and equinoxes. In this way, we might test the influence of the increasing (February–April) or decreasing (August–October) photoperiod, and insufficient (November–January) or excessive (May–July) daylight.

Since Partonen & Lönnqvist do not report monthly frequencies, we cannot apply these criteria to the Finnish data. However, we have applied them to a Portuguese sample of 34 longitudinally followed bipolar patients (Pio-Abreu & Pires, 1985), and to 178 female admissions for mania and depression (Boto *et al*, 1991). Both studies revealed a peak of bipolar episodes during the equinoctial periods, where depressions predominate from February to April, and manias between August and October. In contrast, mixed and switching episodes, as well as some unipolar depressions, tended to occur around the solstices.

Although these results are consistent with an extensive review by Wehr & Rosenthal (1989), they may be idiosyncratic to Portugal. Since sunshine varies with latitude, more studies are needed worldwide in order to understand the problem better. However, it would be preferable if results were presented in terms of monthly frequencies, and not simply as the required figures for testing seasonality as conventionally defined.

Boto, I., Craveiro, A. & Pio-Abreu, J. L. (1991) Manias e depressões: distribuição sazonal e relação com factores climáticos. *Psiquiatria Clinica (Coimbra)*, **12**, 171–174.

Partonen, T. & Lönnqvist, J. (1996) Seasonal variation in bipolar disorder. *British Journal of Psychiatry*, **169**, 641–646.

Pio-Abreu, J. L. & Pires, I. C. (1985) Incidência sazonal das psicoses afectivas bipolares. *Psiquiatria Clínica (Coimbra)*, **6**, 181–188.

Wehr, T. A. & Rosenthal, N. E. (1989) Seasonality and affective illness. *American Journal of Psychiatry*, **146**, 829–839.

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Terminology of learning disability

Sir: Few would disagree with Reid (1997) that learning disability is not an ideal term. It may also be true that it was adopted by the Royal College of Psychiatrists simply for the sake of political correctness. This, however, even when coupled with the objection that the term contains no medical or psychiatric dimension, provides no adequate grounds for yet a further unwelcome change in terminology.

Mental handicap, the term generally discarded in the UK but nevertheless still favoured by Dr Reid and many others, remains less appropriate than learning disability for two important reasons. The first is, as Reid himself points out, because of the objections of those suffering from the condition and able to express an opinion. The second is the difficulty experienced by the general public in distinguishing between mental handicap and mental illness, largely because of the use of the word 'mental'. Not surprisingly, this confusion led to the assumption that mental handicap was primarily a medical problem. If now, instead, it is thought that the term learning disability implies that the condition is essentially educational, rather than register dismay we should instead throw our hats in the air. This description does after all contain a greater element of truth.

The problems of people with learning disability can be met only by a multidisciplinary approach. It is unlikely that the emphasis on the word 'learning' can diminish the contribution of medicine, particularly psychiatry, to the care of this group.

Reid, A. H. (1997) Mental handicap or learning disability. A critique of political correctness. *British Journal of Psychiatry*, **170**, 1.

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Sir: As Reid (1997) points out in his excellent editorial, no-one would wish to revert to the terms 'idiot' or 'imbecile'. None the less, it is interesting to note that these terms were themselves once euphemisms. In classical Greek the adjective *idios* means one's own, private or personal, and the noun idiot (*idiotes*) was a private as opposed to a public person. Idiot then came to mean someone without professional knowledge, and finally, in some post-classical Greek writers, "an ignorant person" (Liddell *et al*, 1961). 'Imbecile' comes from the Latin *imbecillis*, which means weak or frail. Perhaps in our own times terms such as 'intellectually disabled' will soon be found too stigmatising and replaced by 'intellectually differently abled'.

Underlying this absurd quest for ever newer euphemisms there would appear to be two implicit, but partly contradictory assumptions: first, that language is thought; and second, that the language we use determines what we think. There are several good reasons for believing that language is not thought, such as our awareness of not being able to put some of our own thoughts into words, problem-solving involving the manipulation of mental images (i.e. non-verbal thinking), the invention of words or neologisms to express new ideas for which no words previously existed, the phenomenon of ambiguity in language, etc. (Pinker, 1995). Thus, if language is not thought, the notion that language predetermines how we think loses much of its plausibility. It is our thought which contaminates the euphemisms, not the euphemisms which disinfect our thought. For these reasons also Reid is right: in the case of mental handicap or learning disability it is our attitudes which must change, not our terminology.

Liddell, H. G., Scott, R. & Jones, H. S. (1961) *A Greek–English Lexicon*. Oxford: Clarendon Press.

Pinker, S. (1995) *The Language Instinct*. Harmondsworth: Penguin.

Reid, A. H. (1997) Mental handicap or learning disability. A critique of political correctness. *British Journal of Psychiatry*, **170**, 1.

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Valproate and neuroleptic medication

Sir: Barnes *et al* (1996) point out the paucity of data on adjunctive use of valproate in the treatment of psychotic disorders only partially responsive to neuroleptics.

We have conducted an open trial of 17 out-patients (six male; mean age 34; s.d. 10 years) to ascertain whether valproate can be used to 'spare' neuroleptics in patients with bipolar disorder with psychosis (n=13) and schizoaffective disorder (n=5). All patients had been stabilised on neuroleptics for at least six months. Mean pre-valproate neuroleptic dose was 260 mg chlorpromazine equivalents per day (s.d. 150 mg; range 25–500). In the six months post-valproate, only two patients required ongoing neuroleptics, with doses of 100 and 200 mg chlorpromazine equivalents daily (prior doses 200 and 500 mg, respectively).

