

the difference between pre and post therapy nocturnal acid outputs reached statistical significance.

(2) The pretreatment daytime median acidity profile presents pH values which are surprisingly higher than those previously published in identical profiles pertaining to duodenal ulcer patients in clinical remission.^{3,4} Perhaps shortcomings in the calibration procedure or relevant drift of the glass electrodes may have been responsible for this. More coincident daytime pH profiles between the pre and during treatment groups would have also been expected in relation to the authors' statement that single evening doses of nizatidine 300 mg *nocte* only inhibit nocturnal acidity '... without causing any suppression of daytime intragastric pH'. On the contrary, the profile of the final day of treatment runs almost constantly below the basal one throughout the whole day and, as this happens at median pH values which are mainly between 1 and 2 pH units, the difference is very high in terms of hydrogen ion activity.

(3) When performing multiple non-parametric testing, such as the one that the authors applied on daytime pH recordings of 30 min intervals over 12 hours, the correction of the significance level of the α probability is mandatory. This omission can provide differences which are not actual or are too optimistic, especially when the number of patients is too low as in this study. As no mention of its application was made by the authors, there is some doubt as to the reliability of the significant p value (<0.05) related to the mid morning and mid afternoon differences they observed by comparing the multiple 30 min periods of the three daytime pH profiles. If so, these partial differences cannot be considered as '... some evidence for daytime rebound hyperacidity'. Therefore, it is difficult to accept that increased acid secretion presumably caused by up regulation of H₂-receptors occurs only during the night. The fact that measurement of pH instead of acid output might have overlooked this effect during the daytime is a speculation which is a result of the adoption of two different techniques for studying the same biological phenomenon.

Although it is of great interest to establish whether rebound hyperacidity does or does not occur after stopping H₂ antagonist treatment, larger sample sizes and more rigorous methodology are required to provide a satisfactory answer to this question.

VINCENZO SAVARINO AND GIUSEPPE SANDRO MELA

*Instituto Scientifico di Medicina Interna,
Cattedra di Gastroenterologia e Clinica Medica R,
Università di Genova,
Genova, Italy.*

References

- 1 Fullarton GM, McLauchlan G, Macdonald A, Crean GP, McColl KEL. Rebound nocturnal hypersecretion after four weeks treatment with an H₂ receptor antagonist. *Gut* 1989; **30**: 449-54.
- 2 Mc Coll KEL, Fullarton G, Crean GP. Effects of 4 week treatment with nizatidine on nocturnal gastric acid secretion, daytime and integrated gastrin response. European Nizatidine Symposium 1987, Nov 12-13, Brussels.
- 3 Merki HS, Witzel L, Walt AP, *et al.* Comparison of ranitidine 300 mg twice daily, 300 mg at night and placebo on 24-hour intragastric acidity of duodenal ulcer patients. *Aliment Pharmacol Ther* 1987; **1**: 217-23.
- 4 Merki HS, Witzel L, Kaufman D, *et al.* Continuous intravenous infusions of famotidine maintain high intragastric pH in duodenal ulcer. *Gut* 1988; **29**: 453-7.

Reply

SIR, — Dr Savarino is correct to stress the importance of confirming rebound hypersecretion after H₂-receptor antagonist therapy in larger numbers of patients. We have, in fact, recently completed a much larger study where this was confirmed at highly significant levels.¹ In addition, there has been a further study from Dr Pounder's group also confirming rebound hypersecretion after H₂-receptor antagonist therapy.²

There are clearly variations in intragastric acidity as measured by *in situ* pH electrodes particularly when equipment varies between centres. Our pretreatment intragastric pH profiles, in duodenal ulcer patients are certainly lower than our comparable profiles using identical equipment in healthy volunteers.³ We would not therefore accept that we have problems with our combined glass electrodes in terms of calibration or drift as we have recently shown that the combined glass electrode (Radiometer GK 2802C) has a shorter response time, better sensitivity and significantly less drift than other electrodes.⁴

Finally, we cannot accept that more 'rigorous' methodology would provide a more satisfactory answer to the question of rebound hypersecretion. The technique used in this study allows a 24h assessment of related aspects of gastric secretory function (acidity *and* output) which are complementary.

