SIR,—The observations of Dr S K Majumdar and others (18 February, p 409) on serum prolactin levels after cimetidine therapy for 1-6 months do not alter or add to the conclusions of Delle Fave.¹

The mean of the serum prolactin concentration values quoted by the authors do not provide the mean quoted (170, 98, 280, 250, and 370 ÷ 5 is 233, not 167 mU/l as stated); and also the value for one patient was abnormal (370 mU/l compared with the stated upper normal limit of 360 mU/l) despite their assertion that there was no biochemical evidence of hyperprolactinaemia in any of their cases. In addition they have misinterpreted the other report of galactorrhoea after cimetidine² in which it was stated that a spectacular increase in serum prolactin concentration was associated with the administration of 1 g of cimetidine in the absence of any other drug treatment for three weeks previously.

Delle Fave studied two women and four men before and after treatment with cimetidine 1.6 g daily for two months. Their patients were selected in that one had gynaecomastia and one had galactorrhoea. In every case a rise in serum prolactin concentration occurred (from 50 to 112%), but in two patients values remained in the normal range after treatment.

Hyperprolactinaemia provides a rational explanation for gynaecomastia and galactorrhoea. Although some patients may have normal values after treatment with cimetidine, serum prolactin levels seem to be uniformly increased by this drug. To provide further information will require a larger number of patients studied before, serially during, and after therapy. This is of more than academic interest, since hyperprolactinaemia is associated with infertility in some women, and cimetidine is a very recent addition to the pharmacopoeia whose usefulness and toxicity are not yet clearly established.

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¹ Delle Favé, G F, *et al*, *Lancet*, 1977, **1**, 1319.

² Bateson, M C, *et al*, *Lancet*, 1977, **2**, 247.

Relapse of duodenal ulceration after cimetidine treatment

SIR,—Relapses of duodenal ulceration following the interruption of treatment with H₂ receptor antagonists have been observed,^{1,2} but no effective explanation has been reported.³ Previous studies prove that neither hypergastrinaemia nor rebound of acid secretion is involved.⁴⁻⁶

We have studied gastric dissolved mucin in the basal gastric secretion of 12 patients with duodenal ulcer (endoscopic diagnosis) before and after cimetidine treatment. Chemical separation of dissolved mucin in two different fractions was performed as described by Glass.⁷ After four weeks' treatment with cimetidine at a dose of 1 g daily we observed a highly significant ($P < 0.01$ by Student's *t* test) decrease in the concentration of neutral glycoprotein-mucoproteose complex (from a mean of 1.15 ± 0.15 (SEM) to 0.87 ± 0.12 g/l) and a parallel significant ($P < 0.01$) increase in that of acid mucoprotein complex (from 0.41 ± 0.08 to 0.61 ± 0.08 g/l) in the fasting gastric juice of all 12 patients. Two weeks after cessation of cimetidine treatment

measurement of the gastric mucin fractions was repeated in all patients, but no further significant changes were found (0.87 ± 0.12 and 0.62 ± 0.07 g/l respectively).

The inhibition of gastric acid secretion by cimetidine seems to induce alterations of great consequence in the composition of gastric dissolved mucin. A decrease of neutral glycoprotein-mucoproteose complex could mean a reduced protective effect of gastric mucus. That fraction, indeed, represents a group of neutral mucosubstances containing the bulk of the carbohydrates present in gastric juice (hesoxamine, hexoses, and fucose). One of the materials contained therein appears to have similar electrophoretic mobility to the endogenous inhibitor of gastric secretion (gastrophil).⁷ A reduced protective function of gastric mucus could possibly explain relapses when, after interruption of cimetidine treatment, gastric acid secretion returns to previous values.

A similar study in patients with gastric ulcer is in progress.

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¹ Saunders, J H B, and Wormsley, K G, *Lancet*, 1977, **1**, 765.

² Wallace, W A, *et al*, *British Medical Journal*, 1977, **2**, 865.

³ Bardhan, K D, *et al*, *Lancet*, 1977, **1**, 900.

