1990) and 6% of patients have been reported to develop tricyclic-induced delirium on conventional doses of 100–300 mg/day. At plasma levels greater than 1000 μg/l, nearly all will show ECG changes (Spiker *et al*, 1975). The first sign of cardiac toxicity may be a fatal arrhythmia.

Optimisation of clinical response is only one goal of therapeutic drug monitoring and even when well-defined plasma levels are not established for certain tricyclics, it still remains a useful tool in the management of depression. By detecting asymptomatic toxicity, therapeutic drug monitoring can prevent adverse consequences and may have medicolegal implications. Other advantages include assessment of non-responders, provision of a measure of compliance and confirmation of toxicity. In contrast to Taylor and Duncan, we believe that routine estimation of plasma tricyclic levels is an integral component in the rational approach to the management of depression and should be more widely practised.

PRESKORN, S. H. (1993) Sudden death and tricyclic antidepressants (TCAs): a rare adverse event linked to high TCA plasma levels. *Nordic Journal of Psychiatry*, **47**, (Suppl. 30), 49–55.

— , JERKOVICH, G. S. (1990) Central nervous system toxicity of tricyclic antidepressants: phenomenology, course, risk factors and role of therapeutic drug monitoring. *Journal of Clinical Psychopharmacology*, **10**, 88–95.

SPIKER, D. G., WEISS, A. N., CHANG, S. S. (1975) Tricyclic antidepressant overdose: clinical presentation and plasma levels. *Clinical Pharmacology and Therapeutics*, **18**, 539–546.

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Sir: Toxicity may indeed occur in the absence of clinical symptoms. However, 'CNS toxicity' and delirium *are* clinical symptoms and so plasma level determinations are of little use apart from to confirm that the antidepressant is the cause of the problem. Electrocardiogram (ECG) abnormalities may be asymptomatic and so a plasma level may help identify those at risk. However, an ECG will still need to be performed to identify any arrhythmia and one might argue that this should be done for anyone taking moderate or high doses of tricyclics. We are also unsure of the value of a 'cut-off' level of 1000 mcg/l. Presumably anyone with a level above this would have an ECG performed. One wonders what course of action would be taken with a patient with a level of 999 mcg/l, or 950 mcg/l, or 800 mcg/l. We feel the quickest way to detect occult rhythm abnormalities in patients taking tricyclic and related antidepressants is immediately to perform an ECG.

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Improper terminology

Sir: We warmly welcome the issues raised in the paper by Haghighat & Littlewood (*Psychiatric Bulletin*, July 1995, **19**, 407–410) which raises the issue of potential labelling and stigmatisation of people suffering from mental disorders. It is our duty to treat people with respect; as individuals, yet holistically. This should be made clear in the way that we, as professionals, refer to patients, their problems, and their illnesses.

Since Haghighat & Littlewood's paper, we have been surprised and disappointed to note the continued use of terms such as 'schizophrenics' (e.g. Fagin *et al*, *Psychiatric Bulletin*, August 1995, **19**, 533) and even 'dements' (*Psychiatric Bulletin*, November 1995, **19**, 704) to refer to patients.

We strongly believe that as "The Journal of trends in psychiatric practice" the *Bulletin* should take the moral lead on this issue, and avoid publication of such pejorative and stigmatising labels.

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Point taken. – Ed.

Correcting drug-induced hyperprolactinaemia

Sir: Duncan and Taylor (*Psychiatric Bulletin*, December 1995, **19**, 755–757) describe possible clinical usage of two drugs relatively unfamiliar to psychiatrists, amantadine and bromocriptine. They suggest this would correct a common side-effect of antipsychotic drugs, hyperprolactinaemia. We believe that a simpler strategy should be followed initially.

Patients treated with antipsychotic medication can experience a variety of unpleasant endocrine side-effects; most commonly gynaecomastia, galactorrhoea and amenorrhoea. This is considered to be due to hyperprolactinaemia caused by antagonism of the action of dopamine on tuberoinfundibular neurones (Meltzer & Fang, 1976).

The atypical neuroleptic clozapine is known to cause either a minimal or no rise in serum prolactin (Jann *et al*, 1993). Clozapine is indicated for the treatment of schizophrenia in patients unresponsive to, or intolerant of, conventional antipsychotic drugs (*British National Formulary*, 1995).

