a significant inpatient death rate (32% in Whitehead's series and 19% in our own).

E H Forrest, J A H Forrest
Department of Gastroenterology, Victoria Infirmary, Langside Rd, Glasgow G42 9TY, UK

Correspondence to: E H Forrest; Ewan.Forrest@gvic.scot.nhs.uk

Authors' reply

We thank Drs E and J Forrest for the interest they have shown in our article on jaundice and we were pleased to learn of their retrospective assessment of 100 cases of jaundice presenting to acute services in a large Glasgow hospital. Although they emphasised the differences between their experience and ours, this is the nature of medical correspondence and we were more struck by the similarities which we found gratifying. The series cannot be compared too closely because of differences in methodology and case ascertainment. In particular, our study was prospective, community and hospital based, and included all patients with bilirubin values greater than 120 µmol/l. Forrest and Forrest's observations are retrospective, relate specifically to patients presenting to hospital because of jaundice, and use a cut off bilirubin level of >60 µmol/l.

We will respond to their comments seriatim.

(1) The commonest cause of presentation with jaundice to Stobhill Hospital was alcoholic liver disease. In Swansea, if analysis is restricted to those 95 patient *presenting* to hospital with jaundice, then alcoholic cirrhosis ran a very close second to malignancy as the commonest cause.

(2) As Forrest and Forrest point out, sepsis/shock is not a common cause of jaundice *requiring admission* to hospital either in Glasgow or Swansea, but in our experience was the predominant cause of jaundice *developing while in hospital for other reasons*. As to whether it is overlooked, our results speak for themselves—in over one third of our sepsis/shock cases jaundice had been erroneously attributed to some other cause by the clinical team managing the case.

(3) Ten of 29 (34%) Glasgow cases and 16 of 61 (26%) Swansea cases with common bile duct (CBD) stones had bilirubin levels >120 µmol/l. Given the relatively small sample sizes we consider these to be similar rather than dissimilar proportions. The absolute values of bilirubin from the two centres cannot be compared without knowledge of the timing of samples. Clearly, samples taken on admission might show lower bilirubin levels than samples taken later on, particularly with malignant biliary obstruction awaiting mechanical relief. Our experience is that gall stone biliary obstruction was often transient and not profound whereas malignant obstruction led to ever increasing levels of bilirubin unless there was mechanical intervention.

(4) We share Forrest and Forrest's concern about the accuracy of diagnosis on retrospective case note review but respectfully point out that our study was prospective while theirs was retrospective. We accept that not every patient in the Swansea series had every investigation but we cannot consider it good practice to perform tests unless clinically indicated. Thus most patients with proven obstructive jaundice did not have serological tests whereas most patients with intrinsic hepatocyte dysfunction did.

(5) Our observations on aspartate aminotransferase (AST):bilirubin ratios were for interest alone. We did not propose that this should be used as a test but simply commented that the ratio had some diagnostic value. Our only comment on the Glasgow figures relates to their patients with alcoholic liver disease where the ratio was reported to be 3.5. Mean bilirubin level for this group was 142 µmol/l which translates to a mean AST value of approximately 500 IU/l. This is an exceptionally high figure for AST in alcoholic liver disease where AST is characteristically much lower, usually <200 IU/l.

(6) Causes of jaundice and causes of jaundice requiring hospital admission are not the same and clinicians should guard against using the experience of one clinical setting when assessing another.

J G C Kingham, M W Whitehead
Department of Gastroenterology, Singleton Hospital, Sketty, Swansea SA2 8QA, UK

I Hainsworth
Department of Pathology, Morriston Hospital, Swansea SA6 6NL, UK

Correspondence to: Dr J G C Kingham; jkingham@swansea-tr.wales.nhs.uk

Behaviour of Crohn's disease according to the Vienna classification

I hasten to congratulate Louis *et al* on their meticulous and insightful study on the stability of Crohn's disease phenotypes according to the Vienna classification (*Gut* 2001;**49**:777–82). It was particularly gratifying to learn from them (in a separate communication) of the remarkably high degree of interobserver agreement in classifying patients by this system.

The principal message that the authors draw from their study is that the initial "behavioural" classification of B1 (non-stricturing non-penetrating) at the onset of Crohn's disease hardly ever remains stable over the lifetimes of the patient but almost invariably progresses in time to either B2 (stricturing) or B3 (penetrating) disease. Naturally, this finding hardly comes as a surprise either to the authors of the Vienna classification[1] or in fact to any clinician caring for patients with Crohn's disease. More important and revealing, in my opinion, is the observation by Louis *et al* that "the proportion of initially B2 patients changing from B2 to B3 was [only] 15.4% (only 2/13 patients)".

Therefore, once "inflammatory" (B1) disease has made its almost invariable progression to either B2 or B3, why should we not be able to incorporate this relatively stable "choice" of pathway into a phenotyping system suitable for genotypic correlations?

