LEEDS SCALES AND THE GHQ IN WOMEN WHO HAD RECENTLY LOST A BABY

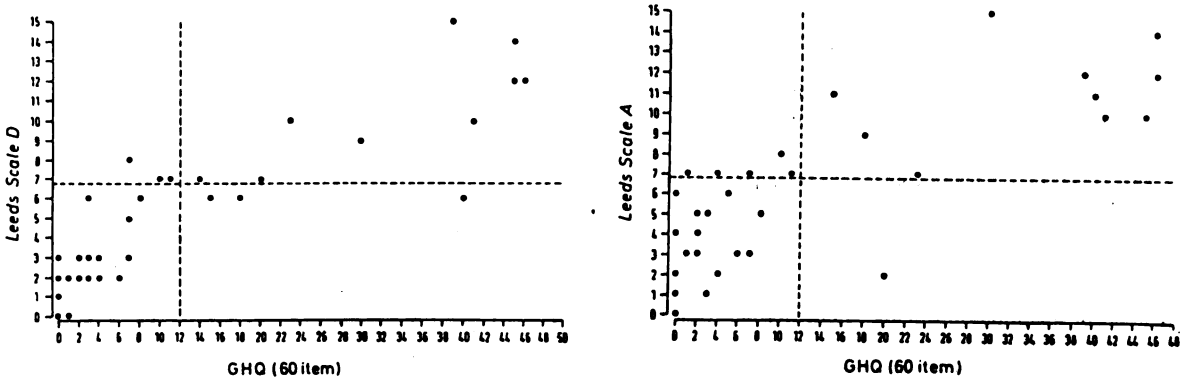
DEAR SIR,

Your readers may be interested in hearing about a comparison between two self-administered scales which can be used for the estimation of minor psychiatric disorder in the community. Thirty-four out of a group of 50 consecutive patients at the John Radcliffe Maternity Hospital in Oxford who had lost a baby in the previous six months agreed to complete the 60 item GHQ—the General Health Questionnaire (Goldberg, 1972) and the Leeds Scales (Snaith, Bridge and Hamilton, 1976). The GHQ was scored in two ways as a bimodal response scale and also by the Likert method to produce four subscales using 28 of the items (Goldberg and Hillier, 1979). The Leeds Scales were scored so as to produce general scale scores which have been recommended for case finding (Snaith *et al*, 1976). Cut-off points used to identify possible psychiatric cases were 12 or more out of 60 on the GHQ (Goldberg, 1972) and 7 or more on either the D (depression) and A (anxiety) Leeds Scales (Snaith *et al*, 1976).

The mean age of the women was 28.3 years (SD = 5.3, range 19 to 40). The mean 60 item GHQ score was 13.3 (SD = 15.7, range 0 to 46). The mean D scale score of the Leeds Scales was 5.4 (SD = 4.0, range 0 to 15). The mean A scale score of the Leeds Scales was 6.3 (SD = 4.1, range 0 to 15). The 28 item GHQ, A subscale had a mean score of 4.8 (SD = 4.2, range 1 to 16), B subscale had a mean score of 7.0 (SD = 5.4, range 0 to 20), C subscale had a mean score of 7.6 (SD = 2.9, range 4 to 16) and the D subscale had a mean score of 3.8 (SD = 4.8, range 0 to 18). Product-moment correlation coefficients were as follows: Leeds D scale and 60 item GHQ bimodal response score .87, 28 item GHQ Likert subscales A 'somatic' .80, B 'anxiety-insomnia' .87, C 'social dysfunction' .75 and D 'severe depression' .78. Leeds A scale and 60 item GHQ .78, 28 item GHQ A .71, B .88, C .45 and D .63. All were highly significant ($P < 0.01$).

Using the recommended cut-off points individuals were compared using scatter plots and also the random coefficient of agreement (Maxwell, 1977). With regard to the 60 item GHQ and Leeds A scale, R.E. = .6, $P_1 = .2$ and $P_0 = .4$. In 11 instances the same individuals were above the cut-off points on both scales and in 17 instances below. Five women had scores above cut-off on the A scale but not on the GHQ, and one was above cut-off on the GHQ but not on the A scale. In respect of the GHQ and D scale, R.E. = .7, $P_1 = .2$ and $P_2 = .5$. Nine individuals were above both cut-off points and 19 were below. Three women had scores above cut-off on the D scale but not on the GHQ, and three were above cut-off on the GHQ but not on the D scale. D and A scales correlated significantly ($r = .7$, $P < .001$). A regression equation with the D scale as independent and 60 item GHQ as dependent variable produced a model which accounted for 76 per cent of the variance of GHQ scores ($P < .001$). A similar equation using the A scale accounted for 61 per cent of the variance of GHQ scores ($P < 0.001$).

The Leeds Scales are derived from the Hamilton Rating Scales for Depression (Hamilton, 1967) and Anxiety (1959). The form is very easy to fill in and it seems could probably be used to estimate minor degrees of psychiatric disturbance in a non-psychiatric population. The finding that A and D scores were highly correlated is not surprising since symptoms of anxiety and depression tend to be associated in the kind of cases studied (Goldberg and Hillier, 1979). This association tended to blur their relationship with 28 item GHQ subscales. The cut-off points for the



FIG—Thirty-four young women who had recently lost a baby. Product moment correlation coefficients (r) between their scores on the GHQ and Leeds Scales.

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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).

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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.

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