Leeds Scales need further study since they are affected by base-rates of disorder in a sample studied (Gathercole, 1968). The GHQ has been looked at to some extent from this point of view (Tarnopolsky *et al*, 1979).

Acknowledgements

We would like to thank Dr Keith Hawton, Oxford University Department of Psychiatry and Mr Ralph McGuire, Edinburgh University Departments of Psychiatry and Psychology, who kindly advised on procedure and analysis, and express our appreciation to Mrs Linda Pollock for administrative work in connection with this small project.

GILLIAN FORREST

The Park Hospital for Children, Old Road, Headington, Oxford

IAN BERG

Department of Psychiatry, University of Leeds

References

GATHERCOLE, C. E. (1968) *Assessment in Clinical Psychology*. Hammondsworth: Penguin Books.

GOLDBERG, D. P. (1972) *The Detection of Psychiatric Illness by Questionnaire*. London: Oxford University Press.

—— & HILLIER, V. (1979) A scaled version of the General Health Questionnaire. *Psychological Medicine*, **9**, 139–45.

HAMILTON, M. (1959) The assessment of anxiety states by rating. *British Journal of Medical Psychology*, **32**, 50–5.

—— (1967) Development of a rating scale for primary depressive illness. *British Journal of Social and Clinical Psychology*, **6**, 278–96.

MAXWELL, A. E. (1977) Coefficients of agreement between observers and their interpretation. *British Journal of Psychiatry*, **130**, 79–83.

SNAITH, R. P., BRIDGE, G. W. K. & HAMILTON, M. (1976) The Leeds Scales for the self assessment of anxiety and depression. *British Journal of Psychiatry*, **128**, 156–68.

TARNOPOLSKY, A., HAND, D. J., MCLEAN, E. K., ROBERTS, H. & WIGGINS, R. D. (1979) Validity and uses of a screening questionnaire (GHQ) in the community. *British Journal of Psychiatry*, **134**, 508–15.

PROLACTIN RESPONSE TO NEUROLEPTIC CHALLENGE

DEAR SIR,

Shur and Checkley (*Journal*, April 1982, **140**, 431–32), commenting on our paper concerning prolactin responses during neuroleptic treatment (*Journal*, November 1981, **139**, 400–4) question our "unconventional" view that the degree of dopamine receptor blockade required for therapeutic effect is below that which produces a maximal prolactin response. The main reason for their objection is that "the paper does not establish that the patients had responded to neuroleptic treatment at a time when they demonstrated only partial blockade of pituitary dopamine receptors". However, we have indicated (both in the text and in the first part of the questioned sentence in the summary) that in several patients their current medication was clinically effective whereas prolactin elevation was not maximal, as shown by further prolactin rise following the test dose of haloperidol (2 or 4 mg i.m.). Perhaps we should have added that clinical improvement had occurred in these patients by the time of testing or earlier and that their plasma prolactin following haloperidol challenge was markedly higher than the levels found in samples taken weekly throughout the treatment. The Brief Psychiatric Rating Scale and a global 4-point clinical scale were used to assess week-to-week changes in symptoms.

The other point of the letter—that haloperidol 1.0 mg i.v. produced no further prolactin rise in two manic subjects tested by Shur and Checkley after one and three weeks of treatment with oral haloperidol—does not contradict our results and does not indicate that in *all* patients maximal blockade in the pituitary precedes clinical response. Moreover, it is possible that a higher test dose would have produced prolactin rise in their subjects: we have reported earlier that in some patients prolactin responded during neuroleptic treatment to chlorpromazine 100 mg i.m. but not to 50 mg (Kolakowska *et al*, 1981).

Finally, there is not much evidence for the "conventional" view that the resting prolactin levels do reach *maximum* before the clinical effects of neuroleptics appear. Thus, although in patients studied by Cotes *et al* during treatment with flupenthixol (quoted in the letter), prolactin elevation preceded improvement, as it usually does, there was no indication whether or not this elevation was *maximal*. On the other hand, the reports showing a graded prolactin response to relatively high daily doses of neuroleptics and a rise in prolactin following morning doses of medication during effective treatment (references on request) speak against the view which became conventional without being properly tested.

TAMARA KOLAKOWSKA

The University of Chicago, 950 East 59th Street, Chicago, Illinois 60637, USA

Reference

KOLAKOWSKA, T., FRASER, S., FRANKLIN, M. & KNOX, J. (1981) Neuroendocrine tests during treatment with neuroleptic drugs. I. Plasma prolactin response to chlorpromazine challenge. *Psychopharmacology*, **72**, 283–5.