D B Sachar
Division of Gastroenterology, Mount Sinai School of Medicine, New York, New York, USA; david.sachar@mssm.edu

References

1 **Gasche C**, Scholmerich J, Brynskov J, *et al*. A simple classification of Crohn's disease: report of the Working Party for the World Congresses of Gastroenterology, Vienna 1998. *Inflamm Bowel Dis* 2000;**6**:8–15.

Authors' reply

We thank Professor Sachar for his kind comments on our work. As it has become obvious that Crohn's disease is a multifactorial polygenic heterogeneous entity, apart from molecular genetic studies a major task is now to identify stable phenotypes of Crohn's disease that may correspond to particular genetic backgrounds. The propensity of Crohn's disease to develop as a stricturing or as a penetrating disease (Crohn's disease behaviour) has been considered for some time as a potential suitable phenotype for genetic correlations. However, results to date have been inconclusive. Several explanations are plausible: (a) there is no major genetic influence on Crohn's disease behaviour and the significant concordance within multiply affected families is essentially due to shared environmental factors; (b) the genes involved have not yet been tested and it is true that only a small number of candidate genes have been tested in this setting; and (c) patients with Crohn's disease have not been classified adequately into subphenotypes, and it is true that several classifications have been proposed and that the application of these various classifications does not result in homogeneous categories.

In relation to the first two hypotheses, progress in the understanding of the physiology and biology of strictures and fistulas as well as of the influence of environmental factors, including smoking and medical treatment of the disease, is needed. Regarding the third point, the classification used necessarily must result in stable categories of patients. As we have shown, even the most recent and reproducible classification[1] is not suitable as patients change categories over time. As emphasised by Sachar, it seems from our data that patients who are classified as B2 (stricturing) tend to remain B2 over time. This is mainly true for patients who are already B2 at diagnosis as 88% remained B2 over a median follow up of seven years (range 1–30 years). It seems as if patients who develop penetrating lesions (B3) associated with stricturing lesions tend to develop these simultaneously and thus are directly classified as B3 while patients who develop clinically significant stricturing disease without concurrent penetrating lesions do not tend to develop such lesions afterwards. Furthermore, in our population, only a few pure stricturing lesions (B2) developed after 10 years of evolution. Therefore, in our experience, patients who develop a pure stricturing disease over 10 years of evolution seem to represent a homogeneous phenotype that may be suitable for studies of genetic factors potentially involved in stricture development. However, this does not seem to be the case for penetrating disease (B3). In our patients, penetrating phenotypes continued to develop at a constant rate (approximately 25% of patients/five years), even after 20 years of evolution, mainly directly from the non-penetrating non-stricturing phenotype (B1). Therefore, the subgroup of patients with non-penetrating non-stricturing disease can never be considered as homogeneous as even after 25 years some may evolve to the penetrating phenotype (B3). Furthermore, a patient who develops penetrating lesions within two years of evolution may be biologically and genetically very different from a patient who develops such lesions after 25 years. To some extent this point can also be applied to the stricturing phenotype (B2).

An alternative would be to take into account the speed of development of the B2 or B3 phenotype. Indeed, the inclination to develop such a phenotype is most probably multifactorial. We would be surprised if a

unique gene were responsible for stricture development for example. Therefore, if a gene is involved it may be rather by facilitating or by speeding up the development of these phenotypes, together with other genes and environmental factors. In this hypothesis we may have more chance to disclose predisposing genes when comparing patients who have rapidly developed stricturing or penetrating phenotypes (within five years for example) with other patients. We believe that when performing genotype-phenotype correlations for Crohn's disease behaviour, several classification options have to be tested according to these various hypotheses of gene implication. Furthermore, we should aim towards disclosing environmental factors and stratify patients according to these factors or to consider these factors in multivariate analyses.

E Louis, J Belaiche
Department of Gastroenterology, CHU of Liège, Belgium

Correspondence to: E Louis; edouard.louis@ulg.ac.be

References

1 **Gasche C**, Scholmerich J, Brynskov J, *et al.* A simple classification of Crohn's disease: report of the Working Party of the World Congresses of Gastroenterology, Vienna 1998. *Inflamm Bowel Dis* 2000;**6**:8–15.

BOOK REVIEWS

Pediatric Gastroenterology and Nutrition in Clinical Practice

Edited by C H Lifschitz. New York: Marcel Dekker, 2002, B/W, pp 869. ISBN 0 8247 0510 6

"Of the making of many books there is no end and much study is a weariness of the flesh." We spend too much time reading—or rather we are expected to take in vast volumes of information from text. Not just the written word in books but from journals and more and more directly from the screen. Few of us have time to sit down to read systematically, and most of us scan contents pages, chapter titles, and abstracts. We take in "new knowledge" more by accident than design, and all forms of the written word compete with each other.

Books have a historical advantage over what we still regard as more ephemeral sources of information—journals and the Internet. Books are portable and we like to think that the effort that goes into writing them is a measure of the quality and authority of their contents. But how confident can we be that this is the case?

