

DAMIAN DOWLING
 Department of Gastroenterology,
 The Royal Melbourne Hospital,
 Parkville 3050,
 Victoria,
 Australia

- Hawkey CJ. Healing and prevention of NSAID-induced peptic ulcers. *Scand J Gastroenterol* 1994; 29 (suppl 201): 42-4.
- Moller Hansen J, Hallas J, Lauritsen M, Bytzer P. Non-steroidal anti-inflammatory drugs and ulcer complications: a risk factor analysis for clinical decision-making. *Scand J Gastroenterol* 1996; 31: 126-30.

Reply

EDITOR,—Dr Dowling is right to emphasise that the eradication rate we obtained in our *H pylori* positive, NSAID associated peptic ulcers is fairly low (56%), and more convincing conclusions could have been made by using a more effective eradicating regimen (that is, omeprazole based triple therapy) providing a cure rate of at least 85-90%. Unfortunately, when this clinical study was originally planned (spring 1993), dual therapy (omeprazole with amoxicillin) was considered one of the most effective eradicating regimens available; however, it should be emphasised that the *H pylori* cure rate obtained with omeprazole/amoxicillin in this series is comparable with that reported previously in NSAID unrelated peptic ulcers using the same combination of drugs.^{1,2} We also agree on the fact that our results should be considered preliminary because of the limited number of NSAID associated ulcers treated, and they need to be confirmed in further clinical trials involving larger series of patients on long term NSAID treatment with gastric or duodenal, or both, ulcer disease.

However, two important points emerge from our study. First, unlike NSAID unrelated peptic ulcers, eradication of *H pylori* is not associated with the "cure" of NSAID associated ulcers which tend to recur rapidly after initial healing if the patient continues to receive the NSAID but stops taking antiulcer medication. This necessarily implies that the risk of recurrence induced by the presence of *H pylori* infection, if any, is small and it is only additional to the main risk of receiving long term NSAID treatment. Second, *H pylori* status does not seem to have an important role in the healing of these lesions. This is not only confirmed by the observation that the rate of healing is not increased by eradication of *H pylori*, but also from the fact that *H pylori* negative ulcers respond to omeprazole just as well as *H pylori* positive ones. The major determinant of healing response to the anti-secretory compound in these patients seems to be the concomitant intake of the NSAID during the healing phase, which delays the healing process irrespective of the type of drug used.

Recently, Hawkey *et al.*³ in a very large study involving 541 patients with NSAID related gastroduodenal ulcers or erosions, have shown that infection with *H pylori* does not hamper the healing efficacy of omeprazole 20-40 mg daily; on the contrary, it tends to be associated with higher healing rates, perhaps as a result of the increased antisecretory effects of proton pump inhibitors in this setting.⁴

F PARENTE
 V IMBESI
 G BIANCHI PORRO
 Department of Gastroenterology,
 L. Sacco University Hospital,
 Via G.B. Grassi, 74,
 20157 Milan, Italy

- Tytgat GNJ, van der Hulst RWM. Important acquisition in *Helicobacter pylori* infection. *Curr Opin Gastroenterol* 1996; 11 (suppl 1): 57-60.
- Parente F, Maconi G, Bargiggia S, Colombo E, Bianchi Porro G. Comparison of two different lansoprazole-antibiotic combinations for the eradication of *H. pylori* infection in duodenal ulcer patients. *Aliment Pharmacol Ther* 1996; 10: 211-3.
- Hawkey CJ, Swannel AJ, Yeomans ND, Carlsson M, Floren I, Jallinder M. Increased effectiveness of omeprazole compared to ranitidine in non steroidal anti inflammatory drug (NSAID) users with reference to *H. pylori* status [abstract]. *Gut* 1996; 39 (suppl 1): A33.
- Labenz J, Tillenburg B, Peitz U, Idstrom J-P, Verdu EF, Stolte M, *et al.* *Helicobacter pylori* augments the pH-increasing effect of omeprazole in patients with duodenal ulcer. *Gastroenterology* 1996; 110: 725-32.

