LETTERS TO THE EDITOR

Drug induced pancreatitis

EDITOR,—I have read with interest the article on drug induced pancreatitis by Lankisch *et al* (*Gut* 1995; **37:** 565–7). It is unclear to me how the opinion that in 1·4% of patients disease was drug induced could have been substantiated.

It is difficult to believe that only 22 of 1613 patients were exposed to drugs. What about drugs taken by the remaining 1591; is opinion enough to exclude possible causation, plainly not.

To estimate the true impact of drug induced disease the authors would have had to conduct a controlled study. Nevertheless, the authors have concluded that drug induced acute pancreatitis occurs rarely in clinical practice. That opinion has not been substantiated by this study.

M J S LANGMAN
Department of Medicine, Queen Elizabeth Medical Centre, Queen Elizabeth Hospital, Edgbaston, Birmingham B12 2TH

Reply

EDITOR,—We are grateful for the interest Professor M J S Langman took in our paper and regret that it has obviously given rise to some misunderstanding.

The preselection of patients diagnosed for drug induced acute pancreatitis was made by the centres where they had been treated. All charts of patients considered to fall into this group were reviewed by us. We looked only for drugs, however, currently held responsible for inducing acute pancreatitis. It is possible that in the 135 patients with acute pancreatitis of unknown aetiology drugs had been given still unknown to induce acute pancreatitis and thus the incidence of this aetiology might be somewhat higher.

Prospective studies help to answer open questions but logistic realities pose problems. The two questions at issue are: how frequently does the application of a certain drug lead to acute pancreatitis and, how frequent is drug induced acute pancreatitis among all patients with acute pancreatitis?

The first question is impossible to answer. In view of the great number of patients receiving drugs such as frusemide and oestrogen, it would be impossible to follow up all of them for signs and symptoms of acute pancreatitis. Even the second question is difficult to answer. To make quite sure that the suspected drug has really induced the disease, a re-exposure to the drug in question is necessary, something ethically difficult to justify.

The message of our paper was simply that drug induced acute pancreatitis is probably rare and that the disease usually takes a benign course. Such a retrospective evaluation in a substantial number of patients has not been done before.

P G LANKISCH
Department of Internal Medicine, Municipal Hospital of Lüneburg, D-21339 Lüneburg, Germany

Biliary stenting in the management of bile duct stones

EDITOR,—We read with interest the leading article by Dalton and Chapman (*Gut* 1995; **36:** 485–7). Their suggestion that a sphincterotomy may not always be necessary in these patients is absolutely valid. We would go a step further in stating that in such patients, if the size of the stone is >15–20 mm at ultrasound examination or ERCP, then stenting should be performed with a 7 French stent without sphincterotomy. This will prevent the complications associated with endoscopic sphincterotomy, which occur in 8–10% of patients undergoing the procedure.[1] Furthermore, this would prevent migration of straight stents. As already mentioned in the leading article, there is no evidence to show so far, that 10 French stents are superior to stents of smaller diameter. Although 7 French stents tend to clog earlier than the 10 French ones,[2] they may easily be exchanged when blockage occurs. Endoscopic sphincterotomy may however be required if multiple stents need to be placed.

Apart from maintaining the flow of bile and preventing stone impaction and cholangitis, stenting has other benefits too. Placement of biliary endoprostheses has been shown to decrease the size of the stones on follow up.[3 4] Moreover, in patients with stricture of the common bile duct, where lithotripsy may be difficult or impossible, biliary endoprostheses may resolve such strictures[5–7] in addition to decreasing the size of the stone.

S P MISRA
M DWIVEDI
Department of Gastroenterology, MLN Medical College, Allahabad – 211 001, India

1 Cotton PB. Endoscopic management of bile duct stones; (apples and oranges). *Gut* 1984; **25:** 587–97.
2 Status evaluation: biliary stents. *Gastrointest Endosc* 1992; **38:** 750–2.
3 Chan ACW, Ng EKW, Lai CW, *et al.* Common bile duct stones become smaller after endoscopic biliary stenting. [Abstract]. *Gastrointest Endosc* 1995; **41:** 393.
4 Vallera RA, McGee SG, Shearin M, *et al.* Biliary stents decrease the size of retained common bile duct stones. [Abstract]. *Gastrointest Endosc* 1995; **41:** 419.
5 Bourke MJ, Elfant AB, Alhalel R, Kotan P, Haber GB. Biliary and pancreatic strictures complicating endoscopic biliary sphincterotomy. [Abstract]. Features and endoscopic management. *Gastrointest Endosc* 1995; **41:** 390.
6 Hmeidan A, Jacob J, Sherman S, Lehman GA. Benign biliary strictures: outcome of endoscopic therapy. [Abstract]. *Gastrointest Endosc* 1995; **41:** 399.
7 Hmeidan A, Jacob J, Sherman S, Lehman GA. Benign biliary strictures: frequency and management at ERCP. [Abstract]. *Gastrointest Endosc* 1995; **41:** 399.

BOOK REVIEW

Inflammatory Bowel Disease. 4th ed. Edited by J B Kirsner, R G Shorter. (Pp 1033; illustrated; £116). Baltimore: Williams and Wilkins, 1995. ISBN 0–683–04627–6.

The fourth edition of 'Kirsner and Shorter' appears exactly 20 years after the first edition and is a tribute to the extraordinary energy of its editors. Compared with the third edition published in 1985, the current volume has expanded by over 200 pages and many new authors have been introduced. It remains an all American book but its perspective of the literature is global and generally well balanced. Inevitably in a book of 41 chapters devoted to two diseases, there is some repetition but this is no bad thing if the volume is used for reference or for browsing. The largest expansion compared with previous editions concerns pathogenesis – 11chapters compared with six in the third edition. This rightly reflects the remarkable explosion of interest in the immunological and inflammatory mechanisms in pathogenesis that has occurred during the past 10–15 years.

This volume provides us with the most comprehensive account of ulcerative colitis and Crohn's disease currently available. It is obsessively referenced and therefore provides an excellent entry into the original literature. It provides elegant accounts of the experimental, immunological, and pathophysiological mechanisms that may be involved in pathogenesis but also provides detailed accounts of medical and surgical treatment. The psychosocial problems of the diseases are amplified by a final chapter written from the perspectives of an affected subject and a multiply affected family.

Clinical gastroenterologists will find this book invaluable, those in training will find a mine of information, and for the IBD specialist it will continue to be a much used book of reference. It well lives up to its aims as described in the preface although I am not as sanguine as the editors that the aetiology of either disease will be understood within the next five years.

D P JEWELL

CORRECTION

An error occurred in the paper by Dr John and others (*Gut* 1996; **38:** 33–39). The title of the paper should read 'Positive somatostatin receptor scintigraphy correlates with the presence of somatostatin receptor subtype 2 and 5'.