Correspondence

Relationship between disease activity indices and colonoscopic findings in patients with colonic inflammatory bowel diseases

SIR.—Gomes et al., found only a poor correlation between the colonoscopic or histologic findings in 50 patients with inflammatory bowel disease, and the indices of disease activity used, they showed that these measures of disease activity did not reflect severity or extent of disease at organ level.

Focusing our attention only on the 28 patients affected by ulcerative colitis, they found no significant correlation between macroscopic or microscopic score and clinical index score, C-reactive protein, white blood cells platelets or albumin concentration, while there was a good correlation between micro and macroscopic score.

In a very similar study (that is currently being published) we used the same clinical scoring system used by Gomes et al., and the one previously developed by Cooke and Prior, to evaluate disease severity in 50 patients with ulcerative colitis, from a clinical and biochemical point of view respectively, who underwent a colonoscopy to investigate possible relationships between clinical, biochemical and morphological appearances.

At colonoscopy the colon was divided into seven regions and macroscopic and microscopic scores were developed for grading morphological severity appearance. Using Spearman’s rank parametric test for statistical evaluation, we found significant correlation between macroscopic and microscopic appearance, but neither of these were correlated with the biochemical disease activity index, as Gomes et al. found; moreover, the clinical evaluation method was correlated statistically with macroscopic (r=0.38, p<0.01), microscopic aspects (r=0.31, p<0.05) and with the activity index (r=0.34, p 140.05), differing from what was observed by Gomes et al. A possible explanation of the different findings between Gomes et al.’s and our series could be because of the different number of ulcerative colitis patients examined (28 and 50 Gomes et al. and in our study respectively). It may also be noted that previous extensive studies supported a correlation between mucosal appearance and some clinical variables—for example, well being, rectal bleeding and stool consistency,—even if no relationship with disease extension can be proposed.

We therefore think that in these patients the indication for colonoscopic examination, except for those periodically established for early detection of neoplastic changes, should be considered only after a clinical evaluation suggesting a possible disease relapse (why carry out a colonoscopy in patients in well established clinical and biochemical remission?). This approach enables to avoid an indiscriminate repetition of endoscopic and histological evaluations also taking into account the high social and individual cost of such examinations.

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Twenty four hour intragastric acidity analysis for the future

SIR.—I welcome the leading article by Dr Walt (Gut 1986; 27: 1–9) and agree with much of it. Unfortunately it contains some misleading conceptions.

The formula relating pH to hydrogen ion activity is incorrect and should read –

\[
\text{pH} = \log\left(\frac{1000}{\text{H}^+}\right), \text{ or more simply as } \\
\text{pH} = 3 - \log (\text{H}^+).
\]

Referring to Figure 1, Dr Walt remarks that ‘although the general pattern of acidity is similar no point coincides’. In fact the total lack of coincident points arises solely from the choice of the range shown for pH. It is the pattern which is important, although this too will differ visually with changes in scale.

Dr Walt uses the skewed distribution of pH recordings shown in Figure 4 to justify the use of medians and ranges to summarise data from several patients. That figure provides no such justification as it includes the changes over time. The distributions to investigate are those of the recordings at fixed times. These may indeed be similar to Figure 4, but they are certainly not necessarily so.

Because of the non-linear relationship between the two measurements, if pH recordings are distributed normally then H+ activities could not be, and vice versa. A normally distributed measurement could be analysed parametrically, and used, if required, to provide summary values in the units of the other measurement via the transformation. Arithmetic averages would be appropriate only for the former measurement. The use of median values obviates the need to decide which, if either, of pH and H+ activity should be analysed parametrically. With an odd number of patients, the analysis of median
values will be identical for the two measurements; the 'minimal differences between analyses (Fig. 6)' is a consequence of the conventional definition for an even number of values of the median as the arithmetic average of the two middle values.

I endorse Dr Walt's exhortation to authors 'to adequately explain their methods of calculation'.

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Reply
sir,—I thank Mr Robinson for the formula correction. I may have misinterpreted but I am worried by the underlying implication of the remainder of his comments. Mr Robinson seems to be arguing in favour of having many available methods of calculation with the ability to choose that which, serves the present purpose best. As I tried to show in my article, the very existence of different methods allows questionable manipulation of data. I would like to see unification of data handling by statistically acceptable means. This may limit confusion when people discuss the inhibition of acidity achieved by various drugs or operations in comparative terms.

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Another example of Strongyloides stercoralis infection associated with cimetidine in an immunosuppressed patient
sir,—We read with interest the paper by Ainley et al on a case of strongyloides hyperinfection associated with cimetidine therapy in an immunosuppressed patient and we would like to report a similar observation.

Mrs L was born in France in 1931 and had never left this country. She was treated in 1970 and 1977 for Hodgkin's disease. She received cimetidine 800 mg daily since May 1984 for a prepyloric ulcer related to non steroidal anti-inflammatory drug therapy; there was neither gastric nor blood hypereosinophilia at this time. She was admitted again in July 1984 for abdominal pain, vomiting, urticarian eruption and diarrhoea. Clinical examination and chest radiograph were normal. Peripheral blood count showed a dramatic increase in eosinophilic cells (28500/mm3). Upper endoscopy showed erythematosus gastritis and duodenitis without any erosion and biopsies revealed an eosinophilic infiltration with numerous cross-sectional views of Strongyloides stercoralis in the duodenal mucosa. Stool specimens were also positive for larvae. The patient was successfully treated with two courses of thiabendazole (25 mg/kg for three days) and had further negative stool examination and returned to a normal white cell count.

As in Ainley et al's case report the timing suggests that hyperinfection was related to cimetidine therapy and we believe that our observation is the second report published to date.

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References

Cimetidine and gastrointestinal haemorrhage in critically ill patients
sir,—I read with interest the report by Groll and colleagues (Gut 1986; 27: 135–40) in which cimetidine was found to confer no statistical benefit in reducing gastrointestinal haemorrhage in critically ill patients. In their discussion, however, the authors made no mention of possible complications arising from this form of treatment and perhaps this could have been noted.

Atherton and White1 found that in ventilated patients, in an intensive care unit, gastric colonisation by Gram-ve intestinal bacteria (GNIB) may occur before culture of the same organisms from the trachea. In a further study from an intensive care unit, in which patients were given cimetidine or antacids, it was concluded that this form of treatment may encourage airways colonisation and predispose patients to develop pneumonia caused by GNIB.2 Another study confirmed that when intragastric pH exceeded 4, the stomach became rapidly colonised by GNIB and it was suggested that this may have implications in terms of crossinfection or the development of aspiration pneumonia.3

In addition, when the effects of cimetidine on vomiting and on the volume of nasogastric aspirate produced postoperatively were studied, an increase in pneumonia was found in the group which had been given cimetidine, although the organisms cultured were not reported on.4 Furthermore, a good correlation has been reported between gastric aspirate culture and cultures from infected wounds, after gastric surgery.5