Endoscopic papillectomy

EDITOR,—I read the recent article on endoscopic papillectomy by Dr Farrell *et al* (*Gut* 1996; 39: 36-8) with considerable interest because of a simple experiment done in 1964, which indicated that papillectomy might be hazardous.¹ The closed duodenal loop model of haemorrhagic necrotic pancreatitis closely resembles severe human pancreatitis and is caused by the reflux of duodenal contents through the papilla of Vater²; it was suggested that pancreatitis could be produced by much smaller intraduodenal pressures provided that the papilla of Vater was damaged of incompetent.

The duodenal papilla in humans and in dogs is lined by mucosal folds or 'valvules', which serve to prevent regurgitation of duodenal contents.^{3,4} The isolated dog duodenum was filled with coloured saline maintained at a pressure of 30 mm Hg without any fluid escaping from the cut ends of the pancreatic ducts or common bile duct. A simple mucosal papillectomy was performed at the main pancreatic duct, the duodenotomy incision closed, and the intraduodenal pressure raised again to 30 mm Hg. Within two minutes coloured saline oozed from the main pancreatic duct but not from the separate lesser pancreatic duct and associated common bile duct. When the common bile duct papilla was also excised saline escaped from the common bile duct.¹ The histological picture of the papilla, before and after excision (photographed), plus the findings above suggest that: (1) the mucosal portion of the papilla of Vater serves a useful purpose in preventing duodenal reflux; (2) papillectomy may increase the risk of developing pancreatitis, depending on the nature of the underlying pathology for which papillectomy was performed.

A D McCUTCHEON
 28 Maitland Avenue,
 East Kew, Victoria 3102
 Australia

- McCutcheon AD. Reflux of duodenal contents in the pathogenesis of pancreatitis. *Gut* 1964; 5: 260-3.
- McCutcheon AD, Race D. Experimental pancreatitis. A possible aetiology of postoperative pancreatitis. *Ann Surg* 1962; 155: 523-31.
- Dardinski VJ. The anatomy of the major duodenal papilla of man with special reference to its musculature. *J Anat (Lond)* 1935; 69: 469-78.
- Sterling JA. The termination of the common bile duct. *Rev Gastroenterol* 1949; 16: 821-45.

Reply

EDITOR,—We are grateful for Dr McCutcheon's comments and while it is possible that endoscopic papillectomy may increase the risk of developing pancreatitis the fact remains that none of our 10 patients have suffered pancreatitis or ascending cholangitis after papillectomy (25 months follow up to date). In reply, we feel two points should be made. Firstly, the closed duodenal loop model described is not comparable with our patients' situation as there was no distal obstruction and hence no cause for reflux of duodenal contents through the damaged papilla of Vater. Secondly, while the common or external sphincter are excised at papillectomy both internal sphincters (biliary and pancreatic) are preserved hence serving as a natural barrier to reflux of duodenal contents. Finally, we would only recommend endoscopic papillectomy as a means of aiding cannulation in limited circumstances such as a large obstructive ampullary tumour, or an exophytic ampulla with an ectopic orifice or low bile duct stones.

R J FARRELL
 P W N KEELING
 Department of Gastroenterology,
 St James's Hospital, Dublin 8, Ireland

BOOK REVIEWS

Textbook of Gastroenterology. Volumes 1 and 2. Yamada T, Alpers D H, Owyang C, Powell D W, Silverstein F E. (Pp 3456; illustrated; £176.00.) Philadelphia: Lippincott-Raven. 1995. ISBN 0-397-51492-1.

A review of the second edition of this book published in 1995 was only requested in late 1996 (accompanied by an invoice marked "rush!") which explains the interval between publication and this review.

I was brought up on *Gray's Anatomy* to achieve adult height around the dining table. A wide selection of two volume books on gastroenterology could now do this job admirably.

