the acute phase and on a prophylactic basis for up to one year.

Of the 54 patients, 52 received variable doses of neuroleptics during the acute trial, and during the follow-up trial patients were given hypnotics, antidepressants, and neuroleptics when clinically indicated. The carbamazepine group required a higher average dose of neuroleptics in the acute phase, and no comparative information is given about neuroleptic dosage during the follow-up phase. This use of three different types of 'rescue' medication, which was not shown to be equivalent in the two groups, undermines the basis of the comparison made.

Our second reservation centres on the drop-out rate; 40 of the the original 54 patients were no longer in the trial at the end of the 12-month period. This also reduces the weight which can be given to the study. The authors' claim that carbamazepine is more effective as a prophylactic agent than lithium would appear to be poorly founded.

We would appreciate further clarification on the above points, particularly in view of more recent studies which take a less favourable view of carbamazepine as a mood stabiliser (Watkins *et al*, 1987; Frankenburg *et al*, 1988).

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SIR: Dr Murphy *et al* are, in a sense, quite right in what they say. Our sample of patients and method could not have allowed us to answer the question "Is carbamazepine better than lithium in the treatment or prophylaxis of the average manic depressive patient?".

They have, however, missed the point, since that was not the question that we were trying to answer. We simply wanted to see which of the two drugs appeared to be more useful when given in the hurly-burly of ordinary acute psychiatric work and, as a supplementary point, whether one could pick out specific patients particularly likely to respond to either carbamazepine or lithium.

They appear also not to have understood the results that we reported concerning drop-out rates. We had an admittedly uncomfortably high proportion of patients who dropped out through non-compliance, and there were other patients who reached an end-point for the trial when they relapsed and were readmitted. Dr Murphy *et al* are combining both groups when they imply that 40 of the original 54 patients were drop-outs.

Most of us have the clinical impression that carbamazepine sometimes works in manic-depressive illness. Our study served to reinforce that impression, and also gave some hints about which types of patients might be expected to do best on carbamazepine (i.e. males with 'textbook' mania). Judging from their address, our critics are writing from a research institute of some sort. One hopes that they will soon get round to doing work with manics, both for its intrinsic value and because it might give them a clearer appreciation of the difficulties involved in doing methodologically pure studies on such a volatile group.

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Is screening for syphilis justified?

SIR: We were interested to read the report by Boodhoo on syphilis serology screening in an elderly population (*Journal*, August 1989, **155**, 259–262). In a recent prospective survey of 659 consecutive elderly hospital admissions, we found that 23 (3.5%) had positive serology (Corrado *et al*, 1989), a similar proportion to that reported by Boodhoo. However, in our study we established the ethnic origin of all patients (to differentiate syphilis from yaws), whether patients showed stigmata compatible with congenital infection, and also whether patients had been previously treated for syphilis in Leeds during the preceding 70 years. Dr Boodhoo has not included this information, which is great import in the interpretation of positive results.

As Lishman (1978) pointed out, syphilis can present with a variety of psychiatric symptoms, and therefore it is difficult to be certain that psychiatric patients with positive serology have not got neurosyphilis. This is particularly true of cognitively impaired patients, and like Dr Boodhoo we had great difficulty in deciding the relevance of positive serology, but only examined the cerebrospinal fluid (CSF) of one patient. It has been suggested that

the diagnosis of neurosyphilis depends on clinical assessment, the results of serological tests, and examination of the CSF (Anon, 1978). Given that two separate studies have encountered the same diagnostic problem, perhaps a strong case can be made for CSF examination in these patients if routine blood screening is to be justified and of value in management.

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SIR: The recent article by Boodhoo (*Journal*, August 1989, **155**, 259–262) discussed the role of routine syphilis serology screening in a psychogeriatric population. We screened 172 psychogeriatric cases (mean age 69.2 years; 91 males and 81 females) for syphilis serology. Only two patients (1.2%) had positive VDRL; none of them had any clinical evidence of active syphilis or of any previous syphilitic processes. The psychiatric diagnosis in one case (a 72-year-old male) was psychotic depression, while the other patient (a 98-year-old female) was diagnosed as suffering from mania. These cases were referred to a venereologist for treatment, but he advised against it. The psychiatric illness in both cases was treated with drugs for about 6 months, but no antisyphilitic medication was given. On follow-up for 3 years, these cases did not develop the psychiatric illness again.

Although Luxon *et al* (1979) have emphasised the need for such routine investigations in psychiatric practice, there have been few cases reported where the antisyphilitic treatment has been instituted in an elderly mentally ill person (Joffe *et al*, 1968; Gilles, 1980).

If the psychiatric phenomena persist even with antisyphilitic treatment (Gilles, 1980), then it is not incorrect to abandon the routine screening of psychogeriatric cases for syphilis, as it would save on limited resources.

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Genetics of alcoholism

SIR: Adityanjee (*Journal*, October 1989, **155**, 564) rightly draws attention to the potential future advances promised by the application of molecular genetic techniques to the study of the genetic predisposition to the development of alcoholism. However, there is a serious flaw in his argument that a linkage strategy should be applied to the study of a hypothetical gene situated on the sex-determining region of the Y chromosome which may confer vulnerability to alcoholism.

Apart from the distal portion of the short arm of the Y chromosome (the so-called 'pseudo-autosomal region'), recombination can only occur on the Y chromosome between genetically identical sister chromatids of the same chromosome. Thus, apart from the pseudo-autosomal region, and assuming an absence of spontaneous mutation, all the sons born to a given father will share with their father identical Y chromosomes. Any male phenotypic variation in such a family will be due to environmental factors, and autosomal or pseudoautosomal genetic effects. Linkage studies of the (non-pseudo-autosomal) Y chromosome are simply not possible, since recombination between homologous chromosomes does not take place.

It is, in any case, debatable whether the Type I/Type II classification espoused by Cloninger will prove valid in the light of a genetic classification of alcoholism. Not all researchers agree that a genetic effect is absent in females (e.g. Cadoret *et al*, 1987). If a subtype is defined as affecting only males it is tautological to then refer to "the conspicuous absence of father-to-daughter transmission".

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