This preliminary study, with the methodological limitations inherent in open, non-randomised, non-blind designs, nevertheless raises the possibility of wider use of valproate to spare neuroleptics in patients with bipolar and schizoaffective disorders, and potentially schizophrenia as well (three further treatment-resistant schizophrenia patients have been commenced on valproate

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with promising clinical improvement). We are currently engaged in a study to pursue this issue in a more scientific manner, and welcome the views of other clinicians.

Barnes, T. R. E., McEvedy, C. J. B. & Nelson, H. E. (1996) Management of treatment resistant schizophrenia unresponsive to clozapine. *British Journal of Psychiatry*, **169**, (suppl. 31), 31–40.

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Genetic polymorphism and drug-induced movement disorders

Sir: We were interested to read the paper by Armstrong *et al* (1997) on drug-induced movement disorders in relation to the *CYP2D6* genotype. We would agree that this important polymorphism may well be a contributory factor in more chronic drug-induced movement disorders.

Several lines of evidence support this. Firstly, one of the most robust findings in the whole field of tardive dyskinesia (TD) research is the link between TD and high neuroleptic dosage. Impaired clearance of antipsychotics due to the poor metaboliser genotype leads to elevated plasma levels, which has clear implications in view of the first association. Other studies have attempted to assess the problem from a different standpoint: looking at the variation in neuroleptic breakdown between those with TD and schizophrenic controls. The best study in this area is that of Yesavage *et al* (1987), which found a significant difference in standardised thioxanthine levels between 21 TD sufferers and 20 controls.

In our recent study (Bates, 1997) we used promethazine, a phenothiazine predominantly metabolised by the CYP2D6 cytochrome, to probe metabolic clearance in 18 patients, 10 with TD and eight controls. We used a high-performance liquid chromatography technique which simultaneously assayed promethazine and its two major breakdown products, the sulphoxide and monodesmethyl metabolites. We found evidence of significant impairment of metabolism of promethazine in the TD group, with raised promethazine levels and raised promethazine to metabolite ratios, indicating this was not an effect of varying absorption or bioavailability. We will be publishing our results more fully soon.

This simple and relatively inexpensive method may prove useful in pretreatment testing to enable prediction of those likely to develop TD or concentration-dependent side-effects, and to guide dosage decisions.

Armstrong, M., Daly, A. K., Blennerhassett, R., *et al* (1997) Antipsychotic drug-induced movement disorders in schizophrenics in relation to *CYP2D6* genotype. *British Journal of Psychiatry*, **170**, 23–26.

Bates, G. D. L. (1997) *Using Promathazine to Demonstrate Abnormalities of Phenothiazine Metabolism in Tardive Dyskinesia*. MMedSc dissertation, Department of Psychiatry, University of Birmingham.

Yasavage, J. A., Tanke, E. D. & Sheikh, J. I. (1987) Tardive dyskinesia and steady-state serum levels of thiothixene. *Archives of General Psychiatry*, **44**, 913–915.

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Demography and age at onset of schizophrenia

Sir: Jablensky & Cole (1997) conclude from their analysis of the WHO 10-Country study of schizophrenia that marital status has a major effect on age at onset of schizophrenia, and that the effect of gender disappears when controlling for marital status (and other variables). However, these findings are partly due to demographic effects. To explain this artefact assume that outbreak of schizophenia occurs (like a random event) irrespective of marital status. As is well known, married people are in our population on average older than single ones. Therefore the mean age at onset of schizophrenia will be later for married people than for single ones. This difference does not reflect a real association between schizophrenia and marriage (which was excluded by the hypothetical model) but it reflects a trival demographic effect due to the different age structures of married and single people. This applies to men and women but in a different way. In general, women marry earlier than men. Thus, in the population, married women are on average younger than married men, and unmarried women are younger than unmarried men. Let us assume that outbreak of schizophrenia is related neither to gender nor to marital status. Then because of these different age structures the mean age at onset will be earlier for married women than for married men. The same relation holds for singles. But, as above, these differences are due to a trival demographic effect. If there is a real association between gender and schizophrenia in the sense that onset is later for women, then this difference is reduced when comparing married women with married men and single women with single men. Thus differences in the age structures between married and single persons in the population explain, at least in part, the effects of marital status and gender on age at onset, described by Jablensky & Cole. The differences in age at onset between developing and developed countries may be attributed to different demographic structures in these countries. We do not deny these effects, but to disentangle demographic effects from real effects the age structures of the underlying population must be taken into account when analysing the data.

Häfner, H., Riecher, A., Maurer, K., *et al* (1989) How does gender influence age at first hospitalization for schizophrenia? A transnational case register study. *Psychological Medicine*, **19**, 903–918.

Jablensky, A. & Cole, S. W. (1997) Is the earlier age at onset of schizophrenia in males a confounded finding? Results from a cross-cultural investigation. *British Journal of Psychiatry*, **170**, 234–240.

Riecher-Rössler, A., Fätkenheuer, B., Löffler, W., *et al* (1992) Is age of onset in schizophrenia influenced by marital status? Some remarks on the difficulties and pitfalls in the systematic testing of a simple question. *Social Psychiatry and Psychiatric Epidemiology*, **27**, 122–128.

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Author's reply: Jennen-Steinmetz *et al* suggest that "differences in the age structures between married and single persons . . . explain, at least in part, the effects of marital status and gender on age at onset, described by Jablensky & Cole". Since the statistical analyses we use uncorrelate gender and marital status prior to examining the relationship of either variable to age at onset, Steinmetz *et al*, can rest assured that no part of our conclusions reflects a spurious correlation between marital status and age at onset of schizophrenia that is induced by those two variables' common association with gender differences in age at marriage. In the general linear model, the effects of gender (and gender-correlated differences in age at marriage) are partialled out, or controlled for, in the calculus underlying multiple regression (Mosteller & Tukey,