taking certain words out of context and introducing concepts which go beyond the original. They also say that "corticosteroids are not particularly effective" in psoriasis. Many published articles, and letters from patients who have obtained rapid relief of their psoriasis after sometimes 25 or even 30 years' fruitless use of alternative remedies, attest the contrary. The therapy cannot be described accurately as "generally unacceptable" when it is apparent that so many patients are satisfactorily controlled with topical corticosteroids.

The advertisement for the use of Dermovate in psoriasis is fully supported by published clinical evidence and several years of clinical experience. Taken in context, the advertisement actually says that "treatment need be of short duration only; and provided dosage does not exceed 50 g weekly, the risk of steroid side-effects is *minimal*" (our italics). In these words the advertisement gives well-supported^{1,2} guidance on the limits to be observed. It also strongly and repeatedly refers to short-term therapy ("a matter of days")—a point which your correspondents ignore in their reference to prolonged usage.

The advertisement also refers to "long periods of remission," not "prolonged" as cited and criticised by your correspondents. It has been demonstrated³ that Dermovate can produce long periods of remission which may on occasion exceed those produced by dithranol.⁴

We are well aware of the need to minimise the adverse effects of potent topical steroids by stressing their correct use. We firmly believe that the advertisement does this and therefore renders a useful service to doctors treating patients with psoriasis.

C H DASH
A H GOODSPEED
C G SPARKES

Medical Department,
Glaxo Laboratories Ltd,
Greenford, Middx

¹ Carruthers, J A, *et al*, *British Medical Journal*, 1975, **4**, 203.

² Allenby, C F, *et al*, *British Medical Journal*, 1975, **4**, 619.

³ Floden, C H, *et al*, *Current Medical Research and Opinion*, 1975, **3**, 375.

⁴ Marriot, P J, and Munro, D, *British Journal of Dermatology*, 1976, **94**, suppl 12, p 101.

Continued use of hormonal pregnancy tests

SIR,—We were interested in the letter from Dr C L Brewer (18 February, p 437), as in Belgium also we found evidence for the continued use of hormonal pregnancy tests (HPTs), despite official warnings.

The 500 women who were delivered between 25 March and 3 June 1977 in the three hospitals of Bruges were interviewed about the use of medication during their pregnancy. Twenty of them (4%) had received HPTs (for 16 prescribed by general practitioners and for three by obstetricians; one woman purchased it without prescription from her pharmacist). Six of the 20 women (30%) were not married at the time of conception, contrasting with 6% for the whole group. One child whose mother had taken an HPT was born with oesophageal atresia.

Belgian physicians were warned about the hazards of HPTs by official letter in June 1975 and details were communicated to all physicians and pharmacists in the *Folia Pharmacotherapeutica*, an independent in-

formation bulletin, in January 1975 and in February 1976. We agree with Dr Brewer that warnings are not sufficient. In November 1977 the Belgian health authorities took the decision to withdraw HPTs from the market.

F MEIRE
K VUYLSTEEK

Department of Social Medicine
and Hygiene,

W BUYLAERT
M BOGAERT

Heymans Institute of Pharmacology,
University of Ghent Medical School,
Ghent, Belgium

* * * Primodos, the only preparation on the British market capable of being used as a hormonal pregnancy test, has now been withdrawn by the manufacturers—Ed, *BMJ*.

Cysteamine or N-acetylcysteine for paracetamol poisoning?

SIR,—We were interested to read the article by Dr Jennifer M Smith and others (11 February, p 331) on the treatment of paracetamol poisoning with cysteamine and would like to comment on several points. It is unfortunate that confusion should be added by the adoption of the term mercaptamine in place of the universally accepted cysteamine. Nor do we share the enthusiasm of Dr Smith and her colleagues for the use of this toxic compound later than 10 h after ingestion. The claims made for its efficacy in late treatment cannot be supported by their data.

