 0.01$ ). No statistical differences were observed between university hospital and district general hospital consultants when considering mean publication rates or obtaining a higher degree (data available but not supplied).

### Comment

This is the first study to demonstrate a significant decreasing trend in the number



**Figure 1** Individual numbers of publications for each consultant appointed in a given year and the median for that year (one mark can obviously represent more than one consultant). Correlation coefficient  $r = -0.96$   $df = 7$ ;  $p < 0.001$ .

of publications obtained by a gastroenterology trainee at time of their appointment to an NHS consultant post. The total publication rate can act as a tool in assessing academic activity among trainees and could be extrapolated to all medical specialties.<sup>5</sup>

There are limitations to our observations—publications may have been missed despite using Embase and PubMed. In addition, we excluded 50 consultants ( $50/349 = 14.9\%$ ) due to an inability to recognise their publication data or they were untraceable. We would not expect this omission to skew our results significantly due to the uniform proportional distribution of these individuals over the nine year period. It may be that they represent trainees who never published throughout their training. This would suggest even less overall academic activity than we have described.

Our data support the hypothesis that medical research (at least among NHS consultants) is in decline. Similar studies are required to validate this observation within other medical subspecialties (in the UK) and in other countries.

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## Diagnostic endoscopy: does it help?

Having questioned the value of therapeutic endoscopy, let me question the value of diagnostic endoscopy, having lived through the days from when it was not available to its current status. Having given the matter considerable thought,<sup>1</sup> I seriously doubt that it has been of any value. It might even have had an adverse effect on outcome by delaying operative intervention in those who need it and among whom most deaths occur.

If done emergently it is difficult to visualise the bleeding site, especially in those who require surgery and whose rate of blood loss is greatest. Its greatest value may be in excluding the presence of oesophageal bleeding from varices but this is arguably best done on the operating table by the surgeon. In the UK, many centres no longer perform endoscopy emergently preferring to wait until the bleeding has stopped and a better diagnostic evaluation can be made on the daily endoscopy list. This may be good for documentation but is of doubtful benefit in managing patients because of the inevitable delay in surgical intervention in those that need it.

Operative mortality for bleeding ulcers used to be in the order of 15%. I suspect it is very much higher today because surgeons do far less ulcer surgery. This is likely to be especially true if the operations are delegated to registrars in the middle of the night. It might be wise to take a fresh look at the problem.

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## Prediction of oesophageal varices with platelet count/spleen diameter ratio or platelets alone

We read with great interest the article by Giannini *et al* on platelet count/spleen diameter ratio or platelets alone as predictors of oesophageal varices (*Gut* 2003;**52**:1200–5).

As the incidence of chronic liver diseases is growing, we are convinced that the development of non-invasive predictive tools to identify cirrhotic patients with oesophageal varices is of major interest. Several markers have been studied, and among them platelet count is commonly reported to be a good predictor of oesophageal varices. However, in the eight studies already published,<sup>1–8</sup> their discriminative power was moderate, with

areas under the receiver operating curve (ROC) of 0.70 or less for platelets alone<sup>4</sup> and for indexes combining platelets with other markers.<sup>1,4,5</sup> Most of these studies included heterogeneous groups of patients, with compensated and decompensated cirrhosis.

In our unit, we performed prospectively platelet count and screening upper oesogastroduodenoscopy on the same day in 41 patients with compensated cirrhosis and confirmed the moderate value of platelet count alone (AUROC = 0.70 (0.07); Thabut, data not shown). The major drawback of platelet count is that it can depend on factors other than portal hypertension in cirrhotic patients. To avoid this bias, Giannini *et al* developed an index based on platelet count/spleen diameter ratio and found far better results than previous studies, with a c index (equivalent to the area under the ROC) of 0.92 for patients with compensated liver cirrhosis. However, we were surprised to see that the use of platelets/spleen diameter ratio did not add significant discrimination to platelet count alone (c index of 0.92 *v* 0.88) in their population.

On this point, their excellent results could not be explained by the discriminative power of their index but by the excellent diagnostic power of platelet count alone in their series. Several explanations can be raised, and one is the high rate of viral related cirrhosis in their patients where platelet count is less liable to variations compared with, for example, in alcoholic patients. This point is of major concern for the further validation of their index, recommended by the authors themselves, in other populations.

In conclusion, Giannini *et al* have found a very good index for predicting the presence of oesophageal varices in cirrhotic patients. We believe that the excellent results they obtained were not due to their index but to the surprisingly good performance of platelet count alone in their patients. Considering the results for platelet count as a predictor of oesophageal varices in previously published studies, we fear that the warranted validation studies of this index will show less exciting results.

