LETTERS TO THE EDITOR

Defining treatment resistance in the irritable bowel syndrome

EDITOR,—The irritable bowel syndrome (IBS) is the commonest gastrointestinal disorder encountered by primary care physicians and a number of treatments, both pharmacological and psychological have been advocated. Individual and group psychotherapy, hypnotherapy and behaviour therapy have been shown to produce improvement. Pharmacological agents reported to be effective include antispasmodics, bulking agents, dopamine antagonists, carminatives, opioids, anticholinergics, and antidepressants.¹ Most patients respond to a combination of antispasmodics, bulking agents, a high fibre diet, and explanation coupled with reassurance.²

A comprehensive Medline search for papers on treatment resistant IBS failed to identify a single study which provided a satisfactory definition of this condition. IBS was considered refractory or intractable if patients had "not responded to any therapy",³ "not responded to standard medical treatment"⁴ or had "no improvement from conventional medical treatment".⁵

It seems that the terms refractory/intractable have been used rather loosely and what constitutes standard or conventional medical treatment is not clear. Moreover, if future studies consider IBS to be refractory as soon as pharmacological treatment fails, they would be ignoring the evidence supporting psychological therapies.

It is therefore proposed that IBS be considered "treatment resistant" if:

- symptoms are present for more than 12 months;
- the patient has received adequate explanation and reassurance for his/her symptoms;
- appropriate dietary interventions have occurred, including the institution of a high fibre diet when indicated;
- absence of response to an adequate dose of at least one pharmacological agent tried for a minimum of six weeks;
- absence of response to a minimum of six sessions of psychological treatment.

There is no consensus on what constitutes response, but one would expect global improvement and at least some specific measures to show significant changes. A 50% reduction in the predominant or target symptom (abdominal pain/abdominal distension/abnormal bowel habit) along with a similar improvement in subjective well being could be a clinically useful index of outcome.

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Oesophageal cancer in patients with Barrett's oesophagus

EDITOR,—I read with interest the article by Van der Burgh *et al* (Gut 1996; **39**: 5-8). I am surprised that the authors did not mention the paper by Hassall *et al*,¹ who collected nine cases of adenocarcinoma in patients between 11 and 25 years of age. Hassall *et al* conclude that adenocarcinoma does occur in patients under 25 years of age as a complication of Barrett's oesophagus arising in childhood. They advocate endoscopic surveillance with multiple stepwise biopsies beginning at 10 years of age in children with Barrett's oesophagus with specialised mucosa and goblet cells.

We have published two cases² of adenocarcinoma in childhood developing in Barrett's oesophagus. The first case was an 11 year old boy admitted to hospital for dysphagia caused by a foreign body in the oesophagus. An oesophageal adenocarcinoma arising in Barrett's oesophagus was diagnosed. The second case was a 14 year old boy who had peptic oesophagitis with ulceration; 11 months later an adenocarcinoma of the oesophagus was diagnosed. Adenocarcinoma arising in Barrett's oesophagus has been recognised in young adults and starts earlier in life than the usual squamous cell carcinoma, but in our review of the literature we found very few examples of oesophageal cancer in children. We emphasise the need for long term follow up of such children with a history of oesophageal abnormalities, even after treatment of reflux, because the peptic oesophagitis scar remains, particularly when peptic stenosis has been treated with dilatation and antireflux surgery.

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Is gastric surfactant related to lung surfactant?

EDITOR,—In his leading article Hills reviews important concepts about a "hydrophobic mucosal barrier" as the main biological basis for protecting the stomach against acid mediated damage to the mucosa (Gut 1996; **39**: 621-4). While we support the author's view that lipid rather than mucus makes the gastric surface less vulnerable to protons, the proposed analogy of this "hydrophobic mucosal barrier" with lung surfactant is supported by little direct evidence. Hills claims that the key molecule of lung surfactant, dipalmitoyl

phosphatidylcholine (DPPC), is also the main factor of gastric surfactant, based on indirect measurements some 20 years ago.¹