⁴ Spence, R W, *et al*, in *Proceedings of the 2nd International Symposium on Histamine H₂ Receptor Antagonists*, Amsterdam, 1977, p 163.

⁵ Spence, R W, Celestin, L R, and McCormick, D A, *Gut*, 1977, **18**, A420.

⁶ Holden, R J, *et al*, *Gut*, 1977, **18**, A949.

⁷ Glass, G B J, *Annals of the New York Academy of Sciences*, 1967, **140**, 804.

Household products and poisoning

SIR,—I have read with interest the letter by Mrs Elspeth G Maclean (25 February, p 512). Regrettably, through the need for brevity, the detailed tabulations had to be omitted from our published paper (4 February, p 286). I will now see that these more comprehensive figures are sent to Mrs Maclean, when I think she will find that the second study did in fact cover household products in their entirety.

I am sorry, also, to read that my conclusions are regarded as "misleading." Nevertheless, I am convinced that countless people, in all good faith, have exaggerated the risks of so-called "poisoning" from household products. After 15 years of close study of this subject clinically my colleagues and I continue to be alarmed at the dangers that can arise to small children from medicines that are still carelessly left about the house. Repeatedly we are negotiating with the pharmaceutical industry and other parties in an effort to contain this evil, and I know that the organisation represented by Mrs Maclean is alert to this need. There is, after all, no earthly reason why medicines need ever be left within reach of small children. Household products, though, are different. To my mind it becomes a practical impossibility to run a home reasonably without the appropriate requisites being conveniently placed rather than stored out of reach, and it is then virtually impossible to prevent youngsters getting their hands or mouths to them. So if doctors, both in general practice and in hospitals, accept our findings they can at once allay the anxieties of parents at the outset and minimise treatment to the

benefit of all concerned. That was the essence of our paper.

If, on the other hand, parents become disproportionately alarmed about household products, then the attention of these adults may be distracted from the other hazards of the domestic scene that are likely to be both more numerous and more serious, as the recently published first report by the Department of Prices and Consumer Protection on the "Home Accident and Surveillance System" makes all too clear.

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Regional secure units and interim security for psychiatric patients

SIR,—In his comprehensive review of regional secure units (25 February, p 489) Dr Robert Buglass quotes the late Dr Peter Scott's proposal that psychiatric units in prison might be more fully exploited. I would support this idea and feel sure that its wide application would meet many of the problems which extend far beyond the question simply of security. The dreary, dehumanised, and therapeutically unproductive atmosphere of many penal and correctional institutions can be changed only by a vigorous approach to the total milieu, the roles given to both inmates and staff, and the relationships between them. My experience of prisons in Scotland and the USA—and particularly Edinburgh prison in the 1960s—convinced me of the widespread effect a psychiatric unit can exert on the prison culture if it has clear goals, the co-operation of senior prison officials, and is around long enough to consolidate the changes it effects.

In Scotland there has been for long a scheme of "visiting psychiatrists," whereby NHS personnel have sessions in prisons, borstals, and list D schools. This gives the opportunity—particularly where forensic psychiatry units exist—to provide a system of comprehensive assessment and realistic placement and treatment of inmates, whether it be in prison, hospital, or community. But what has been sadly lacking is a willingness to experiment and evaluate fresh ideas and methods. The recent exaggerated response to Barlinnie's special unit (which is based on the principles of a therapeutic community) shows how slowly innovation must move and the irrational anxiety and hostility that are so easily provoked.

It seems to me that people, whether prisoners or patients, should not be admitted to hospital care unless they require treatment which cannot be given elsewhere. Prisons are secure places. What they need now is more opportunity and expertise in fulfilling their different roles of containment, treatment, and rehabilitation.

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SIR,—Dr Robert Buglass merits congratulations for his excellent review of this subject (25 February, p 489). However, I would question his assertion that "offender patients and difficult non-offender patients" were easily accommodated in mental hospitals before the Mental Health Act of 1959. Then patients were in grossly overcrowded con-