LETTERS TO THE EDITOR

Coeliac disease and birth defects in offspring

EDITOR,—I read with interest the paper by Martinelli et al (Gut 2000;46:332–335) and letter by Unsworth et al (Gut 2000;47:598) which stress the high prevalence of coeliac disease in anemic pregnant women. Untreated coeliac disease can adversely affect the reproductive system and result in infertility, multiple abortions, low birth weight babies, and short breast feeding periods.1 A low plasma level of folic acid is a common finding in newly diagnosed patients: there are good theoretical reasons for hypothesising that coeliac disease could also be a maternal risk factor for birth defects.2 3 There are coeliac women identified in the study of Martinelli et al that a further pregnancy and reached term. One of them had a boy with a major cardiac malformation; she was the only one on a gluten containing diet.

I examined sera from 40 mothers of children with non-syndromic spina bifida, 37 mothers of children with non-syndromic cleft lip with or without cleft palate, and 24 mothers of children with isolated cleft palate. I found that 1/40 and 1/37 women were positive for IgA class antiendomysium antibody (AEA). Jejunal biopsy of the mother of a child with left lip and palate showed normal villous height/ crypt depth ratio and increased number of intraepithelial lymphocytes; jejunal biopsy of the mother of a child with spina bifida was normal. Both AEA positive women were two of the four smallest (<155 cm) among all investigated mothers (n=101). Coeliac patients have acquired the reputation of being short in stature but tall patients do exist.4

Frequent reports of increased reproduction problems in women with untreated coeliac disease emphasise the importance of early and correct diagnosis followed by adherence to a gluten free diet. Health care professionals need to recognize the manifestations of the disease in women of reproductive age and be prepared to use serological screening tests more frequently to identify patients who could benefit from dietetic treatment.

I suggest that coeliac disease should be considered as a cause of birth defects associated with folic acid deficiency (for example, spina bifida, orofacial clefts, heart defects)5 in offspring of women of short stature.

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Reply

EDITOR,—Women affected by untreated coeliac have indeed a considerable risk of having low birth weight babies and unfavourable outcome of pregnancy. Very recently in Sweden it was suggested that offspring of coeliac fathers are also at risk of low birth weight.1 2 The mean birth weight of offspring of mothers and fathers affected by other autoimmune diseases. The authors of the large population based Swedish study suggest that there are “coeliac” families. The presence of an incentive for categorisation of mother and fathers with untreated CD is very likely to gradually change the estimate of the population prevalence of mother and fathers affected by other autoimmune diseases. The expectation rate among a cohort of women (or without an unfavourable outcome of pregnancy), their values would be well within the 95% confidence interval of a rate of 1%. We reported an increase in the risk of 1:70 among a general population of pregnant women which would be compatible with the number reported by Hozyasz. The increasing sensitivity of the screening tests now available for coeliac disease (antiendomysium and anti-transglutaminase) is likely to gradually change the estimate of the population prevalence of the disease, but also, repetition of screening over time increases considerably the rate of identification of previously negative cases.

I personally feel that we have little evidence that coeliac disease should be considered “a cause of birth defects associated with folic acid deficiency...” but I do agree that doctors and obstetricians should widely screen for coeliac disease as they do (often overdo) for many quite rare diseases.

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H pylori and functional dyspepsia

EDITOR,—I read with interest the results reported by Blum et al (Gut 2000;47:473–80) and the editorial which accompanied this article by McColl (Gut 2000;47:461–4). Professor McColl argues that the greater response to acid suppression observed in the Helicobacter pylori positive compared with the negative subjects in the Blum trial resulted from a lack of blinding of the investigators and the presence of an incentive for categorising the pylori positive and negative responders. In support of this argument, he cites pooled data from two large randomised controlled trials (BOND and OPERA)1 comparing omeprazole 20 mg, 10 mg, and placebo where overall no significant difference in relapse rate was observed. However, there may be other explanations for the results observed by Blum et al.