Peer review has become the test of quality of original articles, and we take most notice of papers published in journals that are most rigorous in this respect. Books on the other hand rely for their credibility on the reputation of their authors. Things are not so clear when it comes to new multiauthor compilations, such as *Pediatric Gastroenterology and Nutrition in Clinical Practice*. Collecting together and publishing papers and reviews from international conferences must be commercially profitable for some publishers, and worthwhile for many authors, even though the price of such books is often extraordinary. This book is not the result of a meeting but brings together chapters from a variety of eminent paediatric gastroenterologists from around the world. Its editor intends it to present a "clear and useful summary of the most relevant new facts in molecular biology and genetics, as well as recently acquired information, in conjunction with a practical approach to pediatric gastroenterology and nutrition".

At first sight the book has no structure, containing 33 chapters with titles as diverse as "New knowledge about protein" and "Microorganisms administered for the benefit of the host" (sounds like a good way to poison your enemies at the Christmas party), alongside more familiar titles such as "Short bowel syndromes", "Celiac disease" and "Food allergy". It seems to fall somewhere between a textbook and a multiauthor collection; it not suitable for undergraduates and it is not the book to reach for when faced with a difficult clinical problem. Its layout and contents assume a basic understanding of the subject, and a familiarity with areas that are topical. It is most likely to be of value to specialists in paediatric gastroenterology and nutrition who wish to keep up to date.

At 854 pages, assuming a reading speed of a page per minute, this book represents 14.2 hours of CPD. In a perfect world I should read it before I pass judgement. Even though I am keen to clock up maximum CPD points, I admit that I have not read this book from cover to cover. However, I would not go as far as Sydney Smith, cleric and wit, who confessed that "he never read a book before reviewing it; it prejudices a man so!"

L T Weaver

Gastrointestinal Polyps

Edited by N Haboubi, K Geboes, N Shepherd, *et al.* Greenwich: Medical Media, 2002, £65.00, colour, pp170. ISBN 1-90015-121-9

I suspect that to the vast majority of gastrointestinal histopathologists, and probably to general histopathologists and endoscopists too, the idea of a book devoted solely to gastrointestinal polyps is appealing. After all, most endoscopists see such lesions every day and most pathologists will see at least one a week. Often a verdict of "hyperplastic" or "inflammatory" polyp is the best that can be offered but this diagnosis is not very satisfying for pathologist and clinician alike. Consequently, it was with eager anticipation and in the hope of transforming my approach to gastrointestinal polyps that I started to read this book.

As luck would have it, the slides for the EQA in gastrointestinal pathology had landed on my desk the previous day. They included at least two difficult polypoid lesions for which a diagnosis was currently eluding me. I thought that this book would be an ideal reference and turned to it for help. I was pleasantly surprised when the answer to my conundrum was available within minutes. A little while later I was approached by one of my SHOs with a question on the genetics of juvenile polyposis. After a short consultation of the book, I was able to give the answer confidently; no need for Internet searches this time.

This book is the first to my knowledge that deals solely with gastrointestinal polyps. It covers all regions of the gastrointestinal tract and is abundantly illustrated with endoscopic photographs and colour photomicrographs. For each type of polyp, descriptions of prevalence, endoscopic appearances, and pathological features are given, followed by discussion of biological behaviour and associated conditions. For some types of polyp, details of management strategies are also provided.

All of the authors are well known gastrointestinal pathologists with a wealth of experience in this field, so it is not surprising that they have managed to put together such a comprehensive text. I could not think of any entities they had omitted, and there were several that I had never heard of. Overall, the presentation of this book is of a high quality; the text is succinct but readable and, apart from a few exceptions, the illustrations are excellent.

This is primarily a diagnostic book and if it does have a defect it is in the descriptions of molecular biology and therapeutic approaches, which inevitably lack the detail that some purists would desire. This aside, the book will undoubtedly appeal to histopathologists and endoscopists alike, not only for the diagnostic details it provides, but also for the associated clinicopathological information. I have found it an ideal companion and am sure that others will think the same.

P Domizio

Upper Gastrointestinal Surgery, 2nd Edn

Edited by S M Griffin, S A Raimes. Philadelphia: WB Saunders, 2001, B/W, pp 474. ISBN 0 7020 2587 9

The *Companion to Specialist Surgical Practice* series aims to meet the need of higher surgical trainees and busy practising surgeons by keeping them up to date of recent developments in the field and consolidating our understanding on key topics. The first series of seven texts met with high critical acclaim and in the second edition of the series this has been expanded to eight volumes. The second edition of *Gastrointestinal Surgery* comprehensively covers the field of hollow organ upper gastrointestinal surgery. There are some minor omissions such as impedance assessment and management of gastric polyps. This however is only a minor criticism of what is otherwise an excellent text. The book occupies an important niche in the field of surgery as each volume is produced in a short period of time in order to ensure that it is up to date, in contrast with some of the larger texts in the field which by virtue of the time it takes to produce a new edition are already somewhat out of date at the time of publication.

The new edition benefits from an emphasis on evidence based practice with up to date key references, some of which include a short commentary. Unfortunately, there is a degree of non-uniformity among chapters, which would benefit from correction in the next edition.

The main contributors are all established figures in the field of upper gastrointestinal surgery and bring an authoritative viewpoint to each chapter. The format is pleasing with