Although eight patients treated after 10 h escaped severe liver damage, it is impossible to assess how severely they were poisoned, because the non-specific acid hydrolysis method of Welch and Conney¹ was used to estimate the plasma paracetamol concentrations. This method also measures non-toxic paracetamol sulphate and glucuronide conjugates and other compounds which can be hydrolysed to *p*-aminophenols. In patients without liver damage the plasma concentrations of paracetamol conjugates greatly exceed those of the unchanged drug after 10 h,² and there may be as much as a 4- to 5-fold difference. The initial plasma paracetamol concentrations reported by Dr Smith and her colleagues in these eight patients must therefore have been gross overestimates of the unchanged paracetamol and in most treatment was probably unnecessary. Since the complex relationship between plasma concentrations of "total" paracetamol and liver damage has not been defined, the conclusions drawn by the authors cannot be justified. There is no need to use this non-specific method, since several simple and rapid methods for unchanged paracetamol have been published,³⁻⁵ all of which should be within the capability of clinical chemistry laboratories.

If cysteamine were innocuous the unnecessary treatment of large numbers of

patients not at risk of severe liver damage might be acceptable. But cysteamine has unpleasant, protracted, and potentially lethal toxic effects⁶⁻¹⁰ and its indiscriminate use in patients who do not need it is indefensible.

From our experience over the last five years we are convinced that intravenous N-acetylcysteine (Airbron) is now the treatment of choice. We have given it to 49 patients with admission plasma concentrations of unchanged paracetamol above a treatment line joining semilogarithmic plots of 200 mg/l at 4 h and 50 mg/l at 12 h as previously described.¹¹ Of 29 patients treated within 10 h of ingestion, only one developed severe liver damage (alanine or aspartate aminotransferase >1000 IU/l), none developed renal failure, and there were no deaths from hepatic failure.

Our experience of the late treatment of severe paracetamol poisoning with cysteamine and N-acetylcysteine in 33 patients clearly shows that these agents are ineffective when given later than 10 h after ingestion, since the incidence of severe liver damage, renal failure, and death was similar to that observed in 52 patients receiving supportive therapy only (see table below). In all patients the admission plasma paracetamol concentration was above the treatment line mentioned above.

Compared with cysteamine, N-acetylcysteine has negligible side effects and has the additional advantage of ready availability as a sterile solution. Although not recommended for intravenous use by the manufacturer, we have not encountered any problems and in our opinion it is infinitely preferable to "home made" solutions of cysteamine prepared from the ordinary chemical laboratory reagent.

L F PRESCOTT
M J STEWART
A T PROUDFOOT

Regional Poisoning Treatment Centre
and Department of Clinical
Chemistry,
Royal Infirmary,
Edinburgh

¹ Welch, R M, and Conney, A H, *Clinical Chemistry*, 1965, **11**, 1064.

² Prescott, L F, and Wright, N, *British Journal of Pharmacology*, 1973, **49**, 602.

³ Routh, J I, *et al*, *Clinical Chemistry*, 1968, **14**, 882.

⁴ Glynn, J P, and Kendal, S E, *Lancet*, 1975, **1**, 1147.

⁵ Dordoni, B, *et al*, *British Medical Journal*, 1973, **2**, 86.

⁶ Prescott, L F, *et al*, *Lancet*, 1974, **1**, 588.

⁷ Prescott, L F, *et al*, *Lancet*, 1976, **2**, 109.

⁸ Douglas, A P, Hamlyn, A N, and James, O, *Lancet*, 1976, **1**, 111.

⁹ Williams, R, and Davis, M, *Acta Pharmacologica et Toxicologica*, 1977, **41**, suppl 2, p 300.

¹⁰ Crome, P, *et al*, *Lancet*, 1976, **2**, 829.

¹¹ Prescott, L F, *et al*, *Lancet*, 1977, **2**, 432.

* * * A copy of this letter was sent by the authors to Dr Smith and her colleagues, whose reply is printed below.—Ed, *BMJ*.

SIR,—We agree about the term mercaptamine. It was imposed upon us by editorial policy in place of the more commonly used cysteamine.

We have now adopted the method of Glynn and Kendal,¹ which measures "free" paracetamol. This method had not been described at the beginning of our study. The other methods

Results of treatment of severe paracetamol poisoning

Treatment	No of patients	Mean ingestion-treatment interval (h)	No with severe liver damage	No with renal failure	No of deaths
Supportive only	52		31 (60%)	6 (12%)	3 (6%)
N-acetylcysteine within 10 h	29	7.7	1 (3%)	0	0
N-acetylcysteine after 10 h	20	15	14 (70%)	3 (15%)	1 (5%)
Cysteamine after 10 h	13	13.5	8 (62%)	1 (8%)	1 (8%)