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 erythematous 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 White 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 airway colonisation and predispose patients to develop pneumonia caused by GNIB. 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.

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. Furthermore, a good correlation has been reported between gastric aspirate culture and cultures from infected wounds, after gastric surgery.