

Correspondence

Bile salt-induced gastric mucosal damage

Sir, – The paper by Drs Lewi and Carter on bile salt-induced mucosal damage (*Gut* 1983; **24**: 33–7) provides clearcut evidence that histamine receptors are not involved in the induction of changes in ionic flux by sodium taurocholate. Those results are in keeping with the conclusions of other studies, from which it may be inferred that the pathogenesis of mucosal damage by bile salts is not to be related to altered gastric acid secretion but to acute impairment of the so-called ‘mucus-bicarbonate’ barrier. In fact bile salts are known to affect the gastric mucus layer by decreasing its viscosity¹ and taurocholate has been shown to inhibit bicarbonate production by the human stomach.² The failure of H₂-receptor antagonists in preventing the taurocholate-induced increase in mucosal permeability confirms that bile salts damage the gastric mucosa through a different mechanism. A direct damaging effect on the parietal cells, as suggested by Lewin *et al* in the same issue of *Gut* (pp. 28–32), may well be the cause of acid hyposecretion but it appears to be a consequence of the fact that the ‘mucus-bicarbonate’ barrier becomes unable to protect the gastric mucosa.

From a practical point of view the findings of Lewi and Carter also imply that there is no point in using H₂-blockers in the treatment of reflux gastritis and that drugs strengthening the mucosal defences and/or binding bile salts should be preferred.

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References

- 1 Martin GP, Marriott C, Kellaway IW. Direct effect of bile salts and phospholipids on the physical properties of mucus. *Gut* 1978; **19**: 103–7.
- 2 Rees WDW, Warhurst G, Turnberg LA. Studies of bicarbonate secretion by the normal human stomach in vivo: effect of sodium taurocholate and aspirin. (Abstract). *Gastroenterology* 1982; **82**: 1158.

Books

Practical gastrointestinal endoscopy By Peter B Cotton and Christopher B Williams. (Pp. 204; illustrated; £16.50.) Oxford: Blackwell, 1982. A review of a second edition assumes that the reader

is already familiar with the first. Suffice it to say that the book, in its first edition, was very good. It concerned itself with the practical aspects of endoscopy and achieved exactly what the authors intended. Blackwells had backed a winner and within three years we are given a second edition. The aims are unchanged, and the book itself only a few pages longer. The print is smaller but very legible; the layout is similar, with wide margins and splendid line drawings galore, more than before, with some improvements; and there are more colour plates of endoscopic appearances, but these are perhaps superfluous. Most chapters now have a small list of key references, a good idea. The text has been revised and some chapters have been considerably expanded, taking recent developments fully into account. This is especially helpful in the chapters dealing with the therapeutic aspects of endoscopy. There is a new chapter on infection, cleaning and disinfection, and a thought-provoking postscript. The book is, thus, a comprehensive guide to the practical aspects of modern gastrointestinal fibre-endoscopy.

Peter Cotton and Christopher Williams are widely admired as clinical gastroenterologists with a flair for practising and teaching endoscopy, for developing new techniques, and for exploring the place of endoscopy in gastrointestinal research. Aply assisted by their artists and their publisher they have produced a distillate of their knowledge and experience, to the great benefit of endoscopists whatever their experience. The book deserves an even wider readership than the first edition.

K F R SCHILLER

Advances in internal medicine Edited by G H Stollerman (Pp. 624; illustrated; £33.25). Chicago: Year Book Medical Publishers. 1982.

Gastroenterologists have many review articles and books to help them keep up-to-date in their own field. Internal medicine specialists have their own annual *Advances* which each year devotes three or four chapters to gastroenterology. In 1979 they had enteric hyperoxaluria, gut fluxes and abdominal CT; in 1980 lipoproteins in liver disease, cholestasis and acute pancreatitis; and last year non-A non-B hepatitis, hyperamylasaemia, and intestinal adaptation to bowel resection.

Cattau and Castell from Bethesda elucidate the symptoms of oesophageal dysfunction. I liked their dismissal of presbyoesophagus as a diagnostic entity, and of iron deficiency anaemia as an association with oesophageal webs in spite of Plummer and Vinson. The mimicry of cardiac pain by oesophageal disease