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.

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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.

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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.

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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

comments thus add further weight to the original observations of Baastrup *et al.*

Doubts regarding the true cause of the clinical events in Cohen & Cohen's patients will never be completely resolved, although the validity of their own postulate of a lithium/haloperidol interaction seems to be diminishing as the full explanation.

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Tuberous Sclerosis and the Autistic Syndrome

SIR: Lawlor & Maurer (*Journal*, March 1987, **150**, 396–397) described tuberous sclerosis presenting with autistic behaviour. Reported cases of this association are rare; however, I believe that it is not that uncommon. I can recall seeing three such cases. The following is an example.

Case Report: A male was born following a delayed labour and assisted delivery but recovered normally. Within 5 days he was found to be a restless baby with much screaming and difficulty in feeding. He walked at 12 months, but at 15 months he started to have temper tantrums. Subsequently, it became apparent that he had delayed speech. Later he developed overactive and disruptive behaviour, which eventually lead to his admission to a child psychiatry unit at the age of 3 years. He was diagnosed as having a profound language disorder with severe autistic traits, but was of potentially normal intelligence. Physical examination at this time showed no neurological abnormalities, but roughened skin on the cheeks and between the shoulder blades was noticed. Over the next few years there were many reports of poor social interaction, delayed speech with reversal of pronouns, mannerisms, inappropriate emotional reactions, resistance to change, and unusually good reading ability. At the age of 6 he developed epilepsy, which remained well controlled with anticonvulsants. At various times during his childhood he attended an ESN (M) day school, several private residential schools, and a school for autistic children.

At the age of 9 he was referred to me by the social worker because of difficult and aggressive behaviour. Physical examination revealed clear facial adenoma sebaceum, several areas of depigmented skin on the trunk and legs, and the previously recorded roughened areas between the shoulder blades. The family was advised of the possible diagnosis of tuberous sclerosis. Both parents were very intelligent and they had a normal daughter. The only significant feature in the family history was that the mother had had a mentally handicapped half brother who died at the age of 22 years with a tumour of the liver. Further physical examination of the family by a consultant geneticist revealed that the mother had a slight skin lesion in keeping with the diagnosis. The father and the sister appeared to be unaffected. The parents were advised of the significance of the findings, but had already decided to have no more children.

The patient has continued to present problems of management, especially aggressive behaviour, and still has many autistic features. He attends an Adult Training Centre and lives in a Social Services hostel.

The above illustrates not only the association of the two conditions but also supports the authors' conclusion that a suspicion of tuberous sclerosis should be aroused if autistic features and epilepsy coexist. It also suggests that history taking and physical examination in child psychiatry should always involve a genuine appreciation of genetic factors.

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Delusional Parasitosis

SIR: Macaskill (*Journal*, February 1987, **150**, 261–263) reports the first case of a non-pharmacologically induced remission of delusional parasitosis. Hunt & Blacker (*Journal*, May 1987, **150**, 713–714) suggest that this was a mild form of the disorder, associated with clear precipitants. I report two further cases of spontaneous remission.

Case reports: (i) A 51-year-old housewife with no previous history of medical or psychiatric illness presented to a dermatology clinic with a one-month history of parasites in her hair. She described hearing them moving, and could feel them burying into her scalp. She produced a matchbox containing skin scrapings as proof. There were no obvious precipitants, although she was under chronic strain through coping with her demented mother. The dermatologist found no abnormality, and prescribed no treatment. However, he concurred with her suggestion of a brief holiday in Spain. On her return two weeks later she was seen by the psychiatrist. She said the parasites had now gone, but was certain they had been present. There was no evidence of