gain insight into their lives, situations and conditions. This, I would argue, involves allocating responsibility for the condition to the patient and may not resemble the treatments of physical medicine. The reader will note that this conclusion is congruent with that of other students of alcoholism, such as Orford and Edwards (quoted on page 452 of my article), whose "primary clinical experience and responsibility prior to plunging into clinical research" can certainly not be doubted. However, given that such corroborative evidence has emanated from "those commanding the heights (of) Denmark Hill" I fear that Dr Macdonald will remain unconvinced.

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VALIDITY AND USES OF THE GHQ

DEAR SIR,

In your May issue, Tarnopolsky *et al* report (*Journal*, May 1979, **134,** 508–15) on the validity of the GHQ in a community sample. They find lower validity than has been reported in samples of general practitioner patients. This finding might be the result of a feature of their research design. The type of illness measured by the GHQ is often quite fleeting. Thus it has been reported that the correlation between GHQ score and total score on the Present State Examination is .8 when the PSE is conducted within a week of the GHQ, but drops below .5 for a longer interval (Duncan-Jones and Henderson, 1978, p. 235). It is clear there was an interval between the GHQ and the validatory psychiatric interview in Tarnopolsky's study, but the length of that interval is not indicated. Since a matching design was used, the interval cannot have been trivial. There was no such interval in the general practitioner studies. Therefore this difference in design might account for the lower validity.

In presenting their data on screening, Tarnopolsky *et al* make the important point that their data for approximately equal numbers of high scorers and matched low scorers give biased estimates of 'sensitivity' and 'specificity' for the community population, and correct for this by weighting up the low scorers. This would be valid and appropriate if their low scorers were a representative sub-sample of all the low scorers in their original sample. But since they were elaborately matched to the high scorer group, this cannot be so.

It seems possible that the use of matching has weakened this study in two ways. It is feasible to pre-allocate respondents to different sub-sampling classes (prior to first interview) so that (a) subjects for the second phase interview are selected randomly but with probability of selection being dependent on GHQ score, and (b) the first-phase interviewer can determine whether or not a second-phase interview is required, and make a tentative appointment for it. Using this procedure, one can keep the interval between interviews short, and make valid estimates for the whole population from the second phase interview. Details are given in Henderson *et al* (in press).

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TARDIVE DYSKINESIA AND DEPOT FLUPHENAZINE

DEAR SIR,

I read with interest Dr Nasrallah's letter (*Journal*, May 1979, **134,** 550) in which he suggested that tardive dyskinesia in patients maintained on depot fluphenazine could be caused by irregular release of fluphenazine from the intramuscular depot. In an earlier study (Nasrallah *et al*, 1978) he and his colleagues had found wide fluctuations in plasma fluphenazine concentrations in 10 patients during 2 weeks following a 50 mg injection of fluphenazine decanoate: varying numbers of fluphenazine peaks occurred at random, separated by periods in which little or no drug could be detected. (Their analytical procedure, gas-liquid chromatography, could measure fluphenazine concentrations above 3 ng/ml). Dr Nasrallah went on to propose that during depot fluphenazine treatment the decline in plasma fluphenazine levels which followed intermittent peaks could act like a drug withdrawal to cause dyskinesia by producing dopaminergic receptor hypersensitivity.

We have also examined plasma fluphenazine levels in patients receiving fluphenazine decanoate (Wiles and Gelder, 1979). We used a different analytical technique, a radioimmunoassay, which can measure down to 0.05 ng/ml (Wiles and Franklin, 1978). In our study, 33 subjects were receiving chronic treatment with a wide range of doses (12.5 to 150 mg) given at intervals of 1–5 weeks. Our results differ

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