currently using balloon dilatation as opposed to bougies, in the hope that shearing stress on the fragile mucosa is reduced,² but repeat dilatation after either technique seems to be equally common. Thirdly, we feel that oesophageal replacement should be avoided, owing to the particular problems of surgical and anaesthetic management.³ Strictures do not always recur after dilatation and none of our 258 patients has needed oesophageal surgery. Lastly, it is worth noting that an 'inverse' form of dystrophic EB exists, probably with recessive inheritance, in which oropharyngeal and oesophageal involvement may be severe, but skin lesions relatively mild.⁴

The management of such difficult problems requires a multidisciplinary approach, with cooperation between dermatologists, gastroenterologists, ENT surgeons, plastic surgeons, anaesthetists, radiologists, and dietitians.

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Limitations of faecal chymotrypsin as a screening test

SIR,—We read with interest the paper by Riedel *et al* (*Gut* 1991; 32: 321-4) on the levels of chymotrypsin in the stools of South African patients with chronic pancreatitis and apparently healthy people. There are some points that we would like to make. Firstly, we wonder if the faecal chymotrypsin test should be regarded as a screening test for chronic pancreatitis. This is in part due to the characteristics of the disease: chronic pancreatitis is rare enough not to make a diagnosis in the asymptomatic phase practical; moreover, we have no proof that early treatment can modify its course. On the other hand, the faecal chymotrypsin assay itself is not a good screening test, since it is consistently abnormal only in patients with advanced exocrine impairment. We, for example, found normal levels in only four of 31 patients with exocrine insufficiency, but in 13 of 44 of the patients with chronic pancreatitis.¹ In our opinion, therefore, the test should be considered as a test for pancreatic insufficiency only. This is not simply a semantic problem. It implies (i) that the control subjects should be patients with chronic pancreatitis without exocrine impairment, rather than asymptomatic subjects from the general population; (ii) that we should choose the cut off point that best distinguishes between

patients with and without insufficiency (malabsorption or severely impaired secretin-pancreozymin test) rather than between asymptomatic control subjects and patients with pancreatitis. We wonder, therefore, what was the specificity of the test for pancreatic insufficiency considering this lower cut off point.

We found the relation between faecal chymotrypsin and stool pH interesting. The correlation was found only in the control subjects. If we have correctly interpreted the data, the chronic pancreatitis patients were studied while on a hospital diet, whereas all the controls were outpatients. The large fibre intake in controls might have induced a large faecal bulk with possible diluting effects on faecal chymotrypsin. All the references in the paper arguing against a diluting effect by faecal bulk on faecal chymotrypsin dealt with the effects of fat malabsorption in Western patients with chronic pancreatitis on a low fibre diet,^{2,3} which represents a different condition. Moreover, different time periods are likely to have elapsed between the bowel movements and stool pH measurement in controls (one morning) and in chronic pancreatitis patients. When we measured stool pH immediately and one and six hours after the bowel movement, a progressive reduction in pH was found in five of six chronic pancreatitis patients (from mean (SEM) 7.03 (0.18) to 6.85 (0.11) to 6.7 (0.12)). We wonder, therefore, if both the low pH and chymotrypsin values in some controls may represent hydrolysis by intestinal bacteria for the longer time elapsed in a warm and sunny country. Again, this would argue against the use of the assay as a screening test in the general population, but not in inpatients.

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Reply

SIR,—We welcome the opportunity to reply to the letter from Drs Cavallini and Benini. Certainly, faecal chymotrypsin is a test to assess exocrine pancreatic function. The diagnostic value of the test for screening for chronic pancreatitis by showing exocrine pancreatic insufficiency has been suggested by others¹ and was not the aim of our study. It has been clearly shown that cases of mild or early disease are often missed.² Most patients with chronic pancreatitis presenting to our unit have advanced disease with exocrine insufficiency. In these patients faecal chymotrypsin determination is a valuable screening test provided faecal pH is taken into consideration. The suggestion that control subjects should be patients with chronic pancreatitis but without insufficiency is of interest but was not the

objective of our study. We emphasise that the question posed was the effect of faecal pH on faecal chymotrypsin.

It is correct that we found a correlation between faecal chymotrypsin and stool pH only in control subjects. Patients with chronic pancreatitis and control subjects were not on a hospital diet. They were studied as outpatients on free living diets. It should be noted that the dietary fibre consumption of urban and rural blacks has decreased. In fact in urban blacks dietary fibre consumption is much lower than in Western populations.³ This makes it unlikely that faecal bulk was an important factor. There was no time lapse between bowel movement and stool pH measurement in control subjects and patients. In all subjects stool was put on ice and pH was measured within six hours. Samples were then deep frozen and faecal chymotrypsin was measured within 10 days. Faecal chymotrypsin activity has been shown to be very stable over several days at room temperature.

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BOOK REVIEWS

Progress in hepatic, biliary and pancreatic surgery. By J S Najarian and J P Delaney. (Pp 172; illustrated; £72.) London: Year Book Medical Publishers, 1991.

The basis of this annual book, with over 60 contributors, is the continuation course in surgery at the University of Minnesota. Its purpose is to provide an update on the specific but fairly broad areas of general surgery. The course from which this book was generated, dealt with the liver, biliary tract, and pancreas. Despite the title, several of the chapters are more medical in orientation but the book does not lose value from that. For example, the chapter by Dame Sheila Sherlock on 'Viral hepatitis and the surgeon' is a model of its kind. The authors have brought together a galaxy of surgical talent, including Henri Bismuth, Martin Adson, Babs Moossa, Seymour Schwartz, Frank Moody, and Ben Eiseman.

