Solvent abuse in psychiatric settings

SIR:

Case report. A 24-year-old man was admitted to a secure ward in a psychiatric hospital following a relapse of his manic–depressive illness. He presented with typical features of hypomania but despite what was felt to be adequate medication (he had several previous admissions), his mental state continued to fluctuate. Following a period in which his affect was objectively euthymic and his behaviour appropriate, he would suddenly become irritable and aggressive. Routine urinary drug screening proved negative. It was then discovered that he had been inhaling the contents of his aerosol deodorant, and confiscation of the aerosol resulted in the settling of his mental state.

It has been reported that the inhalation of fluorocarbons used in domestic aerosols results in euphoria and intoxication (Bass, 1970). Other compounds abused include aliphatic, aromatic or mixed hydrocarbons and anaesthetic gases (Ramsey *et al*, 1989). These may be found in many 'household' products (e.g. adhesives, stain-removers and typewriter correction fluid). It is of particular interest that the newer 'ozone-friendly' aerosols often contain butane, which has previously been abused in the form of cigarette-lighter refills. The signs of intoxication include confusion, hallucinations and delusions, which may lead to aggressive behaviour (Ashton, 1990). These could be misinterpreted as signs of functional psychosis. Increased dosages of these substances may cause ataxia, dysarthria and drowsiness, which may be mistakenly attributed to side-effects of prescribed antipsychotic medication.

The Royal College of Psychiatrists' consensus statement on the use of high-dose antipsychotic medication discussed its possible role in the causation of sudden cardiac death in often previously healthy young patients (Thompson, 1994). We know that inhalation of solvents may also cause sudden cardiac death due to arrhythmias (Shepherd, 1989), and as such we would wish to highlight the particular danger of this practice in patients on antipsychotic medication.

As a result of the case described, the use of all aerosols has been prohibited on this particular ward, and patients are advised to use roll-on deodorants.

ASHTON, C. H. (1990) Solvent abuse. *British Medical Journal*, **300**, 135–136.

BASS, M. (1970) Sudden sniffing death. *Journal of the American Medical Association*, **212**, 2075.

RAMSEY, J., ANDERSON, H. R., BLOOR, K., *et al* (1989) An introduction to the practice, prevalence and chemical toxicology of volatile substance abuse. *Human Toxicology*, **8**, 261–269.

SHEPHERD, R. T. (1989) Mechanism of sudden death associated with volatile substance abuse. *Human Toxicology*, **8**, 287–292.

THOMPSON, C. (1994) The use of high-dose antipsychotic medication. *British Journal of Psychiatry*, **164**, 448–458.

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Prevalence of Huntington's disease in the Oxford region

SIR: The prevalence of Huntington's disease (HD) varies between 25 per million to 99.5 per million in the UK (Harper, 1991). The Department of Medical Genetics at Churchill Hospital, Oxford, was one of the few centres in the UK to set up a regional pre-symptomatic test service for the disease in 1986. The long-term preventive effectiveness of such a programme can only be assessed by establishing the baseline prevalence of HD in the region. A study that relied entirely on the department's case notes for the HD patients, assembled by the author, reported a prevalence of 40 per million in 1988 (Watt & Seller, 1993). This was an underestimate. Details of the method and the criteria used to obtain the results below are described elsewhere (Shiwach, 1994).

On 31 December 1985, 138 patients (from 93 apparently unrelated families) suffering from HD were alive and living in the region (population 2.437 million according to the Unit of Health-care Epidemiology, Oxford). Demographic information was available on 134 patients (65 women and 69 men) and the mean age was 50.6 years (range 18–76, s.d. 13.9 years). The patients were mostly British in origin, although five were from overseas.

The age at onset ranged between 12 and 72 years (mean=42.2, s.d 12.9 years); there were eight cases of juvenile HD, 92 (68%) had a 'classic' age at onset, and 35 (26%) had a late onset. Five patients had no family history despite characteristic clinical features of the disease, but only one patient was considered to have a possible mutation. The multiple ascertainment method, including 'retrospective designation' of cases, in 1991, showed a point prevalence of 56.58 cases/million (95% confidence interval 47.6–66.9).

The rates in different surveys differ because of a natural difference (geographical isolation and aggregation of few cases in a small population) or because of the different methods of assessment. The study by Watt & Seller (1993) did not take account of the patients not referred for genetic counselling