Our practice, in this not uncommon clinical situation, would be to change to clozapine. Duncan and Taylor point out that amantadine may precipitate mania and is unlicensed for hyperprolactinaemia. They also point out that bromocriptine is contraindicated in any psychotic

illness. It is difficult to understand why they recommend the treatment of antipsychotic induced hyperprolactinaemia with the addition of either of these drugs to conventional antipsychotics rather than changing to clozapine. We feel that treatment with clozapine should be considered as an alternative to this polypharmacy, and we are concerned that Duncan and Taylor's review did not mention this option.

JANN, M. W., GRIMSLEY, S. R., GRAY, E. C., *et al* (1993) Pharmacokinetics and pharmacodynamics of clozapine. *Clinical Pharmacokinetics*, **24**, 161–176.

MELTZER, H. Y. & FANG, V. S. (1976) Serum prolactin levels in schizophrenia – effect of antipsychotic drugs: a preliminary report. In *Hormones, Behavior and Psychopathology* (ed. E. J. Sachar), pp. 178–191. New York: Raven Press.

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Sir: In our article, we noted that clozapine was the only antipsychotic not to cause hyperprolactinaemia. Clozapine may therefore successfully be used in patients with neuroleptic-induced hyperprolactinaemia. This fact was only implied in our article and we agree with your correspondents that the option to use clozapine should have been more explicit. In addition, as your correspondents state, clozapine is licensed to be used where patients are intolerant of standard neuroleptics for any reason.

In practice, we always suggest clozapine to prescribers as one of several possible therapeutic gambits in hyperprolactinaemia. In our unit, clinicians prefer to try dose reduction or amantadine. As far as we are aware, none of over 100 patients taking clozapine in our unit were prescribed the drug because of previous problems with hyperprolactinaemia.

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The use of new antipsychotics

Sir: We read with interest Professor Kerwin's article on the use of new antipsychotic drugs such as clozapine (*Psychiatric Bulletin*, January 1996, **20**, 23–29). He argued that "enthusiasm for the use of new antipsychotics has not been as great as one might expect" and attributes this, partly, to cost. We wish to contribute to the debate by discussing further the issue of cost.

The total adult population of our catchment area is approximately 203 878. Assuming the point prevalence of schizophrenia to be between 2.5 to 5.3 per 1000, the number of expected patients with schizophrenia in our community would be between 509 and 1080. Of these 152–324 would be treatment resistant (Kane *et al*, 1988). If the estimated cost of clozapine per annum is £2500, the likely cost to our service of prescribing clozapine to all patients who theoretically could benefit from it would be £380 000–810 000. This upper figure is twice our annual drug budget.

Furthermore, at present, the cost to our service of prescribing clozapine is approximately £175 355 per annum. This is 36% of the total drug budget and amounts to about 54% of the sum expended on antipsychotic drugs. To put it in another way, 36% of the whole budget is spent on 56 patients who amount to less than 1% of patient contacts in one year.

The issue of cost-effectiveness of clozapine must be conducted within the context of actual budgets and of opportunity costs. We mean by this that there are competing claims upon a limited budget and that the needs of other patients and the fixed costs of institutions must be included in the cost-benefit analysis of clozapine. This is particularly true in services which no longer contain large groups of chronically ill patients within long-stay wards. The gains which would have been made in being able to secure discharges to community facilities are not evident in such settings. And, if there are already facilities for intensive follow-up and treatment at home, the cost benefits of reduced admissions would be negligible.

In conclusion, we believe that a thorough cost-benefit analysis of the use of clozapine in the UK context is now urgently needed.

KANE, J., HONIGFELD, G., SINGER, J., *et al* (Clozaril Collaborative Study Group) (1988) Clozapine for the treatment resistant schizophrenic: a double blind comparison with chlorpromazine. *Archives of General Psychiatry*, **45**, 789–796.

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Competence consent and dementia

Sir: The articles by Bartlett (*Psychiatric Bulletin*, November 1996, **19**, 670–672) and Burns & Harris (*Psychiatric Bulletin*, February 1996, **20**, 107–108) help to clarify our ethical responsibilities to patients with dementia. However, competence, consent and dementia remain a dilemma.

When I set up a research project involving home visits to elderly people with dementia I had to