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days when trained nursing staff in institutions for these patients are getting fewer, it is important that any recording method is reliable but not timeconsuming. This scale is now seen to fulfil these criteria.

No claim is made by the authors that the scale could not be improved, nor that it could not be completed during more regular intervals during the 24 hours, giving a more complete record of a patient's behaviour. Clearly it could be so used, and this would answer one of the points in the letter criticising the trial's design (*Journal*, November 1987, **151**, 705– 706). Any scale, no matter how unsophisticated it may appear and even be, is better than none and I, for one, am grateful for the efforts undertaken by Dr Craft and his colleagues in proving the validity of it despite its imperfections. Included in my gratitude are the unnamed nurse assessors who administered the scale during both its verification and use.

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## Peer-Group Support for Patients in the Community

SIR: Ford *et al* (Journal, October 1987, **151**, 479–485) make a valuable statement when they claim that resettlement of "those (patients) currently remaining in hospital will require increasingly extensive provision". However, it is difficult to equate the variables they have measured with the resources and handicaps the patients actually have.

As an illustration of this criticism, the authors have determined how many patients go outside the hospital and how many have visitors. In my experience, patients with no contacts outside the hospital may still have valuable friendships within the hospital. For example, some patients, while not legally married, form stable heterosexual partnerships which are loving and supportive. In Sheffield, longstay patients are being resettled in the community in social groups. This appears to be successful in that it makes the move less frightening and helps reduce post-discharge loneliness. Peer-group support gives the patients the confidence to become friendly with local people in the street, shop, and public house. Several patients for whom previous discharge plans broke down have been enabled to live outside this way.

Interested relatives and friends outside the hospital can provide valuable support; however, even patients without them should not necessarily be considered friendless. As patients left in hospital have fewer supports and resources it is important that we mobilise the ones they do have to best effect.

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# P3 and CT Scan in Patients with Chronic Schizophrenia

SIR: We read with interest the paper by Romani *et al* (*Journal*, October 1987, **151**, 506–513). Using a similar auditory discrimination task in a group of 21 DSM-III diagnosed schizophrenic out-patients and short-term in-patients (Ebmeier *et al*, 1987), we also found reduced P3-amplitudes in patients compared with age-matched controls. Neither history of potential perinatal brain damage, nor – as found by Romani *et al* – psychiatric family history was correlated with P3-amplitude or P-latency within the schizophrenic group. For our patients, calculated means of P3-latencies were about 20 ms longer than for controls, although this difference did not reach significance if P3 was defined as the largest peak between 260 and 450 ms post-stimulus.

Increased latencies of P3 have been reported only by a small sub-group of investigators, most recently by Blackwood et al (1987). Baribeau-Braun et al (1983) incidentally do not report increased latencies, as suggested by Romani et al. Possible additional confounding factors will therefore have to be considered. The obvious one is that schizophrenic patients agreeing to ERP studies are a (self-)selected group which might differ from study to study. Romani et al's patients were receiving "monotherapy" with haloperidol. In the absence of a disclaimer it has to be assumed that anticholinergic drugs were prescribed to at least some of the patients. Callaway (1984) described an increase of P3-latency after scopolamine, and in our patient group P3-latency was correlated significantly with dose of anticholinergic medication (Kendall's  $\tau = 0.48$ , P < 0.01). We agree with Romani et al that neuroleptic medication is unlikely to account for P3 changes. Blackwood et al (1987) found no intra-subject differences before and after initiation of neuroleptic drug therapy, and for our patients there was no correlation between dose of neuroleptic (in chlorpromazine units) and P3-latency or amplitude. Pfefferbaum et al (1984) did not report an absence of a neuroleptic drug effect on ERPs as suggested by Romani et al.

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From our own research and from the literature (e.g. Saletu et al, 1971) we get the impression that P3 amplitude and latency data tend to give a false sense of accuracy. This is also apparent from Romani et al's comment that "N2 was [defined as] the greatest negative peak between P2 and P3". In our own study the schizophrenic group was characterised by the presence of between 1 and 4 positive maxima in the latency range of 260-450 ms. Thus, schizophrenic patients do not only show a larger intertrial and intersubject variability of P3-latency (Pfefferbaum et al. 1984), but also multiple peaks and a variability of waveforms which makes P3 definition a rather arbitrary exercise. Romani et al do not give a clear operational definition of P3, which would be necessary to test their results.

There can be no doubt that the 'late positive complex' is abnormal in many schizophrenic patients. This abnormality might not be described adequately by "P3-latencies and amplitudes", and the analogous interpretation of P3 parameters along functional correlates derived from normal populations are at best hypothetical, at worst misleading. It is hoped that clinical correlative studies like Romani *et al* will shed some light on the significance of these changes.

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SIR: We agree with Dr Ebmeier and colleagues that, within such a broad diagnostic category as schizophrenia, different research findings may easily be due to differences in the patient samples.

All of our patients were long-stay hospitalised schizophrenics, and differed therefore from those of most previous ERP studies, including that of Ebmeier et al (1987). In our study the main finding concerning ERPs was, apart from correlative aspects, the increase of P3 latency of about 40.50 ms depending on the paradigm. Anticholinergic medication, which according to some authors may be related to increased P3 latencies, was not involved in our study, and our use of the expression "monotherapy" excluded all kinds of psychoactive drugs with the exception of haloperidol. Neuroleptic drugs themselves, at least at the commonly used dosages, probably do not affect P3 latency (Blackwood et al, 1987). Pfefferbaum et al (1984), who examined patients both on and off neuroleptic medication, did not report any difference (as we stated), and in fact did not find differences, at least in that patient group (discussion of Pfefferbaum's paper at EPIC 7 conference, Florence, 1983).

Nevertheless, different results may not exclusively arise from differences directly or indirectly related to the patients, but also from differences in methodological recording and scoring techniques. It is undoubtedly true that in some cases problems may arise in the identification of late components. We believe that one of the main reasons for the presence of bifid or multiple peaks is the increased latency variability (Pfefferbaum et al, 1984). In such cases we adopted the rules suggested by Goodin et al (1978), which have been reported in our normative study (Romani et al, 1986). However, in our opinion the presence of "multiple peaks and variability of waveforms" are a challenge to the neurophysiologist for the development of more sophisticated techniques of signal analyses. Variability between single trials may, in our experience, be partially controlled by a selective averaging technique by monitoring spontaneous vigilance fluctuations (Romani et al, 1987).

Other techniques, which completely abandon the hypothesis that brain responses are stationary (Westerkamp & Aunon, 1987), may be also particularly useful.

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