illness in old age (*Journal*, August 1989, **155**, 147–152). We also studied the use of ECT in elderly depressive patients (Malcolm & Peet, 1989), but reached somewhat different conclusions.

Evidence is far from conclusive that ECT is the most effective form of treatment for elderly depressed patients. There are few, if any, methodologically satisfactory controlled trials of ECT conducted specifically in elderly populations. Available data from open, uncontrolled trials reveal widely varying response rates, with short-term response rates being much more favourable than long-term rates. This is in keeping with controlled trials in younger patients, showing ECT to have short-term efficacy only (Johnstone *et al*, 1980; Brandon *et al*, 1984). Depression is a chronic disorder, with prolonged morbidity in the elderly, as Benbow rightly points out. The short-term nature of the ECT response necessitates continuation treatment with antidepressants after ECT to prevent relapse. If the clinician recommends ECT and fails to seek an initial effective antidepressant, there is no way of knowing which drug will be effective after ECT.

Furthermore, trials comparing ECT with antidepressant drugs also suffer from methodological defects (Rifkin, 1988) and do not take into account advances in psychopharmacology, such as the use of combined antidepressants and neuroleptics in delusional depression.

With regard to safety, ECT is held to be superior when compared with older antidepressants. However, as Benbow indicates, the cardiovascular side-effects of ECT are not uncommon. Trials comparing the relative safety of ECT and the newer antidepressants, known to be considerably less cardiotoxic than the older antidepressants, have yet to be done.

Finally, end-point states of depression requiring ECT are not reached suddenly. The duration of depressive symptoms may be especially long in the elderly, and in under-resourced services it is often only those who have deteriorated into severe depressive states who are admitted for treatment, by which time ECT may be considered inevitable. A comprehensive community-based service, which can mobilise pharmacological, psychological, and social help at an early stage of the depressive disorder, may well reduce the need for ECT.

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References

BRANDON, S., COWLEY, P., MCDONALD, C., *et al* (1984) Electroconvulsive therapy: results from the Leicestershire trial. *British Medical Journal*, **288**, 22–25.

JOHNSTONE, E. C., DEAKIN, J. F. W., LAWLER, P., *et al* (1980) The Northwick Park electroconvulsive therapy trial. *Lancet*, *ii*, 1317–1320.

MALCOLM, K. & PEET, M. (1989) The use of electroconvulsive therapy in elderly depressive patients. In *Antidepressants in the Elderly* (ed. K. Ghose). Beckenham: Croom Helm.

RIFKIN, A. (1988) ECT versus tricyclic antidepressants: a review of the evidence. *Journal of Clinical Psychiatry*, **49**, 3–7.

Pimozide in pathological jealousy

SIR: Byrne & Yatham (*Journal*, August 1989, **155**, 249–251) claim that treatment with pimozide was successful but produce no evidence. They are aware that pathological jealousy can occur in relation to alcoholism and one would assume that following admission to hospital this patient would have ceased to drink. In that case one would have expected the symptoms to have subsided in the ensuing few weeks without the use of any drugs; indeed the correct treatment of such a patient after admission would be *not* to give any drugs for some weeks and to review the diagnosis after that time if symptoms persisted. If persisting symptoms then subsided following the administration of a drug, that would be reasonably convincing evidence of the drug's effectiveness; the case report does not state that the patient was managed in this way.

I am bound to comment on the authors' statement that it is "well established" that pimozide is "very effective" in the treatment of monosymptomatic hypochondriacal delusional psychosis. I have tried the drug in a number of patients but found it singularly ineffective. Others have told me that they have had similar experience. It is possible that it has some effect but it is far from "well established" that it is "very effective". On the other hand it *is* well established that morbid jealousy in alcoholics frequently subsides when they stop drinking.

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Syphilis screening

SIR: Boodhoo (*Journal*, August 1989, **155**, 259–262) poses a number of questions relating to syphilis screening in a psychogeriatric population.

Although syphilis remains a cause of reversible psychiatric illness, appropriate studies in patients

over the age of 65 are lacking. Nevertheless, as syphilis may remain latent for over 30 years, syphilis serology should be checked at any age and positive results further investigated by examination of the cerebrospinal fluid (CSF) (Weatherall *et al*, 1985). Negative CSF serology excludes active neurosyphilis, whereas a positive result should lead to treatment and follow-up CSF examination (Adams & Victor, 1985).

