

meticulous detail, and the superiority of a potent antacid, or cimetidine, over placebo is not in question. There is, however, the problem of translating the absolutes of controlled trials into the therapeutics of peptic ulcer. The design of both studies favoured exaggerated remission rates for active and placebo therapy, and it is unlikely that similar rates would have been achieved had patients been enrolled on ulcer healing.

Double blind randomised, placebo controlled maintenance studies, embellished by a plethora of data on frequency of routine endoscopy, definition of ulcer recurrence, asymptomatic recurrence, smoking, duration of disease, previous active therapy etc. have, over the years, assumed an almost unchallengeable mystique. The list of variables, however, is an evolving one. *Campylobacter pylori*, parietal cell sensitivity on ulcer healing<sup>2</sup> and urinary bismuth levels<sup>3</sup> have recently been suggested, and to these must now be added the time interval between recent ulcer healing and entry into study. As the majority of recurrences occur within the first few months after ulcer healing, we would suggest that the time interval between healing and entry be considered before trying to compare apples with pears. Ideally, maintenance studies should only include patients enrolled immediately after endoscopic healing and withdrawal of the healing agent.

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#### Disturbed fibrinolysis in patients with inflammatory bowel disease

SIR.—The potentially important findings reported by Dr de Jong and colleagues (*Gut* 1989; **30**: 188–94) on fibrinolytic abnormalities in inflammatory bowel disease patients are diminished in value by misuse of statistical methods. It is claimed that the finding of a median prothrombin time of 18 s (normal range 15–19 s) in patients is so different from the median prothrombin time of 17 s in controls that such a difference would not be expected by chance if the experiment were repeated 1000 times. In a study of 28 patients with great overlap between the two groups this is clearly nonsensical.

In Fig. 2 where the actual data for plasminogen activator inhibition are shown the groups appear to be virtually identical: indeed if one performs a Wilcoxon's rank-sum test on the points there is no difference between the two groups ( $T_1=724$ ,  $T_2=872$ ), though a level of significance  $p<0.01$  is claimed.

The same considerations apply to other aspects of the data as presented and this renders the conclusions of the study invalid.

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#### Rebound nocturnal hypersecretion after H<sub>2</sub>-receptor antagonist

SIR.—The recent paper by Fullarton *et al*<sup>1</sup> gives rise to some important criticism. There are three major points to emphasise: (1) The small number of patients (eight) enrolled and the marked individual variation of their secretory patterns (see the non-homogeneous nocturnal acid output values in the pretreatment phase) reduce the reliability of the study. This is particularly so when considering that, by simply adding two cases to the six patients of the authors' interim report,<sup>2</sup> median pH values of the three daytime profiles changed dramatically – for example, from pH 0.7 to pH 1.3 on treatment, and