GRANT M FULLARTON AND KENNETH E L MCCOLL
*Department of Medicine,
Gardiner Institute,
Western Infirmary,
Glasgow G11 6NT*

References

- 1 Fullarton GM, McLauchlan G, Macdonald A, Crean GP,

- McColl KEL. Rebound hypersecretion after 4 weeks H2 antagonist therapy. American Gastroenterology Association, May 17th, 1989. Washington DC.
- 2 Nwokolo CU, Smith JTL, Pounder RE. Rebound intra-gastric acidity after acute withdrawal of cimetidine, famotidine or nizatidine is not mediated by persisting hypergastrinaemia. [Abstract] *Gut* 1989; **30**: A725.
 - 3 McLauchlan G, Fullarton GM, Crean GP, McColl KEL. Comparison of gastric body and antral pH: a 24 hour ambulatory study in healthy volunteers. *Gut* 1989; **30**: 573-8.
 - 4 McLauchlan G, Rawlings JM, Lucas ML, McCloy RF, Crean GP, McColl KEL. Electrodes for 24 hour pH monitoring - a comparative study. *Gut* 1987; **28**: 935-9.

Books

Gastrointestinal radiology: self assessment in radiology and imaging. By M Berger. (Pp. 174; illustrated; £19.50.) London: Wolfe Medical Publications, 1985 (Paperback Edition 1988).

This is a paperback edition of a hardback book produced in 1985. It includes not only gastrointestinal radiology, but also a sprinkling of upper abdominal ultrasound. Computed tomography and nuclear medicine. The text is organised in a sensible way. A series of questions is attached to a radiograph or a scan on the right hand page, and the answers are then written on the next (left hand) page with a copy of the original images, often annotated with helpful arrows or numbers related to the answer. The images are of uniform high quality with the exception of most of the ultrasound scans (and to a lesser extent, the CT scans) which are frankly showing their age. The questions are on the whole fairly simple. I was slightly irritated by the manner in which the second part of the question would often imply the answer to the first part.

I have few quibbles with the answers to the text and they often give helpful thumb nail sketches of the condition involved. Q4 suggests cobblestoning is a feature of ileal TB; this is usually considered very rare and a distinguishing feature. I think most people would clear a bile duct of stones at ERCP in a patient with cholangitis rather than just perform a sphincterotomy and hope the stones pass (as is suggested in Q66).

My main problem is in deciding to whom the book should be recommended. Radiologists coming up to FRCR would find the examples too easy and the technique related questions are so basic that they are obviously not aimed at them. I suspect that gastroenterologists in training would find the clinical

information too basic, and the questions are often so written that the answer is given away even without looking at the images. Perhaps first year radiologists and non-gastroenterological physicians working towards MRCP would find it most helpful and certainly light relief in any revision programme.

P J SHORVON

Current hepatology. Vol. 8. Edited by G Gitnick. (Pp. 485; illustrated; £53.50.) Chicago: Yearbook Medical Publishers, 1988.

This is the latest of a popular series describing recent advances in liver disease. It is multi-author, and each year fresh writers are chosen so that chapters do not become stereotyped or stale, and fresh topics may be included.

The present volume covers such subjects as portal hypertension, liver disease and surgery of the gall bladder, bile ducts, and liver. There is even an update by M Sherman on the molecular biology of liver disease. There is also a most welcome chapter, written by K Okuda, covering recent articles on liver disease, published in the Japanese language.

I enjoyed the chapter by R Koretz, entitled 'Hepatitis - words and music'. With great skill he has incorporated the themes of hepatitis into those of such Broadway musicals as *Annie get your gun*, *The King and I*, and *The sound of music*. This needed considerable ingenuity, review of 377 scientific articles and, presumably, reading librettos of the shows.

J Reichen and R Preisig, in their chapter on cirrhosis, note *inter alia* the value of third generation cephalosporins to treat systemic infections including bacterial peritonitis. They applaud the coming of age of quantitative tests of liver function.

References are liberal and well chosen, but unfortunately, most of them have been published in 1985 and 1986. Readers, whether general physicians or gastroenterologists, will find that this book gives them a birds eye view of advances in liver disease in a manageable and readable form.

SHEILA SHERLOCK

News

Endoscopy 1990: The Southern California Society For Gastrointestinal Endoscopy Symposium

This symposium will be held on 24 and 25 February, 1990, in Los Angeles, California. For further information contact: John L Petrini, MD, Sansum