We have recently re-examined this central tenant of the gastric surfactant theory, and have conclusively demonstrated the virtually complete absence of DPPC in gastric mucus lipid. In these studies^{2,3} we extracted the phospholipids from gastric tissue and mucus with chloroform:methanol and determined the compositions of both phospholipid classes and phosphatidylcholine molecular species with two recently developed HPLC techniques, using post-column formation of fluorescent mixed micelles in the presence of 1,6-diphenyl-1,3,5-hexatriene for phospholipid quantitation.^{4,5} While comparative analysis of lung tissue and lavage fluid revealed the presence of large amounts of DPPC in lung tissue and lung lavage fluid,⁶ we did not find significant amounts of DPPC in either gastric mucosa or mucus.^{2,3} Instead, palmitoyloleoyl phosphatidylcholine (POPC) and palmitoylinoyleoyl phosphatidylcholine (PLPC) were abundant in gastric mucosa and were selectively increased in gastric mucus. These results were confirmed recently using electrospray ionisation mass spectrometric analysis of lipid extracts from gastric samples (unpublished data).

Identification of the effective absence of DPPC in gastric mucus has far reaching consequences for our understanding of the mechanism underlying the protection of the gastric mucosal surface by hydrophobic components, in many respects simplifying the concepts involved. The function of DPPC in pulmonary surfactant is dependent on its high gel to sol phase transition temperature (41°C) and consequently, DPPC is a solid material at body temperature. Pulmonary surfactant contains specialised unsaturated phosphatidylglycerols and specific apoprotein components designed to facilitate adsorption of DPPC to the air-liquid interface.⁷ These constraints will not apply to gastric surfactant, as this is composed of the more fluid molecules POPC and PLPC. This conjecture is in agreement with our finding that negligible amounts of phosphatidylglycerol were present in gastric mucus.² Therefore, although we support Hills' concepts of a phospholipid mediated hydrophobic barrier as the main factor in gastric mucosal protection, we disagree that this barrier is biochemically related to lung surfactant.

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Reply

EDITOR,—In responding to the letter by Bernhard and Postle it is important to emphasise agreement on the major issue that surface-active phospholipid (SAPL) protects the stomach wall from autodigestion, providing the gastric mucosal barrier to *hydrated* protons $H(H_2O)^+$. Protons are far too polarising to exist alone in an aqueous environment, the water of hydration being repelled by any hydrophobic domain. In my leading article great care was taken to use the term SAPL in all except the introductory remarks explaining arousal of my interest in mucosal protection on the basis of molecular similarities between cationic corrosion inhibitors and the pseudocationic dipalmitoyl phosphatidylcholine (DPPC) which, I thought at the time, might have reached the stomach by swallowing lung surfactant. Hence, it is incorrect to say that I claim DPPC to be “the main factor of gastric surfactant”. Bernhard and Postle are also incorrect in stating that we based our model on the analyses made some 20 years ago by Wassef *et al*,¹ when we published our own analyses of surface scrapings taken along the whole gastrointestinal tract,² displaying a range of phospholipid species not unlike that found in the lung—predominantly phosphatidylcholine (PC). Unfortunately, we did not have access to HPLC for fatty acid analysis at that time (15 years ago), and so I am quite prepared to accept their elegant and much later analyses displaying partially unsaturated PC. However, even unsaturated phospholipids display much surface activity,³ although their molecules may not pack together quite as neatly as their disaturated counterparts, as depicted in my model for barrier formation. The findings of Bernhard and Postle are consistent with our unpublished findings that, in rats, unsaturated exogenous PC offers comparable mucosal protection to saturated PC. Their point about the sol-gel transition of DPPC is only relevant to the lung where there is a fluid-air interface and even this is open to debate, but such controversies are better left to respiratory texts.⁴ The real point to remember is that it is the surface activity of the phospholipids, particularly the PCs, which permit binding of the surfactant to the tissue surface, enabling it to impart its highly desirable properties whether protection against corrosion in the stomach, lubrication in the joint,⁵ or any of a number of others based upon principles well established in the physical sciences.⁶

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Are Down syndrome and coeliac disease associated?

EDITOR,—The paper by Gale *et al* (*Gut* 1997; **40**: 492–6) fails in its aim to investigate the association between coeliac disease and Down syndrome. The authors use methodology for screening that they have stated is outdated. In a vulnerable population this is ethically unsound and inaccurate conclusions are drawn from incomplete data.

The increased frequency of raised antigliadin levels (and of autoimmune antibodies) in individuals with Down syndrome are already well documented, as is the increased prevalence of coeliac disease.^{1,2} Incidence cannot be assessed by screening.

