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REPLY: Well designed studies that control for stimulus intensity do not support the suggestion that bilateral ECT leads to either longer convulsions or cerebral seizure activity measured by electroencephalogram than unilateral ECT (Sackeim *et al*, 1987; 1993). The study by Abrams *et al* (1973) concerned multiple monitored ECT where three or four electrical stimulations were applied about two minutes apart during prolonged anaesthesia and is of uncertain relevance to the contemporary practice of ECT. Weiner (1980) did not report original data to support the suggestion that bilateral ECT leads to longer cerebral seizure activity.

Bilateral ECT with the bifrontotemporal placement is associated with a higher seizure threshold than right unilateral ECT with d'Elia's placement at the outset of treatment and throughout the course of treatment. The original data in support of this statement and possible explanations are given in the reference cited in the accompanying handbook (Sackeim *et al*, 1991).

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Late-life depressive disorder

SIR: While Van Ojen *et al* (1995) make use of large numbers of subjects and use the AGE-CAT diagnostic system, which is to be commended, there do appear to be a number of drawbacks to their study.

Firstly, one has to question the validity of MMSE scores of less than 26. This is at best a fairly crude screening tool for evaluating cognitive function. Furher *et al* (1992) conclude their study by stating that functional impairment has "a basic confounding effect on cognitive state" as measured by the MMSE in depression in elderly subjects. The CAMDEX schedule does contain sections relating to functional ability, but in this study, only part of it was used.

Van Ojen *et al* also state that a correlation exists between severity of depressive symptoms and cognitive function, but this was only significant in male subjects in the Furher *et al* study. A further area of concern would be the omission of the prevalence of prescribed medication; 80% of subjects over 65 regularly take prescribed medication, and the effect of this cannot be ignored in relation to cognitive function. Similarly, ethnicity and level of anxiety would also be sources of error in the MMSE.

To assume that depression in old age, in the absence of past psychiatric history, is primarily due to "prodromal" Alzheimer's disease appears to be a gross over-simplification. From the organic point of view Rabins *et al* (1991) demonstrated diffuse subcortical abnormality on the MRI in depressed subjects, but concluded that this was unlikely to be an early dementing process, and that it is likely that there are organic changes specific to depression.

FURHER, R., ANTONUCCI, T., GAGNON, M., *et al* (1992) Depressive symptomatology and cognitive functioning: an epidemiological survey in an elderly community sample in France. *Psychological Medicine*, **22**, 159–172.

RABINS, P., PEARLSON, G., AYLWARD, E., *et al* (1991) Cortical magnetic resonance imaging in elderly inpatients with major depression. *American Journal of Psychiatry*, **148**, 617–620.

VAN OJEN, R., HOOIJER, C., BEZEMER, D., *et al* (1995) Late-life depressive disorder in the community I & II. *British Journal of Psychiatry*, **166**, 311–319.

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AUTHORS' REPLY: Among elderly community members, impairments in the functional, cognitive, and effective realm may be entangled. In order to disentangle the interrelationships our research group has recently commenced the analysis of follow-up data describing groups of elderly subjects with either functional or cognitive impairment, depression and anxiety, combinations of these or no

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