This remarkable book has more than 200 contributors, mostly North American with a smattering of other contributors from seven different countries. Its approach is so different that comparison with other textbooks of gastroenterology is inappropriate. No other book could find the reviewer at the end of volume one, some 67 chapters and 1500 pages later, still not yet through the pylorus. This is not an obsession with the oesophagus and stomach, but extensive consideration of basic mechanisms relevant to clinical problems (26 chapters) and, more importantly, an extended section of similar length concerned with approaches to the symptomatic patient. This section is particularly relevant for the younger postgraduate whose patients in real life present with a constellation of symptoms rather than a specific diagnosis. This focused approach is both a strength and a weakness as

it may have more limited appeal for the more experienced gastroenterologist.

The second half of volume two is also unusual, being devoted to diagnostic and therapeutic procedures including endoscopy, imaging, mucosal biopsy, microbiology, and motility studies. Once again of particular appeal to the younger postgraduate.

Sandwiched between these sections on basic mechanisms, symptoms, and diagnostic and therapeutic approaches are some 60 chapters devoted to diseases as they are manifested sequentially along the gastrointestinal tract.

These contributions are all a pleasure to read and are uniformly well presented and illustrated. The presentations of a consistently high quality and the apparently seamless links from one chapter to the next provide remarkable continuity – a major editorial achievement.

Chapters are extensively referenced. For the next edition reference pruning and selection of more recent references would enhance the book's value. For example, a chapter on inflammatory bowel disease includes references for cyclosporin no later than 1989 and the most recent reference cited in the section on cancer and colitis is 1981.

There are some chapters which are a model of clarity. For example, the chapter on tumours of the small intestine is well written and illustrated with excellent radiographs, histology and summary tables.

For improving postgraduate education the two volume set is now available on CD-ROM which, together with an atlas of gastroenterology, is linked with a self assessment review and guidelines which further increases its value in postgraduate training. For the third edition, I would be inclined to shorten the diagnostic and therapeutic section and to use the space to increase the depth of coverage in the section on specific diseases.

I can no longer claim to be a young gastroenterologist myself, but all the young gastroenterologists in our department have purchased a copy from the wide selection of textbooks on gastroenterology that are now available. In my view this is where this remarkable book makes its major impact. If only sitting on this two volume set was enough to imbibe the information, rather than having to read it!

ROBERT ALLEN

Hepatology: A Textbook of Liver Disease. Volumes 1 and 2. 3rd edn. Boyer Z, ed. (Pp 1970; illustrated; £225.00.) Harcourt Brace. 1996. ISBN 0-7216-4836-3.

This very substantial two volume textbook contains over 1900 pages, 68 chapters and over 400 references per chapter (maximum number 834 on receptor mediated endocytosis). It contains 1328 illustrations which are predominantly in black and white. The index contains over 7700 entries. The 105 contributors are overwhelmingly drawn from the USA, with less than 10% coming from Europe and none based in the UK.

What do these facts tell us about this book? The answer lies in our expectations. For many, textbooks of this size are handsome tomes that adorn the office and are used for reference purposes on an occasional basis. When used in this way, the expectation is that

the information required will be found quickly and easily. In that respect this textbook is excellent, covering many fringe issues not included in other major textbooks of hepatology and having a reasonably comprehensive indexing system. There is a great danger that this reference function will become relevant only to historical material as computer based information technology services become the preferred means for acquiring up to date material.

As a cover to cover read, I would prefer to tackle *Finnegan's Wake* and *War and Peace* in a single weekend. The detail and presentation are daunting, and equal measures of high scholarly ambition and stamina are needed for the task. The editors have strived to balance basic science and clinical hepatology, and to give greater emphasis to the pathophysiology of clinical disease. In doing so, they may have unwittingly produced two textbooks that will appeal to substantially different audiences. I suspect I will have retired from hepatology for many years before I am overwhelmed by the temptation to read 28 pages on Vitamin A metabolism!