Serological screening by antigliadin antibodies alone is outdated and the usefulness of antiendomysial antibody levels, which can be easily measured using a commercial kit or human umbilical cord, is well established.³ To compound this error, the authors only regard individuals who have elevated titres of both IgA and IgG as being “gluten antibody” positive. The report does not exclude IgA deficiency in the eight controls positive for IgG alone. By the authors' own admission, antigliadin IgA is more specific for coeliac disease, so individuals with positive IgA but negative IgG should be considered potentially coeliac and investigated further, with antiendomysial levels or by biopsy.

The failure to biopsy the “gluten antibody” positive controls prevents any conclusions about the prevalence of coeliac disease and the relevance of haematological or biochemical parameters in patients with Down syndrome compared with other institutionalised cohorts. The low serum albumin concentrations may reflect poor diet and feeding problems in an institutionalised setting and the control population values are not given for comparison. It is disappointing that no attempt was made to report the grading of the abnormal biopsy samples and the exclusion of giardia infection in this high risk group, purely on absence from a single duodenal biopsy specimen, is inadequate. Lymphocytic infiltration of the lamina propria can be considered to be “latent coeliac disease” but these changes may not resolve on a gluten free diet as was suggested. Formal gluten challenge, longer term follow up on a gluten containing diet with clinical and serological monitoring or further investigation are required to confirm the diagnosis.⁴ A diagnosis of coeliac disease has considerable implications and the failure of the authors to discuss EPSGAN criteria is unfortunate.

The importance of the genetic predisposition to coeliac disease is under emphasised and even dismissed. Monozygotic twin concordance approaches 100% with longer follow up of non-affected twins⁵ and dizygotic

twin concordance is 25%, which is highly indicative of a genetic aetiology. HLA class II DQ2 molecules may well be involved in presentation of the toxic peptide(s) but segregation analyses suggest that at least one other non-HLA linked gene confers genetic susceptibility.⁶ Genome screening has already detected possible loci of interest, including one on chromosome 21.⁷

The estimate of increased prevalence uses as a comparison an observational report which calculated an incidence 10-fold lower than expected.⁸ Data from Australia are rare but the population is genetically similar to northern Europe and diet is also similar, so a broadly comparable prevalence of 0.1–2.93/1000^{9,10} would be expected. A calculated increased risk of 10-fold for individuals with Down syndrome is in keeping with published data.^{1,2}

Research into the immune dysfunction found in Down syndrome and searches for genetic linkage may be more informative.

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Reply

EDITOR,—We thank Morris *et al* for their interest in our article and would like to clarify the points they have raised. Our research commenced in 1992 when only antigliadin antibody testing was available to us. Our study was prompted by a desire to clarify the link between Down syndrome and coeliac disease already reported in numerous anecdotal and inadequate reports. More recent reports documenting this link more strongly appeared after our research was completed. Likewise, at the time of our study an elevated level of both IgA and IgG antibodies was the accepted criterion for biopsy.¹ Despite this

lapse in time between the study and its submission for publication, we felt it was important to proceed because of the continued widespread use of anti-gliadin antibodies and the importance of reporting their shortcomings.

We did exclude IgA deficiency in the eight controls who were IgG positive only. We did not biopsy the two gluten antibody positive controls as the decision to do this was not ours; moreover, the increases were barely above cut off values for IgA in both and for IgG in one.

The lowered serum albumin concentrations in the Down syndrome group are unlikely to be due to diet. These patients did not have feeding difficulties and low serum albumin is uncommon in this setting, even in a more vulnerable group who do have severe feeding difficulties.² In any case, dietary factors cannot explain a difference in albumin concentrations between those with negative and those with positive antibodies.

As far as giardia is concerned, the routine was to take multiple (three) biopsy specimens per examination and each specimen was closely examined. We did under emphasise the genetic aspects of coeliac disease, but this was motivated by our feeling that these were accepted well enough to not require comment, rather than a dismissal of them.

Concerning EPSGAN criteria, in an institutional setting with severely disabled patients and multiple caregivers, compliance with the diet cannot be guaranteed. This makes it very difficult to draw conclusions about the correctness of the diagnosis. While these difficulties need not preclude adherence to EPSGAN criteria, they render interpretation of subsequent biopsy samples difficult.

