Afro-Caribbeans have, for instance, White ancestry. Using the Victorian definitions of 'Caucasian' (predicated upon 'Indo-European' linguistic patterns), South Asian immigrants and their children born in Britain are 'Caucasian'. Or do the authors mean 'White' by 'Caucasian'?

The ambiguous and tendentious use of an idiom which connotes a simple causal relationship between phenotype, genotype, behaviour, and social experience is disturbing. What is wrong with the terms (a) *ethnic group* (as currently perceived), (b) *race* as a sociological term, or (c) quantitative measures of population genetics, as variously needed?

Estimates of various ethnic groups in the general population *can* be derived from the 1981 Census head of household figures using the Labour Force Survey correction factors.

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SIR: We thank Littlewood for giving us an update on the latest anthropological view for describing the demography of samples. To clarify the use of terms in our study, 'Negroid' refers to those individuals who have the appearance of some African ancestry, and 'Caucasian' refers to Indo-Europeans, white and non-white.

Clearly, associations in themselves cannot be used to imply a causal relationship, simple or otherwise.

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Mania Following Head Injury

SIR: Clark & Davison (*Journal*, June 1987, **150**, 841–844) argue that the infarct their second patient was noted to have in the left frontal region during a manic episode was unlikely to be causative because Cummings & Mendez, in their review on the association between mania and the site and nature of intercerebral lesions, did not find any reports of mania associated with cerebrovascular lesions in the left frontal region. There are at least two case reports of mania associated with lesions in the left frontotemporal region: Jampala & Abrams (1983) reported a 52-year-old man with recurrent manic episodes with onset at the age of 24 following a rupture of a left middle cerebral artery aneurysm. Herlihy & Herlihy (1979) have described a 58-year-old lady with manic episodes following a haemorrhage in the left middle cerebral artery. Both these patients were noted to have lesions in the left frontal and frontotemporal region.

Clark & Davison also note that the two previously-reported cases of mania associated with head injury occurred in younger patients and that this is contrary to the commonly held view that secondary mania is more common in the elderly, as suggested by Krauthammer & Klermann (1978). Shukla *et al* (1987) studied 20 patients who developed mania following closed head injury, and the age at first psychiatric episode in their patients ranged from 17 to 42 (mean = 27.5 years). Brackens (1987) reported a lady who developed mania at the age of 48 following head trauma. So, from the above reports, it appears that mania can occur at any age following head injury.

Carbamazepine is very effective in the treatment of secondary mania (as suggested by Jampala & Abrams (1983)) and this should be considered as one of the first line agents in addition to lithium.

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Schizophreniform Episode Following Measles Infection

SIR: The description by Stoler *et al* (*Journal*, June 1987, **150**, 861–862) may be the first adult case reported in the literature, but Nunn *et al* (1986) have previously reported four cases where there was an association between a viral illness (measles, rubella, varicella, and herpes) and adult-type psychosis in children. All the children had some neurodevelopmental disorder rendering the children more vulnerable to psychosis once infected by a virus.

Nunn *et al* drew attention to the need to clarify the role of the virus in the aetiology and suggested a

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