We have previously reported in pooled data from the BOND and OPERA studies that in the H pylori positive group, 43% on omeprazole 20 mg had complete relief of dyspeptic symptoms (by intention to treat) compared with 35% in H pylori negative group (34% and 24% on placebo, respectively).1 Hence there was a small therapeutic gain observed in H pylori positive patients receiving omeprazole 20 mg versus those who were H pylori negative although the differences were not statistically significant. In the pooled data from the BOND and OPERA studies, there was also an association between symptom relief and the presence of H pylori subgroups based on the predominant symptom.1 Moreover, there were differences observed between H pylori infected and uninfected cases, albeit non-significant. Therefore, ulcer-like dyspepsia is a valid approach, it is conceivable that the H pylori positive for example, had complete symptom relief on omeprazole 20 mg in 47% of cases versus 31% on placebo. On the other hand, in those who were H pylori positive with ulcer-like dyspepsia, 35% had complete symptom relief on omeprazole 20 mg versus 24% on placebo. In contrast, no benefit of omeprazole versus placebo was identified in dysmotility-like dyspepsia. Dyspepsia subgroups were not considered in the Blum study. Presumably the decision to conduct statistical analyses separately in H pylori positive and negative patients was exploratory in the Blum trial, as randomisation was not performed stratifying for H pylori status. Although using a valid approach, it is conceivable that the distribution of potential responders to acid suppression in the H pylori positive and negative arms (for example, ulcer-like dyspepsia) was not dissimilar.

Professor McColl suggests that the same therapeutic gain achieved with potent acid suppression may be achieved more permanently by eradication of H pylori. However, this conclusion remains controversial. Moayyed et al recently published a meta-analysis

of 12 trials reporting that the risk reduction with *H. pylori* eradication therapy from the pooled data was 9% (95% confidence interval (CI) 4–14%). However, these results are in conflict with a similarly high quality meta-analysis of 10 trials which failed to detect a significant benefit of *H. pylori* eradication (odds ratio for success 1.3, 95% CI 0.89–1.189). The positive and negative trial results in non-ulcer dyspepsia may be reconcileable if *H. pylori* eradication only benefits those with hidden peptic ulcer disease or those predisposed to subsequently developing peptic ulceration which in turn is likely to vary across different populations. If this hypothesis is correct, then eradication therapy in a subset of patients with non-ulcer dyspepsia would be indicated (as the benefits should then outweigh the risks). In the absence of predictors which usefully identify a subset who will achieve symptom relief with eradication therapy, the decision to treat the infection is by necessity on a case by case basis.

**Figure 1** This figure is derived from Professor Talley’s original paper to which he refers, comparing the effects of omeprazole 20 mg versus placebo in *Helicobacter pylori* positive and negative subjects with functional dyspepsia. It shows that the therapeutic gain from omeprazole is similar or less in *H pylori* positive versus negative patients. Contrary to Talley’s comments regarding the symptomatic benefit from omeprazole in functional dyspepsia, there are only two recent high quality meta-analyses of *H pylori* eradication in functional dyspepsia published as full papers. These both showed unequivocal benefit of the active treatment over placebo. The other meta-analysis Professor Talley refers to has only appeared in abstract form and it is therefore difficult to comment on its quality. In addition, it did not include the most recent studies. None the less, even this study showed a strong trend in favour of eradication therapy over placebo (odds ratio for success 1.2, 95% CI 0.98–1.6). Hence all of the studies and meta-analyses indicate that eradicating *H pylori* infection is superior to placebo in non-ulcer dyspepsia, producing a therapeutic gain of about 10%. Although the magnitude of the benefit may not be large, it is equivalent to the only other treatment which is effective for this common disorder, namely proton pump inhibitor therapy. Therefore, eradication treatment however has the advantage of producing this benefit by a one off course of treatment rather than requiring long term maintenance therapy.

**Reply**

**Editor,** I am very confused by Professor Talley’s comments regarding the symptomatic benefit from omeprazole in *Helicobacter pylori* positive versus negative subjects with functional dyspepsia. In his original paper, he states in the results section that "remission of dyspepsia in the active treatment arms was not significantly different in the *H pylori* infected and uninfected patients" and in the abstract that "symptom relief was similar in *H pylori* positive and negative cases". I was therefore very surprised to read Professor Talley’s letter in which referring to the same paper he states that "there was a small therapeutic gain observed in *H pylori* positive versus negative patients". He goes on to state that "in the *H pylori* positive group, 43% on omeprazole 20 mg had complete resolution of dyspepsia symptoms compared with 35% in *H pylori* negative patients". However, therapeutic gain compares the benefit of the active treatment versus the placebo treatment. The therapeutic gain in the *H pylori* positive group on omeprazole is 9% and the therapeutic gain in the *H pylori* negative group is 11% (fig 1). Therefore, contrary to the comments made in his above letter, Professor Talley’s original study showed that the therapeutic gain for omeprazole in *H pylori* positive versus negative patients with functional dyspepsia.

**Conflict of interest:** Professor Talley is a consultant for Astra Zeneca, Janssen, TAP, Pharmacia, Lederle, and GlaxoSmithKline.