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## Author's reply

We thank Thabut *et al* for their interesting comments on our paper (*Gut* 2003;52:1200-5). Indeed, their letter allows us to focus on some aspects of our study that we feel need to be emphasised further.

As a general rule, a surrogate marker for a given variable (that is, presence/absence of oesophageal varices) that already has a definite diagnostic procedure (that is, endoscopy) should fulfil two major criteria. Firstly, it should be the product of a thorough statistical analysis and secondly, but no less importantly, it has to be biologically plausible.

From a statistical point of view, as Thabut *et al* correctly point out, both platelet count and the platelet count/spleen diameter ratio showed excellent diagnostic accuracy for the non-invasive diagnosis of the presence/absence of oesophageal varices. However, we do not agree with their assumption that the use of the platelet count/spleen diameter ratio did not add significant discrimination to the use of platelet count alone. In fact, the accuracy of the platelet count/spleen diameter ratio for the diagnosis of oesophageal varices was not only better than that of platelet count alone but was also significantly so. Briefly, in the cohort of 145 patients with compensated cirrhosis, which is the group that most likely benefits from screening, the difference between the AUC-ROC of the platelet count/spleen diameter ratio and platelet count was 0.041 (0.013-0.070), with  $p=0.005$  in favour of the platelet count/spleen diameter ratio. Moreover, in the whole cohort of 266 patients, the platelet count/spleen diameter ratio had a c index of 0.902 (95% confidence interval 0.860-0.935) while platelet count alone had a c index of 0.839 (0.790-0.881), with a difference between AUC-ROC of 0.063 (0.038-0.088) ( $p=0.001$ ). Furthermore, the platelet count/spleen diameter ratio was the only parameter significantly associated with the presence/absence of oesophageal varices in a multivariate analysis that also included platelet count. Lastly, as recently highlighted, the negative predictive power of a non-invasive parameter used to predict the absence/presence of oesophageal varices is a fundamental clinical concern.<sup>1</sup> In fact, for such a tool to be adopted in clinical practice it has to achieve a negative predictive value of 100% although maintaining an acceptable positive predictive value. This would preserve the safety of the parameter (that is, virtual absence of missing a diagnosis) and keep a satisfactory cost-efficacy profile. In practice, in our study the use of the platelet count/spleen diameter ratio fulfilled these criteria while platelet count alone did not.

Biological plausibility is a not a secondary concern for the clinician. As we emphasised in our paper and recently demonstrated,<sup>2</sup> and as Thabut *et al* also pointed out, the presence of thrombocytopenia in patients with liver cirrhosis is likely a multifactorial event.<sup>3</sup> Therefore, the use of platelet count to

diagnose a feature that depends on portal hypertension alone may lead to an increase in false positive results, thus decreasing the accuracy as well as the cost-efficacy of the diagnostic procedure. As highlighted in our paper, the use of the platelet count/spleen diameter ratio could bypass this inconvenience by "normalising" platelet count to the platelet count decrease effectively dependent on hypersplenism.

Lastly, some methodological issue should be taken into account when the two items of the ratio are singularly evaluated. On the one hand, the spleen diameter measurement should be performed by a skilled operator, and its results should have excellent accuracy and reproducibility. On the other hand, we have shown that the consistency of the ratio is maintained, even considering the expected mild fluctuations in platelet count commonly seen in cirrhotic patients during a limited period of time.

All in all, we did not presume to propose a diagnostic "magic bullet", as is more and more commonly being proposed in clinical hepatology. We are well aware that the results we obtained have to be validated in independent series and/or in cohorts with different aetiologies of liver disease before being widely accepted,<sup>4</sup> and in our paper we clearly stated the limitations of our study. However, our patient population is that which we commonly encounter in everyday clinical practice, and it is not very different from that seen in other parts of our country (that is, viral cirrhosis in approximately 70% of patients).<sup>5</sup> Moreover, if we look outside our borders, we see that our population is not very different from others, viral aetiology of liver disease being the leading cause of liver transplantation in Europe during the period January 1998 to December 2001 (22 924 cirrhotic patients).<sup>6</sup> Nevertheless, if we examine our data we see that the use of the platelet count/spleen diameter ratio performs equally good in the limited subset of patients with alcoholic cirrhosis ( $n=53$ , platelet count/spleen diameter ratio c index = 0.958, platelet count c index = 0.740, difference between AUC-ROC = 0.218;  $p=0.001$ ), although we feel that focusing on a specific subgroup of patients that does not reflect the true prevalence of the disease in the population would introduce bias.