It is as a guide to day to day clinical practice that this textbook is least attractive. The bulk is prohibitive and the layout is not conducive to assisting in the management of clinical problems and dilemmas. The US near-monopoly of authorship detracts from the balance of the clinical chapters, but this may have been considered to be an unavoidable marketing strategy. The winds of change that liver transplantation is blowing through the practice of hepatology is not reflected in this textbook, with transplantation issues getting neither the degree of integration nor the scope that they deserve.

The third edition of any book is often the best. It is still sufficiently close to the first publication to be fresh and true to its objectives, and yet mature enough to have learned from its mistakes. That is probably true of Boyer's *Hepatology*. It is also probably the best of its kind. It will continue to sit on my shelf for some time to come.

J O'GRADY

Operative Laparoscopy and Thoracoscopy. Bruce V MacFayden, Jeffery L Ponsky, eds. (Pp 768; illustrated; £180.25.) Philadelphia: Lippincott-Raven. 1996. ISBN 0-7817-0279-8.

This is a comprehensive book with detailed description of just about every minimal access surgical procedure so far conceived. It has a very strong North American bias which is particularly evident in the opening section with regard to training, accreditation and economic considerations. The book is divided into further sections essentially by organ. Most of the chapters are written in the "how I do it" fashion with lots of technical details. There is quite a bit of variation, though, in the introductions and discussions relating to these procedures. In any large book there are bound to be some contentious points. For instance, the interesting chapter on laparoscopic pancreaticoduodenectomy includes advice that percutaneous biopsy should be performed along with a mesenteric angiogram. Many surgeons would argue against the use of both of these techniques in patients with a resectable pancreatic tumour.

For such an expensive book there are far too many typographical errors. The editorial hand is generally weak. Not all of the intra-operative photographs are of textbook quality. There is a lot of anatomical repetition, particularly in the chapters on hernias, and for me, the balance of the book is not right. Eight chapters are devoted to gallstone surgery and three to groin hernias but only one to anti-reflux surgery. There is no mention in the chapter on colon resections of tumour implantation and port site recurrence. The repair of perforated peptic ulcer by omental patching and fibrin glue is not included, nor is the performance of splenectomy by the lateral approach. With one or two notable exceptions, most of the authors have relied on anecdotal rather than published evidence. I am not quite sure who this book is aimed at. It lacks much of the objectivity necessary for the surgical trainee to place minimal access procedures in their correct context. Conversely, the practising surgeon may find large parts of this book irrelevant. Although I personally enjoyed reading many of the chapters, at £180 this book is too expensive for the trainee and does not represent much value for money for the consultant.

D ALDERSON

NOTES

Laparoscopic Surgery

The European Course on Laparoscopic Surgery will be held at University Hospital Saint-Pierre (U.L.B.), Brussels, Belgium, on 13–16 May 1997 (French) and again on 18–21 November 1997 (English). Further information from: Administrative Secretariat, Conference Services s.a., Avenue de l'Observatoire, 3 bte 17, B-1180 Brussels, Belgium. Tel: +32 2 375 16 48; Fax: +32 2 375 32 99.

Clinical Nutrition and Metabolism

The 19th ESPEN Congress on Clinical Nutrition and Metabolism will be held at the RAI Congress Centre, Amsterdam, The Netherlands, on 31 August–3 September 1997. Further information from: Van Namen & Westerlaken Congress Organisation Services, PO Box 1558, 6501 BN Nijmegen, The Netherlands. Tel: +31 24 323 4471; Fax: +31 24 360 1159.

Digestive Endoscopy

A Course in Digestive Endoscopy will be held at the Academic Medical Centre, University of Amsterdam, The Netherlands, on 4–5 September 1997. Further information from: Helma Stockmann, Managing Director, European Postgraduate Gastro-Surgical School, G-4-zuid, Academic Medical Centre, Meibergdreef 9, 1105 AZ Amsterdam, The Netherlands. Tel: +31 20 566 3926; Fax: +31 20 691 4858.