hierarchy of questions exploring the strength of relationship between viral presence and psychosis: (a) does the patient have a systemic viral illness? (b) is the virus present in the cerebrospinal fluid (CSF) or is there evidence of an immunological response even if the virus is not present? (c) is the virus actually infecting the central nervous system (CNS)? and (d) is the viral infection of the CNS responsible for the observed psychosis?

Stoler *et al* would not appear to have sufficient evidence to answer the second question as, in the case they describe, the presence of monocytes in the CSF only indicates involvement on the meninges and is not adequate proof of brain tissue involvement. This highlights the need for rigorous use of more sophisticated techniques to demonstrate CNS involvement by viral agents (e.g. virus isolation, CSF banding) before moving from temporal associations to causal relationships.

NOEL MCCUNE

The Department of Child Psychiatry
The Royal Belfast Hospital for Sick Children
Belfast BT12 6BE

Reference

NUNN, K. P., LASK, B. & COHEN, M. (1986) Viruses, neurodevelopmental disorder and childhood psychosis. *Journal of Child Psychology and Psychiatry*, **27**, 55–64.

Electrodermal Response as a Monitor in ECT

SIR: Simpson & Hyde (*Journal*, April 1987, **150**, 549–551) give a description of the "cuff" technique (Adderley & Hamilton, 1953) for monitoring ECT.

I regret to have to point out that the description is incorrect. Before giving ECT, a sphygmomanometer cuff is applied to one arm and the pressure raised to above that of the systolic blood pressure. The suxamethonium is then injected through another vein, and after all the muscular twitching has stopped the sphygmomanometic cuff is released and the electric shock administered.

One should be wary of forcing a muscle to contract vigorously when its blood supply has been cut off.

MAX HAMILTON

School of Medicine
University of Leeds
30 Clarendon Road
Leeds LS2 9NZ

SIR: We thank Hamilton for clarifying details of his technique. It does not invalidate the result of our brief study, as the convulsion was observable as by the original method. Total systolic occlusion time was very brief and no harmful sequelae have been observed.

C. J. SIMPSON
C. E. HYDE

Withington Hospital
West Didsbury
Manchester M20 8LR

Neuroleptic Malignant Syndrome or Lithium Neurotoxicity?

SIR: Jee (*Journal*, April 1987, **150**, 568–569) argues that Abbott & Loizou (*Journal*, January 1986, **148**, 47–51) in their review of the neuroleptic malignant syndrome (NMS) appropriately excluded the data from Cohen & Cohen (1974) as an example of this syndrome on the basis that their evidence was inconclusive.

However, Abbott & Loizou had cited the observations of Baastrup *et al* (1976), whose study "was carried out in order to determine whether the syndrome described by Cohen & Cohen is, in fact, seen frequently in patients given both lithium and haloperidol", without reference to Cohen & Cohen's original findings. We were concerned by this omission and our own correspondence (*Journal*, September 1986, **149**, 385) simply drew attention to the descriptive superficial resemblance of the Cohen & Cohen cases to NMS.

We think that the evidence for lithium neurotoxicity in these cases as proposed by Jee is no stronger than our own postulations. He quotes Schou (1984) who reviewed case reports on 40 patients with persistent neurological sequelae after lithium intoxication, but fails to mention Schou's special comments on the Cohen & Cohen cases whom he regarded as atypical. Schou noted that none of them had particularly high serum lithium concentrations compared with the group as a whole; also, there was a high fever of unknown origin in all four cases, whereas in the rest of the group fever, where it was documented, was identified with a somatic illness in all but one case. Finally, on follow-up 2–10 months later none of these patients had the clear-cut cerebellar syndrome characteristically attributed to lithium toxicity.

More recently, Goldney & Spence (1986) in a retrospective study of 60 manic patients treated with neuroleptic drugs alone and 69 manic patients treated with neuroleptic drugs and lithium could demonstrate no significant differences in side-effects between the two groups, including comparisons made between patients on haloperidol only and those treated with haloperidol and lithium. Their