

Hospital in the five years prior to the study commencing. Less than half (49%) of our study sample were in regular contact with a psychiatrist (see Lang *et al.*, 1997, Table 1).

We would agree with Dr Simpson that the development of a comprehensive primary care record for patients with schizophrenia would be a welcome advance. We are of the opinion, however, that if people other than GPs are to contribute to such records, it is essential that a standardised method of recording consultations in the primary care setting is devised.

We agree with Dr Simpson that alcohol misuse in both younger and older people with schizophrenia significantly contributes to the difficulties which arise in patient management, but we did not find that a past history of alcohol misuse was independently related to whether or not a patient was considered by the GP as causing a problem for the practice.

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### Lethal lithium poisoning with sustained-release preparations

**Sir:** Cookson (1997) and Moncreif (1997) have mentioned serious toxicity and death as a consequence of lithium intoxication. It would be useful to stress that more serious poisoning usually occurs after intoxication by sustained-release lithium preparations.

In 27 years of lithium medication in Southern Moravia, four cases of lethal intoxication have been reported, three of them suicides. All three patients took a high dose of the sustained-release lithium product. In two cases, the approximate doses were known (50 and 70 g lithium). In one case, the dose has not been established. Two of the patients died on the fifth day and the other died on the 12th day after taking the lethal dose of lithium. In one of the patients, the autopsy revealed undissolved lithium tablets in the stomach and the remains of tablets in the intestines, despite a gastric lavage. In another patient, high levels of lithium (34 and 11.3 mmol/l, respectively) were found in the gastric lavage returns on the first and second days.

It seems that the advantage of sustained-release preparations becomes a disadvantage during intoxication. Standard gastric lavage does not remove the lithium tablets from the gastro-intestinal tract, and if they are not removed, lithium is released for a surprisingly long period of time.

One effective method, proposed by Smith *et al.* (1991), is gastric lavage with polyethyleneglycol, which can prevent the absorption of up to 67% of the ingested lithium. Other authors recommend gastroscopy combined with aspiration, under visual control, or enterotomy to remove the tablets in addition to lavage (Olson, 1991; Ševčík *et al.*, 1994).

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### Tardive dyskinesia and CYP2D6 polymorphism in Chinese

**Sir:** Epidemiological studies have shown a lower prevalence rate of tardive dyskinesia among Chinese than Caucasian subjects receiving neuroleptics (Swartz *et al.*, 1997). A pharmacogenetic factor may underlie this cross-cultural difference. The study by Armstrong *et al.* (1997) which suggests an association of neuroleptic-induced movement disorders, including tardive dyskinesia, with genetic polymorphism of CYP2D6 supports this hypothesis. About 7% of the Caucasian population lacks the CYP2D6 enzyme (i.e. poor metabolisers) compared with about 1% of the Chinese (Brosen, 1996). The majority of these Caucasian poor metabolisers have a mutant allele 2D6(B), which contains a splicing defect that is not present in a large section of the Chinese population (Brosen, 1996). New mutations

have recently been reported, including the C/T<sub>2938</sub> variation that results in one amino acid difference (R296C). This mutation, postulated to be in the putative substrate-recognition site of the CYP2D6 enzyme, also shows ethnic differences in its distributions (26% in Caucasians, 19% in Chinese) (Wang *et al.*, 1995). It has also been reported to be associated with Parkinson's disease (Tsuneoka *et al.*, 1993). This is intriguing because patients with a family history of Parkinson's disease have a high risk for developing tardive dyskinesia (Swartz *et al.*, 1997). This suggests an association of a vulnerability to tardive dyskinesia with the R296C mutation – a possibility that merits further investigation.

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**Brosen, K. (1996)** Drug-metabolism enzymes and therapeutic drug monitoring in psychiatry. *Therapeutic Drug Monitoring*, **18**, 393–396.

**Swartz, J. R., Burgoyne, K., Smith, M., et al (1997)** Tardive dyskinesia and ethnicity: review of the literature. *Annals of Clinical Psychiatry*, **9**, 53–59.

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### Evidence-based psychiatry

**Sir:** The practice of evidence-based medicine has been defined as the conscientious, explicit and judicious use of current best evidence in making decisions about the care of individual patients (Sackett *et al.*, 1996). A systematic review of randomised controlled trials can be a very useful source of evidence. The key point about a systematic review is that it has a method section which clearly states how the primary studies were identified and selected for inclusion in the review, and how their quality was assessed (Oxman *et al.*, 1994). Neither the article by Moncreif (1997) nor the commentary by Cookson (1997) provided this information, despite their inclusion under the heading of 'evidence-based psychiatry'. It is therefore difficult to assess the validity of these articles, and to what extent the authors attempted to avoid bias.