

- GOODIN, D. S., SQUIRES, K. C., HENDERSON, B. H., STARR, A. (1978) Age-related variations in evoked potentials to auditory stimuli in normal human subjects. *Electroencephalography and Clinical Neurophysiology*, **44**, 447–458.
- PFEFFERBAUM, A., WENWGRAT, B. G., FORD, J. M., ROTH, W. T. & KOPELL, B. S. (1984) Clinical application of the P3 component of event-related potentials. II. Dementia, depression and schizophrenia. *Electroencephalography and Clinical Neurophysiology*, **59**, 104–124.
- ROMANI, A., MARIOTTI, G. & COSI, V. (1986) The effect of aging on the P3 component in different auditory paradigms. *Revue d'Electroencephalographie et de Neurophysiologie Clinique*, **16**, 423–431.
- , TAVA, G., APIH, G., COSI, V. (1987) Some relationships between spectral EEG parameters and late AEPs. *Bollettino della Societa Italiana di Biologia Sperimentale* (in press).
- WESTERKAMP, J. J. & AUNON, J. I. (1987) Optimum multielectrode a posteriori estimates of single-response evoked potentials. *IEEE Transactions on Biomedical Engineering*, **34**, 13–22.

### 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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### Reference

- SHERLOCK S. (1985) Alcohol and the liver. In *Diseases of the Liver and Biliary System* (ed. Dame S. Sherlock), p 355. London: Blackwell Scientific Publications.

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 occurrence of