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**The future of ERCP training in the UK**

**Editor,** We read with great interest the leading article by Helliwell and Morris (Gut 2000;47:459–60) regarding the future of endoscopic retrograde cholangiopancreatography (ERCP) training in the UK. They discuss the importance of restricting training in ERCP to some trainees and training only in centres that are accredited for training by the Joint Advisory Group (JAG) in Endoscopy. JAG, at the request of the Conference of Royal Colleges, produced endoscopy training guidelines. For ERCP training, these state that a centre should be performing a minimum of 250 procedures per annum (with specialist centres performing 500 per annum). They should also have regular meetings to discuss radiological findings with a radiologist with special interest in ERCP. While we certainly agree that ensuring that performance of ERCPs and several trainees learning to determine the likely impact of the JAG recommendations on ERCP training and whether trainees were likely to have an individual exposure to procedures in larger units, we performed a questionnaire based study, which was mailed to all acute UK hospitals. A total of 178 of 292 units replied (61%) to a single mail-shot questionnaire, of whom 151 (84.8%) were performing ERCPs. Forty teaching hospitals (THs 75.5%) and 74 district general hospitals (DGHs 61.2%) offered ERCPs per annum. The service was mainly consultant based (92.1% of lists being consultant led). Gastroenterologists (68.0%), surgeons (29.2%), and radiologists (2.8%) performed ERCPs. The number of procedures performed per annum in DGHs was 40–800 (mean 228) and in THs 60–2000 (mean 336), 57.2% of which were therapeutic. Of those performing more than 250 procedures per annum, 82.1% of DGHs and 77.3% of THs held regular radiology meetings to discuss findings. Of those performing less than 250 procedures per annum, 72.9% of DGHs and 75.0% of THs held radiology meetings.

Each centre trained up to six trainees per annum, most training one or two. The average number of procedures which trainees could perform per annum was 176 (48–431) in DGHs and 144 (range 54–311) in THs. This was dependent more on the number of trainees per centre than on the type of hospital in which the training was performed with...
Before performing the procedure independently. Nevertheless, the general message is accepted that we must look at quality rather than quantity.

Although it is clear that good opportunities do exist for gaining training in units undertaking smaller numbers of procedures, it is difficult to define a precise number that takes into account the needs of the individual trainee in getting the required experience and the numbers required for the trainer to maintain competence.

In the new JAG document, which we hope to publish in the next month, the emphasis has moved away from numbers to other means of assessing competence. We are still of the opinion that those units with lower throughput should combine with larger units to provide training as part of a regionally organised training scheme. Trainees are already organised regionally and decisions as to who will undertake ERCP training will be made in consultation with the regional training director. Several hospitals have already applied for, and been granted, ERCP training status on the basis of a combined training unit. A total of 74 units have now registered with JAG, of whom 115 have been approved for ERCP training.

The new JAG document makes it clear that trainers should undergo training on endoscopy specifications. The first six have already been piloted by the Raven Department of Education at the RCS. In future, it will be essential for re-accreditation of units that trainers will have gone on such courses.

Reviewer—We read with interest the letter and enclosed copy of a survey on endoscopic retrograde cholangiopancreatography (ERCP) training. We, like the authors, have been concerned since the initial Joint Advisory Group (JAG) documentation that a large extent of competence is judged by numbers rather than other measures of endoscopy skills. Indeed, reliance on numbers has been the cause of most of the correspondence that JAG has received following the publication of the initial training document. However, at that time and to a large extent at present, there are few satisfactory measures of competence to replace numbers and time. The authors state in their letter “that only centres that are accredited for training by JAG will be permitted to continue endoscopy training”. Strictly speaking this is not true. Lack of recognition of a unit for training would mean that any training that takes place in that unit would not be recognised by the statutory bodies but this would not prohibit the units from training. They also state that “the guidelines base competence for training in ERCP solely on the number of procedures carried out in the individual centres”. The training does actually require a little more than numbers, for example the training document states that “endoscopists cannot be considered competent in ERCP until they are able to c四个方面 drainage. Trainees should carry out at least 100 procedures under supervision and be achieving a high percentage of success

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An accessible practical text for doctors and senior health workers at first referral centres in developing countries is indeed a worth-while objective and this WHO sponsored guide largely succeeds. An impressive list of contributors combine to produce a 160 page manual to some of the more common paediatric presentations which maintains throughout that these children can be managed successfully with a combination of good clinical assessment, basic diagnostic facilities, and a core of essential drugs.

