a significant imipenem patent death rate (32%) in whitehead's series compared to 7% (4 of 61) in Svansoe's series.

E H Forrest, J A H Forrest
Department of Gastroenterology, Victoria Infirmary, Longside Rd, Glasgow G21 9TL, UK
Correspondence to: E H Forrest
E-mail: jor@forrest.arts.sci.gla.ac.uk

Authors' reply

We thank Drs E and J Forrest for the interest they have shown in our article on jaundice and we are 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 authors have overestimated the differences in methodology and case ascertainment. In particular, our study was prospective, community-based 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 separately.

(1) The commonest cause of presentation with jaundice to Stobhill Hospital was alcoholic liver disease. In Swansea, if analysis is restricted to those 95 patients 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, scyphoskias is not a common cause of jaundice and we have never seen this in our hospital, either in Glasgow or Swansea. However, in our experience this 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 accidentally attributed to some other cause by the clinical team managing the case.

(3) Forrest and Forrest's figures (cases 16 and 17 of 61) (26%) of Swansea cases with common bile duct (CBD) stones had bilirubin levels >120 μmol/l. Given the relatively small sample size 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 a specific 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 where theirs was retrospective. We accept that not every patient in the Swansea series was examined by every investigation but we cannot consider it good practice to perform tests unless clinically indicated. Thus the most patients with proven obstructive jaundice did not have serological tests whereas most patients with intrinsic hepato-pancreatic dysfunction did.

(5) Our observations on asymptomatic anicteric (ASYA) bilirubin tasters were not of much 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 figure 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
Correspondence to: J G C Kingham
E-mail: jkingham@swansea.wales.nhs.uk

I Mainwaring
Department of Pathology, Morriston Hospital, Swansea SA6 6LY, UK
Correspondence to: I Mainwaring
E-mail: mainwaring@swansea.wales.nhs.uk

Behaviour of Crohn's disease according to the Vienna classification

I hasten to congratulate Lewis et al on their meticulous and insightful study on the stabili

1 behaviour 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-secreting 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 (secreting) or B3 (penetrating) disease. Naturally, this finding hardly comes as a surprise either to the authors of the Vienna classification or in fact to any clinician caring for patients with Crohn's disease. More important and revealing, in my opinion, is the observation by Lewis et al that "the proportion of initially B1 patients changing from B2 to B3 was [only] 15.4% (5/31 patients)".

Therefore, once "inflammatory" (B1) disease has made its almost inevitable 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 genetic correlations?

D B Sachar
Division of Gastroenterology, Mount Sinai School of Medicine, New York, New York, USA
E-mail: dsachar@mssm.edu

References


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 disease with no single molecular studies a major task is to identify stable phenotypes of Crohn's disease that may correspond to specific genetic backgrounds. The propensity of Crohn's disease to develop as a penetrating disease (Crohn's disease behaviour) has been considered for some time as a potential suitable phenotype. Despite correlations, however, results to date have been inconclusive. Several explanations are possible: (1) the genes involved have not yet been tested and it is true that only a small number of candidates have been tested in this setting; and (2) 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 two hypotheses, progress in the understanding of the physiology and biology of structures and fistulas as well as 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 obvious and reproducible classification is not suitable as patients change categories over time. As emphasised by Sachar, it comes from our data that patients who are classified as B2 (secreting) 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 period of 10 years. However, it seems as if patients who develop penetrating lesions (B3) associated with structuring lesions tend to develop these simultaneously and thus are directly classified as B3 while patients who develop clinically significant structuring disease without concurrent penetrating lesions do not tend to develop such lesions afterwards. Furthermore, in our population B2 patients have developed after 10 years of evolution. Therefore, in our experience, patients who develop a pure structuring disease over 10 years of evolution seem to represent a homogenous phenotype that may be suitable for studies of genetic factors potentially involved in structuring 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/year), even after 20 years of evolution, mainly directly from the non-penetrating non-structuring phenotype (B1). Therefore, the subgroup of patients with non-penetrating non-structuring 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 structuring phenotype (B2). An alternative would be to take into account the speed of development of B2 or B3 phenotype, indeed, the inclination to develop such a phenotype is most probably multifactorial. We would be surprised if a
unique genes were responsible for stricture development for example. Therefore, if a gene is involved it may be rather by facilitating or by specifying the development of these phenotypes, together with other genes and environmental factors. In this hypothesis we may have more chance to display predicted genes when comparing patients who have rapidly developed structuring or persisting 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 hypothesis 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 Liege, Belgium

Correspondence to: E Louis; e-mail: louis@ulg.ac.be

References

BOOK REVIEWS

Pediatric Gastroenterology and Nutrition in Clinical Practice

“Of the making of many books there is no end and much study is a weariness of the flesh.” We read 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 pride in the practice of peer review. But we take pride of practice of papers published in journals that are most rigorous in this respect. Books on the other hand rely on the 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 financially 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 35 chapters with titles diverse as “New knowledge about proteins” and “Microorganisms administered for the benefit of the host” sounds like a good way to poison your enemies at the Christmas party, along-side more familiar titles such as “Short bowel syndrome”, “Celiac disease” and “Food al- lergy”. It seems to fall somewhere between a textbook and a multi-authored collection; 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 want 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 check 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 com-fessed “he never read a book before reviewing it: it prejudices a man so!”

Gastrointestinal Polyps

I suspect that to the vast majority of gastrointestinal biologists and probably to a number of gastroenterologists 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 veritable “hyperplasia” or “inflammatory” polyp is the best that can be offered but this diagnosis is not very satisfying for pathologists and clinicians 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 SROs 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 easy to the 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 book is a textbook 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 endoscopic and histological approaches, which inevitably lack the detail that some purists would desire. This aside, the book undoubtedly appeals to histopathologists and endoscopists alike, not only for the diagnostic details it provides, but also for the diagnostic and clinical correlations within. I believe that it has an ideal companion and am sure that others will think the same.

LT Weaver

Gastrointestinal Surgery, 2nd Edn

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 with 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 this 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 second 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