Towards a better assessment of reflux oesophagitis

Sir,—We read with interest the exhaustive and balanced overview by Dr Colin-Jones on gastro-oesophageal reflux. We were particularly interested to see that an authoritative reviewer has at last officially suggested an adaptation of the notorious endoscopic classification of oesophagitis by Savary and Miller.1 For reasons which are beyond our understanding, we feel it important to point out that the term ‘grade 0 oesophagitis’ is often used. In our opinion mild (non-erosive) oesophagitis should be distinguished from normal reflux esophagitis, since the latter is not endoscopically visible. A possible solution to this problem is to use the terms ‘mild reflux esophagitis’ or ‘‘oesophagitis’’ to refer to reflux that is visible on endoscopy but not accompanied by lesions or symptoms. This approach would also allow us to include non erosive lesions within the concept of ‘oesophagitis’.

On the other hand, in clinical trials the endoscopic evaluation criteria are often at variance with Savary and Miller’s classification and tend to include non erosive forms as well, in order to obtain a more realistic approach to the problem.

We are not aware of any attempt to improve the results of H2 receptor blockers in the treatment of reflux oesophagitis, we believe that the time of administration can also play a major role. Contrary to that reported in the past,2 mild reflux oesophagitis has been claimed to be an important factor in the pathogenesis of the disease.3 Therefore a single dose of a H2 blocker at night might not be ideal in some subjects. The results of a recent cooperative study performed in northern Italy suggest to support this view.

A group of 33 healthy controls was initially examined by means of 24 hour ambulatory pH-metry to determine the upper normal limit, on the basis of De Meester’s criteria (mean ± 2SD) of the time with pH < 4. Accordingly, 112 consecutive subjects with normal pH-metry were detected and could be divided in upright (53%) or supine (41%) refluxers and in patients with reflux in both positions (36%). These figures differ from De Meester’s findings and in particular the number of upright refluxers is substantially higher (53% v. 9%). The reasons for these discrepancies are unclear. It must be noted, however, that the Italian study was carried out in outpatients and not subjected to dietary restrictions, whereas De Meester examined only hospitalised patients on a standard diet. At any rate, the high number of upright refluxers in the Italian series makes the habit of indiscreetly treating reflux oesophagitis with a single bedtime dose of a H2 receptor blocker questionable.

To achieve better results, the choice of administering the drug in the morning and at bedtime only at night should be based on the results of 24-hour pH-metry. For practical reasons we cannot expect that each and every subject with reflux oesophagitis can have previously been submitted to the test in order to obtain a ‘personalised’ therapy. On the other hand, at least in patients who fail to respond to treatment, the time of administration of H2-blockers should be adjusted to the results of pH-metry. This does not apply to omeprazole, the long lasting action of which makes it irrelevant the time of administration. The superior results observed with omeprazole, including healing of most resistant to H2-blockers, possibly rely not only upon its greater antisecretory effect, but also upon its ability to suppress the acidity of refluxate throughout the whole day.

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The oesophageal burst activity of intestinal macrophages in normal and inflammatory bowel disease.

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Helicobacter associated gastritis in patients with duodenal ulcer: the influence of various drugs

Sir,—We read with great interest the study reported by Loffeld et al4 on the effects of colchicine bisulfate (CBS) on Helicobacter associated gastritis in patients with active duodenal ulcer (DU).

Thirty one patients with active duodenal ulcer who fulfilled the following criteria were included in the study: patients with HLO test was positive in the same biopsy, the histology failed to show HLOs, or vice versa, the present of Helicobacter associated gastritis was initially confirmed and subsequently followed up in all the patients. Sections for histological detection of Helicobacter like organisms (HLO’s) were stained with Giemsa stain.

HLO test results were arbitrarily classified into four grades, as follows: grade 3: positive within the first 20 min of inoculation; grade 2: positive within the first 24 hours; grade 1: positive within the first 24 hours; grade 0: negative.

Antral gastritis was classified histologically into four grades (1, 2, 3, 4) according to Halter and Siebenmann5 by one pathologist (PD); semi-quantitative estimation of HLO’s presence on biopsy material was by the same pathologist “blindly” that is, without any information on the HLO test results. There was, however, no correlation between his semiquantitative estimation and the HLO test results. If HLO test was positive and the histology failed to show HLOs, or vice versa, the HLO test was considered to be positive grade 3.

The patients were divided into three groups (a, b, c) according to the medication given: Patients in group a (12) were given CBS, 240 mg/bid, in group b (13) sucralfate, 2 g/bid, and

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