study" listed by Short *et al.*³ is that of gastric acidity in patients with Sjogren's syndrome.

Furthermore, they have reviewed the question of gastric acidity in rheumatoid arthritis,³ particularly with reference to Edstrom's studies.² The latter author found achlorhydria among 12.8% of patients with rheumatoid arthritis under 40 and 28.6% of those over 40, a higher incidence than in comparable control groups. Short *et al.* confirmed these findings for the under 40 age group. While it may be questionable whether the data as presented by these workers can be subjected to proper statistical analysis the studies clearly deserve mention. Together with the point about Sjogren's syndrome they imply that studies of acid secretion should be done before implicating other factors—for example, anti-inflammatory drugs—as a cause of hypergastrinaemia.—I am, etc.,

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Gastric Secretion over the Years

SIR,—In your leading article (18 August, p. 368) you concluded reasonably that "it is not possible to decide from these relatively limited data whether hypersecretion precedes or follows duodenal ulceration, though the evidence weighs in favour of hypersecretion being an aetiological factor." However, the follow-up studies cited¹⁻⁵ were all of small numbers. There are other, larger, series in which the measurement was of serum pepsinogen and not gastric acid.

Fourteen of 2,031 recruits developed, during their two-year army service, x-ray evidence of duodenal (13) or gastric (1) ulcers, and their mean serum pepsinogen level (707 units) was significantly higher than that of the whole population (440 units).⁶ Thirteen of the 14 with ulcers had a serum pepsinogen level above the normal mean and 10 above the mean plus one standard deviation. In these two years 15% of men in the top 5% of the serum pepsinogen range developed a duodenal ulcer.⁷ A two-year follow-up of a smaller group of 263 craftsmen showed an identical tendency for radiological ulcers to appear in those subjects previously free of dyspepsia who originally had high levels of serum pepsinogen.⁸ A four-year follow-up of 931 Yale freshmen showed that seven of the eight who developed duodenal ulcer originally had high or high normal serum pepsinogen levels.⁹

In so far as serum pepsinogen is an index of gastric secretory mass, these American studies support the hypothesis^{10 11} that hypersecretion precedes and predisposes to duodenal ulceration and that duodenal ulcers develop almost exclusively in those in the upper half of the range of secretion of acid and pepsin.—I am, etc.,

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Forcible Examination

SIR,—I found that your leading article on the Frau Meinhof case (1 September, p. 466) only added confusion to an already puzzling situation.

I understand that Frau Meinhof's fitness to plead is in question. A prisoner awaiting trial in Scotland or England whose fitness to plead was questioned by the prosecution would be referred for psychiatric examination. If the accused objected to such examination and the psychiatrist believed there was good evidence from the nature of the crime and observations by others that the accused might be suffering from serious mental illness he would persist in attempting a psychiatric examination despite the prisoner's objections and would submit a report to the court. Is the writer of your leading article objecting to this?

To a Scottish psychiatrist the Frau Meinhof case is puzzling as in this country a decision on fitness to plead is based on an opinion of the accused's mental processes—a demonstration of structural brain damage would be essentially irrelevant. An account of the German view of fitness to plead would have been more helpful to puzzled readers like myself than the naive comments of your leading article.—I am, etc.,

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Childhood Obesity and Carbohydrate Intolerance

SIR,—Having read with interest your leading article (21 July, p. 122) on this subject I read the instructions on the packets of rusks and cereals freely available in this area. I was shocked to see that one manufacturer recommends the addition of $\frac{3}{4}$ rusk dissolved in boiled milk to each bottle feed at 2 months, increasing to 1 $\frac{1}{4}$ rusks at 5-6 months. There is no suggestion that reference should be made to the health visitor or doctor. I wonder how many of us are allowing these products to be sold in our clinics without being aware of the instructions?—I am, etc.,

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Published Format of Clinical Trials

SIR,—Dr. G. Nyberg's letter (7 July, p. 47) prompts me to record my agreement with his general criticisms of published trials in

angina and to make similar criticisms of trials of antihypertensive therapy.

In the course of recently reviewing the published papers on the actions of the various beta-adrenergic blockers in angina and in hypertension I found 23 acceptable double-blind randomized controlled trials in hypertension, but in only 11 were the basic observations (as opposed to "processed data") included. The reader, therefore, is unable to ascertain for himself whether each patient was in fact hypertensive (some otherwise admirable studies have included patients with diastolic pressures less than 90 mm Hg on placebo!) and whether the hypertension was "controlled." I think most physicians would agree that our aim should be the achievement of a diastolic pressure of 90 mm Hg or less, at least in mild and moderate hypertension (W.H.O. stages 1 and 2), but such data are available only in 50% of published papers.

The score is approximately the same in angina. Of 30 double-blind controlled trials there was adequate definition of patients with individual data on attack rates, glyceryl trinitrate consumption, and exercise tolerance in only 12.

I suggest that much of the blame lies with the editorial policies of journals, many of which insist on "processed data" because of pressure on space (for which reason I do not have the temerity to ask you to publish the 50 or so references related to this letter) and the cost of tabulation. I would like, therefore, to plead with all editors not merely to accept but to demand that the basic variables be presented for publication and, if necessary, restrict statistical comments to the text.—I am, etc.,

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Lavatory Doors

SIR,—A 20-year-old epileptic patient of mine has just died in the lavatory at his home, after having a fit during which he fell against the lavatory door. The door had not been locked but as he was a heavy man and had fallen against the door it took his father half an hour to get through a window to release him. By this time he had died, presumably from asphyxia.

I have known people have heart attacks, pulmonary emboli, hypoglycaemic attacks, fainting after passing melaena stools, fainting after valsalva manoeuvres, miscarriages, and even babies in the lavatory. It is obvious that some of these emergencies are more likely to happen in the lavatory than anywhere else.

The old fashioned smallest room tended to be long and narrow and so there was always a good chance of getting the door open provided it was not locked. With the modern demand on space the lavatory is tending to be increasingly small, with a correspondingly greater chance of an unconscious person lying jammed against the door. I wonder whether architects could be persuaded in these circumstances to hang lavatory doors outwards, and also devise some mechanism rather than a conventional bolt so that they could easily be opened from the outside in an emergency.—I am, etc.,

M. D. BEGLEY

Frome