Books such as this are essentially ephemeral, but with the rapid pace of development in this field, even encyclopaedic tomes are usually of only temporary interest. The particular value of this book is that all the contributions are brief, written attractively, and well illustrated.

Topics of essentially practical interest have been selected for succinct description and discussion. By avoiding the encyclopaedic approach, controversial topics have been highlighted.

Who, therefore, would find the book of interest? Obviously the working surgeon with a broad knowledge of the field would encounter certain topics often neglected by even the largest encyclopaedia. The trainee, preparing for postgraduate examinations, will find the surgery of this field recast in a way different from the conventional textbook. An interesting and stimulating read, easy to take up and put down, at £72 this book is not a good buy for the individual and at best would be recommended for a library which is well funded and already possesses the classic text monographs in this field.

ROBERT SHIELDS

Gastrointestinal physiology (4th ed). Edited by L R Johnson. (Pp 176; illustrated; £16.) St Louis: Mosby Year Book, 1991.

The book is aimed primarily at medical students. It is concise and readable, with different aspects of gastrointestinal physiology divided into easily digestible chapters. The formula is a tried and tested one, with separate chapters dealing with motor and secretory function on a regional basis. In addition, there are two chapters on regulation by hormones and nerves and a final chapter on the splanchnic circulation. The book is well illustrated with simple line drawings, flow diagrams, and tables. Although the artwork is basic in nature, this is probably an advantage for conceptual understanding and suits the needs of the target readership. In terms of depth of coverage there is little more than one finds in some general medical physiology textbooks. In its favour, however, it has as its source material *Physiology of the gastrointestinal tract* (1987: New York, Raven Press) also edited by L R Johnson. This has ensured that the book is in touch with present concepts. Clinical material is used to illustrate physiological concepts and most chapters contain a section on clinical tests. The contents and direction have therefore been well chosen for the medical student but the book lacks depth for the more serious gastrointestinal physiologist.

D GRUNDY

Non-responders in gastroenterology: causes and treatment. Edited by G Dobrilla, K D Bardhan, and A Steele. (Pp 222; illustrated; IL 90 000.) Verona: Cortina International, 1991.

The authors are to be congratulated on their choice of the original topic of non-responders in gastroenterology. Unfortunately, presumably because 'the Editors decided not to add to their burden with strict rules regarding the size and structure of their articles', the experts selected, many of whom are 'long-standing personal friends', have mostly ignored their brief. They have instead given a textbook review of the topic with little on the size of the problem, appropriate further investigations, management plans to bring the patient into remission, and evidence of trials of the efficacy of such treatment, all of which the reader has the right to expect.

Moreover the pace of advances in our understanding of pathophysiology, pathogenesis, and pharmacology of gastrointestinal disease is so fast as to make some of the chapters outdated even as they appear in print. Thus, omeprazole has only 30 lines in 16 pages on refractory reflux oesophagitis, and only two of 10 pages on H₂ antagonist resistant ZE syndrome. Cisapride has one citation in 10 pages on idiopathic constipation. The editors' 39 page chapter on duodenal ulcer devotes nine lines to *Helicobacter pylori*. No recommendations are made for the investigation (such as serum gastrin and acid studies) of postsurgical recurrent ulcers.

Honourable exceptions are S Domschke's resistant pancreatic steatorrhoea, Howdle's algorithm for coeliac non-responders, and Maydeo and Soehandra, who confidently 'conclude there is no patient with oesophageal varices who is recalcitrant to . . . aggressive . . . sclerotherapy . . . and the use of . . . Histoacryl-Blau' (n-butyl 2 cyanoacrylate). The chapters follow in the usual caudad direction from burning mouth to solitary rectal ulcer via the liver (ascites, itching) and pancreas. Perhaps the wisest words in this book are by Tygstrup: Most studies 'are casuistic rather than therapeutic trials . . . physicians try many different types of treatment, and it is not surprising that the capriciousness of the reactions occasionally leads to a small series of favourable results, which is then published'. Because of this 'publicity bias', 'unless a completely effective and safe therapy becomes available, there is no alternative to randomised clinical trials . . . to quantitate the problem of non-responders, to decide if they have special clinical characteristics, and to select combinations for optimal treatment of non-responders'.

J H BARON

All titles reviewed here are available from the BMJ Bookshop, PO Box 295, London WC1H 9TE. Prices include postage in the UK and for members of the British Forces Overseas, but overseas customers should add £2 per item for postage and packing. Payment can be made by cheque in sterling drawn on a UK bank, or by credit card (Mastercard, Visa, or American Express), stating card number, expiry date, and full name.

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Topics in colorectal disease. R John Nicholls, Neil J McC Mortensen, John M A Northover, eds. (Pp 160; illustrated; DM 86.) Heidelberg: Springer-Verlag, 1991.

Gastric peptic secretion. R Cheli, A Perasso, G Testino. (Pp 125; illustrated; 60 000 lira) Verona: Cortina International, 1991.

Mechanisms of injury, protection and repair of the upper gastrointestinal tract. A Garner, P E O'Brien, eds. (Pp 540; illustrated; £50.) Chichester: John Wiley, 1991.