unusual and florid nature of Matthews' symptoms: "having scarcely in the whole period of his professional practice and experience known any patient more completely and equivocally mad" (Monro, 1809). Haslam's clinical descriptions of his patients are noted for their clarity and keenness of detail, and he would not have failed to record the symptoms of schizophrenia if he encountered them.

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Compulsive water drinkers

SIR: Lee *et al* (*Journal*, April 1989, **154**, 556–558) describe the case of a 16-year-old Chinese girl with water intoxication following the ingestion of large quantities of water for traditional medicinal purposes. I was surprised to read the suggestion that the use of water to induce an altered state of consciousness (ASC) had not previously been described. Many of the patients I have seen with excess fluid intake present as 'drunk'; intoxication is defined in most dictionaries as including a feeling of excitement, exhilaration, and inebriation. Perhaps the usual description of the symptoms of water intoxication does not include pleasure as this is not something the patient complains about. The literature does contain references to ASC, though this title is not used. For example, Singh *et al* (1985) describe how Case 1, on becoming unemployed and thus no longer able to afford alcohol, turned to regular excessive water drinking because it "made him feel slightly drunk" (this paper is quoted by Dr Lee *et al*). In a more recent paper on the possible association between water intoxication and alcohol abuse, Ripley *et al* (1989) write that "mild overhydration may be experienced as pleasurable, leading to further polydipsia".

With regard to the case described, the authors make no mention of the girl's tobacco smoking habits, although they acknowledge that the clinical picture suggests a degree of SIADH (syndrome of inappropriate antidiuretic hormone secretion). Nicotine stimulates the release of arginine vasopressin, and thus may be significant in the aetiology of water intoxication and SIADH. Other associated factors in SIADH (according to Singh *et al*) are stress, head injury, medications (antipsychotics, anticonvulsants, diuretics, and anti-cancer drugs), and various medical conditions (especially malignancies).

If, as the authors imply, drinking large quantities of water is a culturally accepted practice in traditional Chinese medicine, I wonder if this girl's complications are unusual, and if so, why she specifically was so adversely affected.

The seeking of an ASC may be an aetiological link in a proportion of compulsive drinkers. It is not, however, a 'missing' link.

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Risk factors in schizophrenia

SIR: In their elegant study Baron & Gruen (*Journal*, April 1988, **152**, 460–465) found a greater familial risk for schizophrenia and schizophrenic spectrum disorders among probands born in the winter and spring than among those born during the remainder of the year. This finding is not in line with the major part of the pre-existing literature (Torrey, 1987), which primarily indicates an opposite trend. Aside from the results, the study also differs from previous ones in that it also considers the specific morbidity risk in first-degree relatives. In light of these discrepancies, we undertook a study to confirm whether schizophrenics born between December and April differ from those born during the other months of the year, both in relation to incidence of a family history of schizophrenia and to a familial morbidity risk for

the disorder (cumulative evidence from the literature indicates winter and early spring as the period when most schizophrenic births occur).

This study, comprising 187 patients (117 males and 70 females under 40) who fully met the DSM–III–R criteria for schizophrenic disorder (American Psychiatric Association, 1987), showed considerable differences in familial predisposition to schizophrenia in the two seasonal groups. Five of the 84 patients born in the winter and early spring (6%), and 17 of the 103 patients born in the other months (16.5%) had a family history of schizophrenia ($\chi^2 = 4.9$, $P = 0.025$). Furthermore, morbidity risk data confirmed the role of family history. Five of the 241 age-adjusted relatives of the probands born between December and April (age adjustment according to Weinberg's abridged method) and 18 of the 323 age-adjusted relatives of the probands born during the remainder of the year turned out to be schizophrenics ($\chi^2 = 4$, $P = 0.043$), so that the latter group had a familial morbidity risk (5.3%) 2.65 times greater than that of the former (2%).

Consequently, our findings are apparently compatible with previous hypotheses of a lowered familial loading of schizophrenia among patients born in the winter and early spring, but manifestly differ from those of Drs Baron & Gruen. Undoubtedly, the lack of complete comparability of the protocols may have accounted for some of the differences in the results: for example, we used the end of April instead of the end of May as one of the benchmarks for separating the season of birth, and the Weinberg's abridged method instead of the more precise age correction based on continuous distribution of the ages at the disease onset. But we should not neglect the possibility that the samples selected in one or another or both of the studies are not completely representative of the phenomena under analysis. In fact, the figures for the secondary cases in our sample are somewhat inferior to those to be expected for relatives of Italian schizophrenics (Macciardi *et al*, 1987) and, on the contrary, the greater risk found by Drs Baron & Gruen (among the relatives of the patients born in winter and spring) apparently seems largely sustained by a few families with many cases of schizophrenia, i.e. by high-risk families. If this is true, then some of the discrepancies could be reconciled, given that systematic diathesis–stress interactions along a continuum of susceptibility are the bases for the seasonality phenomenon. Within this framework, we cannot only expect that the harmful effect of seasonal environmental factors is maximised when, as was apparently the case in our sample, the weight of familial predisposition to schizophrenia is in some way minimised, but also that the former further increases the likelihood of the schizophrenic disorder when subjects at high risk are predominantly considered.

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Antidepressant toxicity

SIR: I read with interest the review by Beaumont (*Journal*, April 1989, **154**, 454–458), and was surprised to note that there was no mention of the relatively new antidepressant fluvoxamine, which is reputed to be very safe when taken in overdose. Fluvoxamine inhibits the neuronal uptake of 5-hydroxytryptamine with little or no effect on the catecholamine system. Minor reported side-effects include: nausea, vomiting, dizziness, dyspepsia, headache, anxiety, palpitations, diarrhoea, and a rash (Roos, 1983; Classen *et al*, 1977).

There have been 42 cases of self-poisoning with fluvoxamine (Banerjee, 1988). One patient died, but necropsy showed the fluvoxamine tablets to be intact in the stomach, and the death was attributable to an overdose of propanolol. In another reported case, the patient was unconscious for five days, but recovered fully from her coma (Banerjee, 1988). The prolonged cerebral depression was thought to be due to a possible interaction between fluvoxamine and