Designed to complement the Integrated Management of Childhood Illness guidelines, it has the feel of a classic textbook with many simple black and white drawings and tables which are also refreshingly new, given over as they are to demonstrations of practical clinical assessment and diagnostic procedures. There is even an appendix of toys for severely malnourished children. There is also an excellent chapter on supportive care, and solutions to feeding problems and nutrition is an important theme throughout. Monitoring of children's progress and an encouragement to audit outcomes are worked in as is the pragmatic approach to human immunodeficiency virus. By virtue of the fact that it is designed with brevity and clarity in mind, it is very didactic however. The reader is not invited to question why for instance “very severe pneumonia” is treated with chloramphenicol, “severe pneumonia” with penicillin, and “pneumonia (non-severe)” with streptomycin. More leeway is given in the advice to monitor local antibiotic sensitivity patterns in treating Shigella and Salmonella infections. Many may appreciate this approach but some will not.

The text on the management of the sick child in a developing country should perhaps be best reviewed by a doctor in a developing country. I agree, but in my own mitigation I might add that I have been lucky enough to have worked for several years in Bangladesh and have sent this copy onto a good home—the hospital library of the paediatric hospital where I was based. This book deserves a wide distribution and a cover price of Sw Fr 10.50 will help.

C. DOHERTY


The book contains 138 short chapters on almost all practical issues in the management of patients with inflammatory bowel disease (IBD). It is the second edition of a book published in 1989 entitled Current Management of Inflammatory Bowel Disease. Obviously, in the last 12 years since the publication of the first edition, much has changed both in the medical and surgical therapeutic approaches to IBD. All of the current and most up to date attitudes are well reflected in this book.

The format of the book is very convenient and helpful to every physician, especially gastroenterologists who treat IBD patients and are frequently confronted with a specific problem or question related to their management and treatment. The 138 chapters cover practically every issue related to the management of ulcerative colitis and Crohn’s disease. Readers will find up to date application of diagnostic tools such as computed tomography and magnetic resonance imaging to the handling of IBD patients. A significant number of chapters are dedicated to the medical treatment not only of straightforward IBD but also its complications, such as pouchitis, severe diarrhoea, the need to speak of the management of extraintestinal manifestations. Thirty two chapters deal with the surgical aspects of ulcerative colitis and Crohn’s. These chapters provide gastroenterologists with in depth information regarding surgical options, their advantages, and disadvantages. Even though the chapters that relate to surgical intervention of IBD have been written by experienced surgeons, they are easily and clearly understood by medical people, who are usually those who refer patients for surgery. Reading these pertinent chapters will enable the medical man to better understand what is to be expected from the intervention, its details, and place in the therapeutic approach to the treatment of IBD.

The book contains all of the necessary and most current information about the novel medical treatment of IBD. A special chapter deals with anticytokine therapy and another with novel manipulation of the inflammatory mediator pathways. Another feature that characterises this book is inclusion of a rather unusual number of chapters on the humanistic aspects of the management of chronic IBD and patient-physician interactions.

Most of the chapters include a short list of additional references and a brief comment by the editor calling attention to other views and highlighting other areas of potential interest. Because of the broad spectrum of the book and because the 138 chapters have been written by different authors, repetitiveness is unavoidable and many issues are dealt with more than once. In spite of this drawback, the book is an excellent source of information, practical as well as basic. It will be helpful for clinicians who handle IBD who are looking for a comprehensive resource, containing pertinent information on almost every problem that may arise in the management of patients with ulcerative colitis and Crohn’s disease.

D. RACHMILEWITZ


Should general pathologists attempt to interpret liver biopsy specimens at all? I enjoy the great luxury of being able to spend my working day exploring aspects of pancreatic and hepatobiliary pathology almost exclusively, and the more I study the liver the surer I am that doing justice to a liver biopsy specimen is a tough job.

But that job has been made much easier now. The new version (sixth edition) of Scheuer and Lefkowith's Liver Biopsy Interpretation is a superlative guide for those venturing into liver work. It should be required reading for trainees not only in anatomical pathology but also in hepatology and in surgery of the liver and biliary tree, whom it will assist immeasurably in understanding how the biopsy diagnoses are made that are used to monitor their patients’ clinical courses.

