

During the past decade a number of studies have examined the effect of prophylactic lithium treatment on the mortality and suicidal behaviour of manic-depressive patients. Two reviews, Tondo *et al* (1997) and Schou (1998), have analysed them and found a statistically significant association between long-term lithium treatment, on the one hand, and reduced mortality and suicidal behaviour on the other. According to the first review, the number of suicidal acts was on average 3.2 per 100 patient-years in patients without lithium and 0.37 per 100 patient-years in patients with lithium, an 8.2-fold difference. The second review showed that whereas manic-depressive patients have a mortality that is two to three times that in the general population, the mortality of patients with lithium treatment was not higher or was only slightly higher than in the general population. The number of suicide attempts was 6–15 times lower and the number of completed suicides 3–17 times lower when the patients were on lithium than when they were not. When lithium treatment was discontinued, the mortality and the suicidal behaviour increased.

These observations indicate strongly that prophylactic lithium treatment has an anti-suicidal action, but correlation is not the same as causation, and other factors than the treatment may have played a role. It is, however, noteworthy that an association between suicidal behaviour and drug treatment has not been demonstrated for use of other mood stabilisers, where the same factors must have been at work. In a European, randomised, collaborative long-term trial, no suicidal act occurred in patients on lithium, whereas nine suicidal acts were observed in patients having received carbamazepine (Thies-Flechtner *et al*, 1996).

The findings reviewed above do not prove definitely that lithium has an anti-suicidal action but the evidence does not reject such a hypothesis. We suggest that prophylactic lithium treatment should be given to manic-depressive patients at high risk of committing suicide, that is patients with severe depression or depression with persistent suicidal ideation or with a history of suicide attempts.

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**Thies-Flechtner, K., Müller-Oerlinghausen, B., Seibert, W., et al (1996)** Effect of prophylactic lithium treatment on suicide risk in patients with affective disorders: Data from a randomized prospective study. *Pharmacopsychiatry*, **29**, 103–107.

**Tondo, L., Jamison, K. R. & Baldessarini, R. U. (1997)** Effect of lithium maintenance on suicidal behavior in major mood disorders. *Annals of the New York Academy of Sciences*, **836**, 339–351.

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### Alcohol, hypertension and cognitive decline

**Sir:** Stewart (1999) highlights some interesting issues regarding the role of hypertension in dementia. No reference is made, however, to the potential role of alcohol in these conditions. Alcohol excess has been linked independently to both cognitive impairment and high blood pressure, although the concept of a distinct primary alcoholic dementia remains controversial (Smith & Atkinson, 1995).

Stewart observes that an apparent fall in blood pressure can occur at the onset or during the development of dementia. This process may well be related to alcohol withdrawal in some individuals. One of the pathways into abstinence seen in older alcoholics occurs when some consequences of the dementing process (e.g. institutionalisation) limit the supply of drink and propel the individual into an ‘involuntary’ abstinence. In such cases the patient may also experience a fall in blood pressure as alcohol is withdrawn, contributing to the clinical picture described by Stewart. Curtis *et al* (1997) have documented alterations in systolic blood pressure which correlate with changes in drinking status. The role of alcohol should be investigated in future longitudinal studies of hypertension.

**Curtis, A. B., James, S. A., Strogatz, D. S., et al (1997)** Alcohol consumption and changes in blood pressure among African Americans: The Pitt county study. *American Journal of Epidemiology*, **146**, 727–733.

**Smith D. M. & Atkinson, R. M. (1995)** Alcoholism and dementia. *International Journal of Addictions*, **30**, 1843–1869.

**Stewart, R. (1999)** Hypertension and cognitive decline. *British Journal of Psychiatry*, **174**, 286–287.

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### Prolonged seizures detectable by electroencephalogram in electroconvulsive therapy

**Sir:** Mayur *et al* (1999) reported that 38 (16.4%) of 232 patients experienced prolonged cerebral seizure activity detectable by two-channel electroencephalogram (EEG) at the first treatment in a course of electroconvulsive therapy (ECT). In contrast, Abrams (1997) argued that the existing evidence suggested prolonged cerebral seizure activity is rare in the routine practice of ECT, and is usually associated with coexisting brain disease. This debate prompted us to re-analyse the findings of two Scottish studies previously published in the *Journal* to report for the first time the prevalence of prolonged cerebral seizure activity in UK practice.

The Royal College of Psychiatrists (1995) recommended that seizures that last longer than 120 seconds ought to be regarded as prolonged and be terminated. The American Psychiatric Association recommended a cut-off of 180 seconds (American Psychiatric Association, 1990).