In conclusion, we have proposed a new evaluation tool and called for validation of our method, being conscious that only differences in opinion that arise from results obtained in well conducted studies contribute to scientific progress, and most importantly that "life is short, and art long; the crisis fleeting; experience perilous, and decision difficult. The physician must not only be prepared to do what is right himself, but also to make the patient, the attendants, and externals cooperate".<sup>7</sup>

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## Computed tomography colonography: colon examination or Pandora's box

We read with interest the article by Ginnerup Pedersen *et al* (*Gut* 2003;**52**:1744–7) investigating the frequency and diagnostic consequences of extracolonic findings at multidetector computed tomography (MDCT) colonography.

The authors noted extracolonic findings in 65% of cases, with the need for further workup in 12% and surgery in 3%. The authors concluded that the high prevalence of extracolonic findings may make MDCT colonography a problematic colorectal cancer screening tool for both ethical and economic reasons.

We would like to comment on the question raised by Ginnerup Pedersen *et al*—namely, whether MDCT colonography should be regarded as a colon examination or a sort of “Pandora's box” (if used for abdominal screening).

Notably, a recent article has emphasised that one of the major potential advantages of MDCT colonography in comparison with all other existing colorectal diagnostic tests is its ability to detect disease outside the colon.<sup>1</sup> Indeed, the possibility that extracolonic disease can be readily identified at CT colonography has been extensively investigated in the literature,<sup>2–4</sup> with results similar to the ones presented by Ginnerup Pedersen *et al*.

However, there is evidence that although the vast majority of extracolonic findings are of little clinical importance, such findings may lead to unnecessary further workup, with obvious economic, medicolegal, and psychological implications.<sup>3</sup> Therefore, the possibility of “looking” outside the colonic lumen can be seen as a “double edged sword” or “Pandora's box”.<sup>3</sup>

In this regard, we feel that three important issues need to be emphasised. Firstly, CT colonography is usually performed with a low dose technique which exploits the high contrast that exists at the colonic mucosa-air interface.<sup>4,5</sup> Such a low dose technique is adequate for evaluation of colorectal lesions but substantially limits the assessment of solid organs.<sup>4,5</sup> In addition, there is a recent trend to reduce even further the radiation dose of CT colonography.<sup>5</sup> For instance, the radiation dose in milliSieverts (mSv) is 10 mSv for standard abdominal CT,<sup>6</sup> 6 mSv in the study of Ginnerup Pedersen *et al*, and 1.8–2.4 mSv in our hospital.<sup>5</sup> Clearly, the lower the radiation dose, the lower the extracolonic diagnostic ability.

Secondly, in order to reduce the cost and increase the safety of the examination, CT colonography is usually performed without administration of intravenous contrast material.<sup>3</sup> Clearly, this reduces even further the ability of CT colonography to detect and characterise extracolonic findings.

Thirdly, and perhaps most importantly, CT colonography has recently been demonstrated to be suitable for colorectal cancer screening purposes.<sup>7</sup> However, there is no agreement regarding the use of standard abdominal CT for general abdominal screening.<sup>8,9</sup> At present, abdominal CT screening is not supported by scientific evidence,<sup>9</sup> United States Food and Drug Administration approval,<sup>6</sup> or the American College of Radiology recommendation.<sup>10</sup>

It is clear that due to the use of a low dose technique and lack of intravenous contrast administration, CT colonography has even lower diagnostic ability than standard abdominal CT for assessment of disease outside the colon. Thus it is of paramount importance that radiologists, referring physicians, and patients are aware that CT colonography is not designed for the detection of extracolonic findings and should therefore be considered primarily as a colon examination. Due to the high prevalence of extracolonic abnormalities, radiologists should be alert to appropriate additional workup for triage patients to avoid opening a potential “Pandora's box”.

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- 10 **American College of Radiology Web site**. Available at: [www.acr.org/publications](http://www.acr.org/publications), Accessed 1 November 2002 (accessible only to ACR members).

## Patients' understanding of colonoscopy risk is suboptimal

We read with interest the British Society of Gastroenterology (BSG) lead audit by Bowles *et al* (*Gut* 53:277–83) into colonoscopy practice within the UK. As part of the audit the authors questioned 1200 patients on their experience of the procedure. Of the respondents, only 81.5% received written information, with only 54.9% recalling information on possible adverse events such as bleeding and perforation. The poor recollection of potential problems is perhaps to be expected if the audit questionnaire was sent to patients sometime after the procedure.

In common with many endoscopy units, we send out an information leaflet with the patient's appointment details. This explains the preparation required, what to expect on the day, and any potential complications, with advice as to what to do should these complications arise. As there are concerns regarding patients' understanding of potential complications related to endoscopy, we designed a short questionnaire to determine how much information patients were able to recall from the information leaflet sent to them prior to colonoscopy. This consisted of four multichoice questions with five possible answers, of which only one was correct. The correct answers were all in the information leaflet. Patients were requested to complete the questionnaire just prior to discharge, at least one hour after procedure completion, thus minimising the effects of sedation. The questions related to: the risk of perforation; the degree of rectal bleeding that required medical assistance; what to do should a problem arise out of office hours; and the correct means of getting home after receiving sedation.