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Helisal saliva assay

EDITOR.—We read with interest the papers by Reilly *et al* (*Gut* 1997; **40**: 454-8) and Christie *et al* (*Gut* 1996; **39**: 27-30) focusing on the Helisal saliva assay (Cortecs Diagnostics, UK) for the diagnosis of *Helicobacter pylori* infection. These papers reported that this test has a sensitivity of 84-88% and a specificity of 70-71%.

Firstly, in both studies the inter- and intra-assay coefficients of variation were not measured. This may well affect the reproducibility of a test. In fact, Fallone *et al.*¹ using the Helisal assay, reported a sensitivity of 66%, a specificity of 74% and a coefficient of variation of 16±12%. Simor *et al* reported a sensitivity of 81% and a specificity of 75%, but the reproducibility of the Helisal assay results was not evaluated.²

Secondly, it is worth noting that in the study by Christie *et al* the age range of the reference population is not given. As Reilly *et*

al found in their series, the Helisal saliva assay performs differently according to age (that is, 91.7% sensitivity and 35.3% specificity in the subgroup of patients under 50 years of age). Furthermore, Christie *et al* suggest that the Helisal saliva assay may be useful in reaching a diagnosis in children but it is conceivable that, as with *H. pylori* serology,³ salivary antibody testing for *H. pylori* needs to be validated separately in adults and children.

Thirdly, Reilly *et al* comment that the Helisal saliva assay they used, when tested clinically by Lahaie *et al.*⁴ has a sensitivity of 97% and a specificity of 90%. However, in the latter study, published as an abstract, the figures refer to serum and not to saliva: 93% sensitivity and 85% specificity are the correct figures. Furthermore, these authors evaluated a relatively small number of patients, used a ¹⁴C-urea breath test as the absolute reference, and did not give sufficient methodological details.

Finally, we believe that salivary *H. pylori* testing may play an important role in selecting candidates for vaccination programmes.⁵ However, optimal validation of any diagnostic salivary kit for *H. pylori* is warranted in order to assess reproducibility and whether it can be applied generally to both adults and children.⁶⁻⁸

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BOOK REVIEWS

Helicobacter pylori. Harris AW, Misiewicz JJ. (Pp 66; illustrated; £9.95.) London: Blackwell Healthcare Communications, 1996. ISBN 0-86542-639-2.

Is there anyone left who would like more information on *Helicobacter pylori*, or has

saturation point been reached? According to my local virtual bookstore (amazon.com), there are currently 22 book titles available dealing specifically with this germ, in addition to thousands of research papers and hundreds of reviews and editorials in the specialist and general literature. So, why do we need another book, especially when the guidelines for treatment seem to change every few months? For those who are sceptical (as I was initially), let me persuade you that in this small paperback of 66 pages Drs Harris and Misiewicz have served a very useful role in educating UK general practitioners on the management of their patients.

This user-friendly book slips into a white-coat pocket easily and could be read in an evening. It contains all the essential information, from epidemiology and pathophysiology through to who and how to treat. It is well, but not overexcessively referenced, has useful summary sections (which could easily be the basis for MRCP-type MCQs), and offers guidelines without dogma. The latter is the book's strength, but also its weakness, because by making (mostly correct) generalisations about the treatment of *H. pylori*, the authors run the risk of not being sufficiently specific. For example, a variety of eradication regimens are listed, but no best bet is recommended. Thus, the authors will certainly not offend any researcher or pharmaceutical company, but this generosity may be less than helpful for the busy practitioner, who needs to know what to write on the prescription pad.

The other problems with any treatment guidelines, especially for *H. pylori*, are that the goalposts move, and that *H. pylori* may have different implications in different populations. Who would have anticipated, writing in 1996, that up to 25% of duodenal ulcers in the United States may be unrelated to *H. pylori*, or that both the 1997 International Update Conference and the 1997 "Maas-tricht" consensus would recommend offering treatment to all patients found to have a positive test? Thus, although representing the "state of the art" when written in 1996, the book already seems a little dated.

So, as much of the information in this primer has a short half-life, we can anticipate a second edition. I would like to see some more appealing figures (the colour photos especially, which take us back to the bad old days of fiberoptic endoscopy) and a more basic introductory chapter as the current chapter one simply summarises treatment guidelines. As this book is written very much for the home market, it is much less useful to those in other countries, particularly when the relative costs of alternate clinical strategies may be wildly different.