In the Dumfries study, single-channel EEG monitoring was conducted at most treatments given to 41 patients (McCreadie *et al*, 1989). Usable EEG recordings were obtained at 263 treatments (101 unilateral treatments and 162 bilateral treatments). One 23-year-old woman experienced prolonged seizures at her first four bilateral treatments in a course of six treatments. One 55-year-old woman experienced a prolonged seizure at her first bilateral treatment only. The prevalence of prolonged seizure activity at the first treatment was 2 out of 41 patients (4.9%, 95% CI – 1.7 to 11.5%) for seizures longer than 120 seconds, and 1 out of 41 patients (2.4%, 95% CI – 2.3 to 7.1%) for seizures longer than 180 seconds.

In the Edinburgh study, six-channel EEG monitoring was carried out at the outset and end of treatment plus a variable number of intermediate treatments in 22 patients (Scott *et al*, 1989). Usable EEG recordings were available for 100 treatments (43 unilateral treatments and 57 bilateral treatments). No prolonged seizures were detected.

We calculated a 95% CI for the reported prevalence from Bangalore, which was 11.6–21.2%. This suggested that it was unlikely that the differences in prevalence between Scotland and Bangalore were simply because of sampling error. Other possible explanations include variations in ECT technique, anaesthesia, or the methodology of EEG recording. These topics merit further research and may inform the debate about the importance of routine EEG monitoring.

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**American Psychiatric Association (1990)** *The Practice of Electroconvulsive Therapy: Recommendations for Treatment, Training and Privileging*. Washington, DC: APA.

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**Mayur, P. M., Gangadhar, B. N., Janakiramaiah, N., et al (1999)** Motor seizure monitoring during electroconvulsive therapy. *British Journal of Psychiatry*, **174**, 270–272.

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## Mental health and Irish ethnicity

**Sir:** Given Bracken *et al's* (1998) use of mental hospital admissions data of the

1971 and 1981 Mental Health Enquiries to argue that the Irish have the poorest record of mental health of all ethnic minorities in Britain, it is surprising that the investigators offer no explanation of why reliance was placed on such outdated information.

The Hospital Episode Statistics (HES) system was introduced in April 1987 to replace the Hospital Inpatient Enquiry (HIPE) and the Mental Health Enquiry (MHE). HIPE ceased to exist in 1985 and the MHE in 1986, these changes resulting in major deficiencies in HES data until the early 1990s. Routine hospital activity data did not record any measure of ethnicity until the mandatory collection of ethnic group data for National Health Service (NHS) hospital in-patients was introduced in April 1995. However, the use of a national standard for collecting the data – the 1991 Census classification without provision for free-text responses – meant the only options for the majority self-identifying as 'Irish' were to tick 'White' or 'Any other ethnic group'. Although the NHS offered a detailed set of local codes, including 'Irish', scarcely any trusts have used these (Aspinall 1998a). Also, the continuing high level of incompleteness of ethnic coding (44% invalid in 1996/97) means that HES is unlikely to provide in the near future the data needed to establish current levels of admission for psychiatric and other specialities among the Irish. This is unfortunate in view of the Commission for Racial Equality's efforts in 1995–96 to get NHS trusts to include an 'Irish' monitoring category and the reported wish of the trusts themselves to see the national standard 'White' category broken down to include groups like the Irish (Aspinall, 1998b).

What is needed is some measure of psychiatric morbidity in the community, including those in communal establishments. Clearly with health and lifestyle surveys collecting data on up to 25 000 residents (South East Institute of Public Health, 1993), there is scope to accrue basic data on the mental health of the Irish (through the use of, for example, the generic instrument SF-36 and associated three-item depression screen, or the General Health Questionnaire (GHQ-12)). However, the regular inclusion of an 'Irish' category among the population groups used in such surveys will depend upon its widespread recognition as a legitimate ethnic group. Perhaps that has been brought a step nearer with the recently announced decision to include an 'Irish' category in the 2001 Census (Her Majesty's Treasury *et al*, 1999).

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## One hundred years ago

### Morphine habit of long-standing cured by bromide poisoning

Dr. Neil Macleod, *Brit. Med. Journ.*, July 10th, 1897. – Two very interesting cases of treatment, to say the least heroic, are here recorded. The first case, a lady aged

32, had been the victim of the morphine habit for seven years. The extent to which she had taken this drug is not given, but when she came under treatment with symptoms of great nervous irritability she had reduced her morphine to the, for her, very small dose of 10 minims of Majendie's solution

(gr.  $\frac{1}{3}$  rd) every four hours. She was ordered bromide of sodium in 30-grain doses every four hours, but must have trebled this dosage, for in two days 18 drachms had been consumed. She was removed to hospital, and for four days received hypodermically  $1\frac{1}{2}$  grains of morphine per diem. On the