Thirty three patients completed the colonoscopy questionnaire and of these only 37% answered all four questions correctly. Only 52% of patients remembered correctly the perforation rate from diagnostic colonoscopy, which was stated as 1 in a 1000 in our information leaflet. Worryingly, 12 patients (36%) thought that perforation rates were 10–100-fold lower than stated in the information leaflet.

Our study demonstrates that patients fail to fully appreciate the risks of colonoscopy despite the distribution of detailed written information prior to the procedure. This could have medicolegal implications should complications arise and reinforces the need for improved methods of informing patients.

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## CORRECTION

In the paper by Hawkey *et al* (*Gut* 2003;**52**: 820–826), the key relating to figure 2 was incorrectly labelled. The key currently shows “Placebo/ Rofecoxib 500mg/ Naproxen 500mg.” The drug dosage should have read “Placebo/ Rofecoxib 50mg/ Naproxen 500mg.”

## NOTICES

### British Society of Gastroenterology Paul Brown Travel Fellowships

The Paul Brown Travel Fellowships are awarded by the Endoscopy Committee of the BSG. They are intended to assist trainee gastroenterologists and established consultants in visits to units outside the United Kingdom for specialist experience and training in endoscopy.

Specialist registrars who have not achieved their CCST are expected to have the approval of their Postgraduate Dean and their Regional Training Director when they apply for a Travel Fellowship. Applicants are expected to provide confirmation that they have been accepted for training in the unit that they wish to visit.

Successful applicants will be expected to provide a brief written report to the Endoscopy Committee of the outcome of their visit.

Application forms are available from the British Society of Gastroenterology Office, 3 St Andrew's Place, London NW1 4LB. Email: [bsg@mailbox.ulcc.ac.uk](mailto:bsg@mailbox.ulcc.ac.uk)

### European Postgraduate Gastro- surgical School (EPGS) Courses 2004

The EPGS at the Academic Medical Center of the University of Amsterdam will be

holding the following courses during the year: 'Endosonography live in Amsterdam' will be held on 2, 3 & 4 June 2004, and 'Update in Coloproctology' will be held on 28 & 29 October 2004. For further information, please contact: J Goedkoop (tel: (31) 566 3926; fax: (33) 267 5594; e-mail: [j.goedkoop@amc.uva.nl](mailto:j.goedkoop@amc.uva.nl); website: [www.epgs.nl](http://www.epgs.nl)).

### 8<sup>th</sup> Southeast European Symposium of Paediatric Surgery

The 8<sup>th</sup> Southeast European Symposium of Paediatric Surgery will focus upon 'Infectious Problems in Paediatric Surgery.' The event will be held between 24–25 September 2004, at the University of Graz, Austria. For further information, please contact: Professor M E Höllwarth, Department of Paediatric Surgery, Medical University of Graz, Austria, Auenbruggerplatz 34, 8036 Graz; tel: + 43 316 385 3762; fax: tel: + 43 316 385 3775; e-mail: [kinderchirurgie@uni-graz.at](mailto:kinderchirurgie@uni-graz.at).

### 12<sup>th</sup> European Symposium on Neurogastroenterology and Motility

The 12<sup>th</sup> European Symposium on Neurogastroenterology and Motility will be taking place at Robinson College, Cambridge, UK.

The symposium will be taking place on 15–18 September 2004.

On Wednesday 15 September, there will be a postgraduate teaching day. This will cover established and evolving assessments of oesophageal, gastric and intestinal function, visceral sensitivity and brain responses. Basic science techniques including electrophysiology, imaging of gut movements and neural activation will be covered in the afternoon. Finally there will be a session on GI pharmacology covering cytokines, capsaicin and tachykinins.

On Thursday 16 September through to Saturday 18 September midday, the main meeting will be held. This will include symposia, oral free papers and poster rounds. The symposia will be designed to move from basic science to clinical practice and will include sessions on stress and the gut, appetite and obesity, serotonin and inflammation, and inflammation and GI motility. There will also be state of the art lectures and prize presentations.

For registration and further information, please see the website [www.neurogastro.org](http://www.neurogastro.org), and follow links for '12th European Symposium on Neurogastroenterology and Motility.' Please contact the conference organizers at: Confrex, PO Box 21, Rottingdean, East Sussex, BN2 8WZ (tel: +44(0)1273 302200; fax: +44(0)1273 302334; e-mail: [confrex@easynet.co.uk](mailto:confrex@easynet.co.uk)).