Correspondence

stool samples and rectal biopsy showed a moderately active non-specific proctitis. Two weeks after his admission discrete ulceration and a thickened polypoid mucosa were evident in the descending and distal transverse colon at a limited colonoscopy. The appearances were thought to favour Crohn’s disease and the histology was equivocal. He was treated with corticosteroids and was discharged feeling well three weeks after admission on maintenance sulphasalazine. Six months after presentation repeat colonoscopy showed a pancolitis which histologically looked more like Crohn’s disease than ulcerative colitis. He remained asymptomatic until four years after his initial admission to hospital when diarrhoea recurred. A sigmoidoscopy showed a granular mucosa and barium enema showed a pancolitis. Rectal biopsy favoured the diagnosis of ulcerative colitis.

This patient had no bowel symptoms before his infective diarrhoea and it seems likely, as in the cases reported by Willoughby et al., that the chronic colitis was triggered by the enteric infection. There is much evidence to link the onset of chronic colitis to enteric infection with a variety of pathogens which more usually cause a self-limiting inflammatory colitis like Aeromonas hydrophila. Examples other than those referred to by Willoughby et al include cases where chronic disease was linked to infections with Staphylococcus aureus,1 Entamoeba histolytica,2 and salmonella sp.3 Furthermore in a prospective study of acute colitis E coli with characteristics associated with pathogenicity were found in patients in their first attack of ulcerative colitis.4

We agree that wide ranging microbiological studies are necessary early in the course of chronic colitis to elucidate this relationship more fully.

We thank Dr G Neale for allowing us to report a patient in his care.

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References


Omeprazole versus ranitidine

Sir,—I was interested to read the recent paper by Delchier et al in which omeprazole 20 mg once daily was compared with ranitidine 150 mg bd in the treatment of ‘resistant’ duodenal ulcers. The findings that the two treatments are equivalent is at variance with other studies in which omeprazole has been compared with ranitidine in the treatment of duodenal ulcers including ‘resistant’ ulcers.

The authors comment on some of the reasons for this unexpected finding. It is difficult to accept that duodenal ulcers can be regarded as truly ‘resistant’ after only six weeks’ treatment with an H2 blocker, and it would seem important to have established that the patients were compliant with the prescribed H2 blocker before entering such a study. Moreover, 60% of patients were asymptomatic at entry to the study. In practice, outside the constraints of a clinical trial, such patients would not be likely to be detected, and certainly not identified as a separate group, for endoscopy to confirm the healing of straightforward duodenal ulcers is uncommon.

The unusual findings in this study seem likely to be a reflection of patient selection, and do not bring into question the consistent findings from comparative studies of omeprazole and H2 blockers which show a greater proportion of patients treated with omeprazole healed and experiencing symptom relief within two to four weeks, irrespective of the presence or absence of complicating factors.

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References

Processing gastric pH measurements

Sir,—The paper by Chiverton et al not only provides stimulating clinical pharmacological results, but also uses a non-traditional approach for processing gastric pH measurements.

For each subject, a working file was obtained by averaging raw fast acquired pH recordings of each minute (1440 points/24 h). The average acidity during a given time window was expressed as the arithmetic mean of pHs. This acidity index was normally distributed on the basis of the assessment of skewness and kurtosis among their 15 subjects, and therefore it could be handled using parametric statistics for assessing differences, if any, between treatments. The 0-01 probability threshold was used and mean circadian pH profiles were drawn to describe the average behaviour of each treatment.

This work contains several interesting innovations. The use of one minute averaged working files reduces the reading noise and provides tracings which are comparable with those obtained with higher frequency scanning rates, without loss of relevant information. Such a standardisation is necessary, as the value of acidity indexes is function of the working sampling rate. The mean of pHs is an index of average acidity during a predefined time window which is more reliable than the median, particularly when drug related events are concerned. Where appropriate, parametric statistics allow the use of numerous powerful techniques which are more robust than equivalent non-parametric methods or exist only in the parametric form. The 0.01 probability threshold is five times more severe than the commonly adopted 0.05, thus making it possible to arrive at firmer clinical conclusions.

The demonstration that means of pHs are normally distributed among the whole population is a crucial point of this paper. The authors calculated skewness and kurtosis to assess the normality of this acidity index, but, as the distribution of these moments of the mean does not approach the normal closely until the sample size is over 1000, the results they obtained from 15 subjects must be confirmed on larger samples.

In the Figure are reported the frequency distributions of both 24 h means and medians of pHs calculated from one minute averaged 259 pH profiles we obtained with bedtime and twice daily doses of various H2 blockers (famotidine 40 and 20 mg; ranitidine 300 and 150 mg, nizatidine 300 and 150 mg, cimetidine 800 mg). As one can see, the individual 24 h means of pHs are normally distributed, whereas the medians are far from having a gaussian distribution.

![Graph](image-url)
Omeprazole v ranitidine.

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_Gut_ 1989 30: 1437-1438
doi: 10.1136/gut.30.10.1437

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