However, despite these caveats, this book will be useful to those still struggling to define the relevance of *H. pylori* in their clinical practice. This should by now include gastroenterologists and junior doctors, and in addition to the target audience of GPs, may encompass general medicine physicians and surgeons. Finally, it will undoubtedly be of interest to the scientifically literate patient, especially if they still prefer to read books than to download from cyberspace.

S MOSS

Baillière's Clinical Gastroenterology. Volume 10. Number 4. Gastrointestinal Endocrine Tumours. O'Shea D, Bloom SR. (Pp 211; illustrated; £30.00.) London: Baillière Tindall, 1996. ISBN 0-7020-2186-5.

This book consists of a series of chapters on each of the major gut endocrine tumour syndromes and four general chapters on the pathology, the peptide receptors possessed by these tumours, and medical and surgical treatment. The emphasis of the book is clinical, and all of the chapters are written by authors who have a large personal experience of these rare tumours.

A chapter on carcinoid tumours discusses the characteristics of the various different tumours that arise from the different sites in the gastrointestinal tract. The carcinoid syndrome is briefly discussed, as is prognosis. In this chapter there is virtually no discussion of treatment modalities. The chapter on gastrinoma details the diagnosis and clinical features and the unique problems of tumour localisation in this syndrome. In contrast to the previous chapter, one third of this chapter is devoted to treatment, including a discussion of the place of acid antisecretory therapy, surgery, chemotherapy, somatostatin analogues, hepatic embolisation, and even hepatic transplantation. A section on insulinoma is provided by the Mayo Clinic group. Unlike the other gut endocrine tumours, most insulinomas are benign. The implications of this for both medical and surgical therapy are fully analysed. The experience with VIP producing tumours, both of the gastrointestinal tract and of the neuroblastomas that occur in children, is described by the Ohio group, and the Hammersmith Hospital experience with the glucagonoma syndrome, is described by Drs Frankston and Bloom. Surprisingly, in a book like this, no account is given of the very rare gut endocrine tumours, including GHRH producing tumours, Cushing's syndrome, hypercalcaemia producing tumours, somatostatinomas, or the non-functioning endocrine tumours. They are, however, mentioned briefly in the chapter on surgical treatment.

The chapter on pathology by Drs Bishop and Polak is the clearest account I have ever read of the significance and limitations of the various immunochemistry techniques used to identify these tumour types. The chapter on peptide receptors on gut endocrine tumours is almost exclusively about the clinical relevance of somatostatin receptors for tumour localisation and therapy with octreotide. The chapters on medical and surgical therapy are, in some instances, the only source of information about a syndrome, but in other cases the information is redundant and, on occasion, potentially confusing. For example, in the surgical chapter, less emphasis on gastrinoma and insulinoma would have been appropriate, and in the medical chapter, more information on treatment of carcinoids, and a more critical discussion of the limitations of chemotherapy studies would have been useful.

At whom is this book directed? The emphasis is clinical, so presumably it is directed at clinicians. These tumours are rare and most clinicians when they see such a patient, are likely to turn to what they hope is a definitive account of diagnosis, management and prognosis. Have there been major advances in this field in the past few years that justify a new monograph? Most of these gut endocrine syndromes were described by the 1970s, and the role of chemotherapy and hepatic embolisation has changed little in recent years. Octreotide did change medical therapy in the 1980s and tumour imaging in the 1990s, and surgical therapy has become more refined, particularly in gastrinoma.

Does this monograph provide better information than is available in standard textbooks? In some cases that is certainly true, but in others it is questionable, and the chapters vary widely in the references provided.

If you do not possess a large standard textbook and need an account of gut endocrine tumours, this monograph is excellent in many ways, but may not provide you with all you want. For the rest of us, this is one to look at in the library.

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NOTES

Sir Francis Avery Jones BSG Research Award 1998

Applications are invited by the Education Committee of the British Society of Gastroenterology who will recommend to Council the recipient of the 1998 Award. Applications (TWENTY COPIES) should include:

- A manuscript (2 A4 pages ONLY) describing the work conducted
- A bibliography of relevant personal publications
- An outline of the proposed content of the lecture, including title
- A written statement confirming that all or a substantial part of the work has been personally conducted in the UK or Eire.