Until a study in this age group finds evidence to the contrary, we believe that syphilis screening remains an essential investigation.

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References

ADAMS, R. D. & VICTOR, M. (1985) *Principles of Neurology* (3rd edn). New York: McGraw-Hill Book Company.

WEATHERALL, D. J., LEDINGHAM, J. G. G. & WARRELL, D. A. (1985) *Oxford Textbook of Medicine*. Oxford: Oxford University Press.

Lipopigment in the CNS

SIR: Dowson (*Journal*, July 1989, **155**, 1–11) highlights the uncertainty surrounding the origin and significance of lipopigment in the central nervous system (CNS). Most authors would share the view that lipofuscin is not itself a causal factor in ageing, but rather a by-product, indicating that destructive oxidative processes have taken place (Sohal & Wolfe, 1986). Lipopigment itself may originate from several possible sources, but there is strong evidence to suggest that free-radical damage is an important factor. Damage to biological molecules produces malonaldehyde and other substances, inducing polymerisation of amine-containing molecules. The conjugated Schiff bases thus formed have similar emission spectra to those of chloroform extracts of purified lipofuscin, suggesting a common biochemical link (Tappel, 1975).

Dowson also suggests that chronic neuroleptic administration may protect against ageing, extrapolating from the effects of chlorpromazine on intracellular pigment in rat neurons. It has been suggested that phenothiazines, being heterocyclic compounds incorporating ring nitrogen and sulphur atoms, may act as free-radical scavengers. However, the 'cross-over effect' has also been noted, for instance with promethazine, which may be protective towards membranes at one concentration but deleterious at another (Slater, 1972). There is, indeed, growing evidence to suggest that neuroleptics may themselves induce free-radical damage in the CNS. This has been proposed as one mechanism by which tardive dyskinesia may be produced. Metabolites of phenothiazines, particularly the ortho-dihydroxylated derivatives, have been shown *in vitro* to generate toxic free-radical species (Heikkila & Cohen, 1975). Pall *et al* (1987) demonstrated increased products of free-radical damage in the cerebrospinal fluid of patients taking phenothiazines. In a trial of the antioxidant alpha-tocopherol, a marked reduction in the symptoms of tardive dyskinesia was described (Lohr *et al*, 1988).

The situation is clearly highly complex, as psychotropic drugs and their metabolites may exhibit different properties at various sites. Despite the difficulties of studying esoteric biochemical reactions in the CNS, the results may have considerable therapeutic implications. This line of inquiry therefore merits further research.

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References

HEIKKILA, R. E. & COHEN, G. (1975) Reactivity of various phenothiazine derivatives with oxygen and oxygen radicals. *Biochemical Pharmacology*, **24**, 363–368.

LOHR, J. B., CADET, J. L., LOHR, M. A., *et al* (1988) Vitamin E in the treatment of tardive dyskinesia: the possible involvement of free radical mechanisms. *Schizophrenia Bulletin*, **14**, 291–296.

PALL, H. S., WILLIAMS, A. C., BLAKE, D. R., *et al* (1987) Evidence of enhanced lipid peroxidation in the cerebrospinal fluid of patients taking phenothiazines. *Lancet*, *ii*, 596–599.

SLATER, T. F. (1972) Free radical scavengers. In *Free Radical Mechanisms in Tissue Injury*, pp. 48–61. London: Pion Limited.

SOHAL, R. S. & WOLFE, L. S. (1986) Lipofuscin: characteristics and significance. *Progress in Brain Research*, **70**, 171–183.

TAPPEL, A. L. (1975) Lipid peroxidation and fluorescent molecular damage to membranes. In *Pathobiology of Cell Membranes*, Vol. 1 (eds B. F. Trump & A. V. Arstila), pp. 145–170. New York: Academic Press.

Tardive dystonia

SIR: Cooper *et al* (*Journal*, July 1989, **155**, 113–115) reported tardive dystonia in a schizophrenic patient in his 20s which was worsened by anticholinergic drug treatment. This case is atypical. In a report by Kang *et al* (1986), 57% of patients with tardive dystonia were improved with anticholinergic drugs. Of the other patients with tardive dystonia reported in