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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Alcohol and Ageing

SIR: Nordstrom & Berglund (*Journal*, September, 1987, **151**, 382–388) fail to provide vitally important information about what would otherwise be one of the few tangible pieces of information resulting from their follow-up study of male alcoholics. They state that they wish to examine the issue of ageing and recovery from alcoholism. However, in their final analysis of 45 males selected from an original mixed-sex sample of 1312 patients, they state, "The main finding of the present study was that the processes of improvement differed between older and younger alcoholics. Improvement in older subjects was related to a pattern of gradual change from abuse to social drinking." The authors examine three possible physiological mechanisms to explain their observations: two of these they were only able to speculate about in their patients, and the third – the possibility of liver damage causing decreased alcohol tolerance and a gradual reduction in alcohol consumption – they dismiss with the bland statement that "while subjects with pathological liver function blood tests in our sample were typical abusers, there was no indication of liver damage in the improved subjects". What evidence do the authors have that their improved subjects did not have liver damage? It is well-recognised that alcoholic liver damage may be present in the reformed non-abusing patient even if the routine laboratory liver function tests are normal. The only way to be sure whether or not the patient has suffered permanent liver damage is by histopathological examination of a liver biopsy (Sherlock, 1985).

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SIR: As is correctly pointed out by Dr Roberts, we cannot exclude the possibility of liver damage in the absence of pathological test results in our subjects. Apart from liver function blood tests at the follow-up, however, we also studied all psychiatric and several somatic hospital case records concerning our subjects. We were interested, among other things, in data concerning somatic complications. In short, we found no evidence of liver damage in the group of subjects referred to in our paper as improved, whereas some of the subjects with an unsuccessful course had been under medical treatment for liver damage. Although this does not prove anything about absence or presence of liver damage in any of our subjects, we still feel that, on a group level, our suggestion that "severe liver disease. . . does not seem to be a plausible explanation for improvement in the present sample" is reasonable and justified.

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Constant Current vs Constant Voltage ECT Devices

SIR: Railton *et al* (*Journal*, August 1987, **151**, 244–247) report a comparison of various electrical parameters measured during clinical use with Ectron Duopulse Mark 4 and Ectron Series 2A ECT devices. They then go on to interpret their findings on the basis of differences in the mode of stimulus delivery between these two machines, i.e. constant voltage for the former and constant current for the latter. In fact, the mode of stimulus delivery is only one of two major differences between these two machines, the other being stimulus waveform: partial sine wave for the Duopulse and brief pulse for the Series 2A devices.

Various investigations have demonstrated that the sine wave stimulus requires several times more stimulus energy and charge to produce a seizure than does the pulse stimulus (Weiner, 1980). By extrapolation, it is reasonable to assume that the pulse stimulus is also more efficient in inducing seizures than the 60% sine wave stimulus used in the Ectron Duopulse. This difference in seizure threshold, which appears to be independent of mode of stimulus delivery, therefore means that in order to assure the occurrance of