Entrants must be 40 years or less on 31 December 1997 but need not be a member of the BSG. The recipient will be required to deliver a 40 minute lecture at the Annual Meeting of the Society in March 1998. Applications (TWENTY COPIES) should be made to the Honorary Secretary, BSG, 3 St Andrews Place, London NW1 4LB, by 1 December 1997.

International Workshop on Variceal Haemorrhage

The International Workshop on Variceal Haemorrhage will be held in Hong Kong on 1–2 December 1997. Further information from: Professor Joseph Sung, Endoscopy Centre, Prince of Wales Hospital, The Chinese University of Hong Kong, Hong Kong. Tel: +852 2632 2233; Fax: +852 2635 0075.

12th International Workshop on Therapeutic Endoscopy

The 12th International Workshop on Therapeutic Endoscopy will be held in Hong Kong, from 2 to 4 December 1997. Further information from: Professor Sydney Chung, Endoscopy Centre, Prince of Wales Hospital, The Chinese University of Hong Kong, Hong Kong. Tel: +852 2632 2233; Fax: +852 2635 0075.

Second International Conference on Therapies for Viral Hepatitis

The Second International Conference on Therapies for Viral Hepatitis will be held in Kona, Big Island, Hawaii, from 15 to 19 December 1997. Further information from: Dora Moya, International Medical Press, 3112 E. Shadowlawn Ave., Atlanta, GA

30305, USA. Tel: 800 868 2022 or 404 233 0261; Fax: 404 233 2827; Web site: www.intmedpress.com/hawaii/.

Course in Postgraduate Gastroenterology

A Course in Postgraduate Gastroenterology will be held in Oxford, UK, on 4–7 January 1998. This course has been designed for consultants and registrars, including those who do not specialise in gastroenterology. Topics will include:

- Liver disease
- Colonic neoplasia
- Acute pancreatitis
- Osteoporosis, arthritis and GI disease
- Food allergy and intolerance.

Course fee £200 (\$330). Board and accommodation are available at Wadham College at extra cost. Six bursaries will be available for applicants training in gastroenterology or in research posts at British hospitals. Further information from: Dr DP Jewell, Gastroenterology Unit, Radcliffe Infirmary, Woodstock Road, Oxford OX2 6HE.

Colorectal Disease in 1998

The 9th Annual Colorectal Disease in 1998: An International Exchange of Medical and Surgical Concepts will be held at Marriott's Harbor Beach Resort, Fort Lauderdale, Florida, USA, from 19 to 21 February 1998. Further information from: Cleveland Clinic Florida, Department of Education, 2950 West Cypress Creek Road, Fort Lauderdale, FL 33309-1743, USA. Fax: 954 978 5539; Other: 800 359 5101, ext 5056; Local/international: 954 978 5056; email: jagels@cesmtp.ccf.org.

6th Southeast European Congress of Paediatric Surgery: Short Bowel Syndrome

The 6th Southeast European Congress of Paediatric Surgery: Short Bowel Syndrome will be held in Graz, Austria, on 22–23 May 1998. Further information from: Dr Günther Schimpl, Department of Paediatric Surgery, Auenbruggerplatz 34, A-8036 LKH-Graz, Austria. Tel: +43 316 385 3762; Fax: +43 316 385 3775.

9th International Symposium on Cells of the Hepatic Sinusoid

The 9th International Symposium on Cells of the Hepatic Sinusoid will be held in Christchurch, New Zealand, from 27 September to 1 October 1998. Further information from: Professor Robin Fraser, I.S.C.H.S., Christchurch School of Medicine, PO Box 4345, Christchurch 8001, New Zealand. Tel: +64 3 3640 587; Fax: +64 3 3640 593; email: grogers@chmcs.ac.nz.

Advanced Course in Gastroenterology

An Advanced Course in Gastroenterology will be held at the Royal College of Physicians of Edinburgh, UK, from 3 to 7 November 1998. Further information from: Miss Lee Ross, Symposium Assistant, Education, Audit and Research Department, Royal College of Physicians of Edinburgh, 9 Queen Street, Edinburgh EH2 1JQ, UK. Tel: +44 131 225 7324; Fax: +44 131 220 4393.