The reader is taken by the hand through what to do—and what not to do—in organising all aspects of biopsy planning and handling. The authors move from discussions that should precede tissue sampling in unusual or complicated cases through the advantages and disadvantages of processing, sectioning, and staining techniques; they discuss clinical pathological entities, issues in differential diagnosis, and approaches to be taken in clarifying problems, and even give advice on how to structure to its users’ best profit the report of findings. The text is buttressed by well selected references that neglect neither classic work nor the most recent advances, with attention to molecular genetics and cellular physiology in our understanding of disease as faced in the clinical and laboratory. The figures that the authors provide illustrate their points admirably, and the increased use of colour in this new edition is a delight.

The authors ride their hobby horses now and again, and theirs are not mine (I have never understood why sections stained specifically for elastic tissue, or to point up type I collagen, or with the periodic acid-Schiff technique in the absence of preliminary diastase digestion are so often among those referred for consultative opinion). Nor do I have much use for aspiration cytology of solid rather than cystic liver lesions which the authors now give half a chapter. If you have a needle in the liver, I say take a true biopsy specimen for a more certain diagnosis.

But, quibbles aside, I know of no better English language liver biopsy handbook than this one. It should be part of the microscope-side library of any pathologist whose practice involves the liver. There it will help him or her not only to do good work on his or her own, but to raise higher expectations because as consultant internists and surgeons familiarise themselves with this book, the standard of practice of liver histopathology that they demand, as better informed consumers, will necessarily rise.

A. KINSELLY


Hirschsprung’s disease, or congenital intestinal aganglionosis, was named after Carl Hirschsprung, a Danish paediatrician who gave the first description of the condition in 1886. At first the massive dilatation of the colon was believed to be caused by an abnormality within the dilated colon itself and the condition was therefore called “congenital megacolon”. Sir Frederick Treves (1898) performed the first curative operation. While operating on a six year old girl he noted that although there was massive dilatation of the proximal colon, the lower sigmoid colon and rectum were narrow. He performed an abdominoperineal resection of the distal colon and rectum with anastomosis of the proximal colon to the anal margin. The
chapters discuss the reported complications consistent with the artwork. Further mention could have been improved by a more comprehensive description of the six most examples of histochemical techniques. Enhanced by inclusion of colour illustrations described in association with Hirschsprung's disease; and techniques and results of surgical intervention have a hesitation in recommending it as a very useful reference book for surgeons and gastroenterologists, both in their research work and clinical practice.

E R HOWARD

NOTES

Sir Francis Avery Jones British Society of Gastroenterology Research Award 2002

Applications are invited by the Education Committee of the British Society of Gastroenterology who will recommend to Council the recipient of the 2002 Award. Applications (TEN 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 2001 but need not be a member of the Society. The recipient will be required to deliver a 30 minute lecture at the Annual meeting of the Society in Birmingham in March 2002. Applications (TEN COPIES) should be made to the Honorary Secretary, British Society of Gastroenterology, 3 St Andrews Place, London NW1 4LB by 1 December 2001.

Hopkins Endoscopy Prize 2002

Applications are invited by the Endoscopy Committee of the British Society of Gastroenterology who will recommend to Council the recipient of the 2002 Award. Applications (TEN 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.

The recipient will be required to deliver a 20 minute lecture at the Annual meeting of the Society in Glasgow in March 2002. Applications (TEN COPIES) should be made to the Honorary Secretary, British Society of Gastroenterology, 3 St Andrews Place, London NW1 4LB by 1 December 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: Falk Foundation e.V., Congress Division, Leinenweberstr. 5, PO Box 6529, D-79041 Freiburg, Germany. Tel: +49 7651 15 14 0; fax: +49 7651 15 14 395; email: symposia@falkfoundation.de

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

50th Anniversary of the First Right Hepatectomy: From Resection to Donation

This event will be held on 14–15 December 2001 in Paris, France. Further information: Michèle Centonze Conseil, 6 bis rue des Cendriers, 7020 Paris, France. Tel: +33 1 44 62 68 80; fax: +33 1 43 49 68 58; email: mail@m-centonze-conseil.com; website: www.m-centonze-conseil.com

14th Intensive European Course of Digestive Endoscopy

This course will be held on 17–18 December 2001 in Strasbourg, France. Further information: Michele Centonze Conseil, 6 bis rue des Cendriers, 75020 Paris, France. Tel: +33 1 44 62 68 80; fax: +33 1 43 49 68 58; email: mail@m-centonze-conseil.com

GI Malignancies Can be Prevented and Treated: from the Bench to the Bedside

This international meeting will be held on 15–20 January 2002 at the Dead Sea, Israel. Further information: Secretariat, GI Malignancies, PO Box 29041, Tel Aviv 61290, Israel. Tel: +972 3 5175150; fax: +972 3 5175155; email: gis@targetconf.com