If we start from the well-researched position that severe disturbances of emotional development are likely to be enacted in later life, and add Adshead's observation that this is often manifest in care-seeking behaviour with psychiatric services, it is a small step to propose that resources are used in some proportion to that disturbance. Conversely, disturbance of attachment can be 'weighed' like patients' case notes, by the input from the services that they receive.

So perhaps the cost-offset studies for personality disorder, such as the Henderson Hospital work (Menzies *et al*, 1993) are measuring improvement in dysfunctional attachment patterns as well as counting money. Clinicians in therapeutic communities and other settings for treating emotionally unstable personality disorder are likely to be more comfortable thinking that they are making a fundamental difference to the way patients relate to others, than that they are doing it to save money, but perhaps they are the same thing.

Adshead, G. (1998) Psychiatric staff as attachment figures. Understanding management problems in psychiatric services in the light of attachment theory. *British Journal of Psychiatry*, **172**, 64–69.

Menzies, D., Dolan, B. M. & Norton K. (1993) Are short term savings worth long term costs? Funding treatment for personality disorders. *Psychiatric Bulletin*, **17**, 517–519.

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PTSD and victims of torture

Sir: I read with interest the article by Gorst-Unsworth & Goldenberg (1998) on psychiatric morbidity in Iraqi victims of torture. The authors speculate on the causes of the relatively low level of post-traumatic stress disorder (PTSD) within their sample compared with the rate found among Vietnam veterans.

While it is of interest to compare the rates of PTSD in various high-risk groups, it is important not to lose sight of the specific features of each of these groups. Torture is, by and large, a highly selective act employed by tyrannical states against certain individuals for a complex range of political purposes. It is rarely an indiscriminate act. In the Iraqi context, torture is used as a routine form of social control which is meant to terrorise the individual concerned (and/or extract information from the victim) as well as to act as a deterrent to others. The targets of the torture are carefully selected from suspected activists or other independently minded individuals who appear to stand out among their peers and who are judged to pose a short- or long-term threat.

From the point of view of the tyrannical state, torture is a finite resource (limited by the availability of 'skilled torturers') that must be deployed to best effect. The non-random nature of the targets of torture is in clear contrast to other victims of disaster, where either minimal or no selection applies (e.g. victims of road traffic accidents or the veterans of a conscript army).

An understanding of the interaction beween the victims' characteristics and the characteristics of the trauma may offer us a better chance of predicting the level of risk of developing a particular psychiatric syndrome following traumatic and stressful events.

Gorst-Unsworth, C. & Goldenberg, E. (1998) Psychological sequence of torture and organised violence suffered by refugees from Iraq. Trauma-related factors compared with social factors in exile. *British Journal of Psychiatry*, **172**, 90–94.

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Cost-effective community psychiatry

Sir: There is good evidence to support Dr Tyrer's (1998) point that fragmenting community care for serious mental illness leads to longer hospital admissions. In a natural unplanned experiment during the Daily Living Programme of home-based care in south-east London, removing the community care team's responsibility for any crisis admissions of their patients led to a trebling in duration of those crisis admissions (Marks *et al*, 1994). Having the same staff responsible for the patient's community care and any crisis admissions seemed critical if care was to remain cost-effective.

Marks, I. M., Connolly, J., Muijen, M., *et al* (1994) Home-based versus hospital-based care for people with serious mental illness. *British Journal of Psychiatry*, **165**, 179–194.

Tyrer, P. (1998) Cost-effective or profligate community psychiatry? *British Journal of Psychiatry*, **168**, 1.

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Sulpiride augmentation on schizophrenia

Sir: I would like to comment on the article by Shiloh *et al* (1997) which suggests an augmenting effect of sulpiride in patients who failed to respond satisfactorily to clozapine.

The population studied comprised patients with schizophrenia who, after receiving clozapine for 12 weeks at daily doses ranging from 350 to 600 mg, showed a partial clinical response. Most British psychiatrists would not consider these patients as resistant to clozapine and would increase the daily dose, side-effects allowing, up to 900 mg.

Recent clinical plasma levels studies on clozapine support this practice, pointing to the importance of achieving values in excess of 370 ng/ml (Buckley, 1996). When Miller *et al* (1994) raised the dose in seven patients not responding to clozapine who had plasma concentrations below 370 ng/ml, five of them improved. It has to be added that Kane *et al* (1988), in their seminal study on the effectiveness of clozapine in treatment-resistant patients, prescribed daily doses of up to 900 mg.

It is therefore possible that the clinical improvement obtained in the study was not due to any specific pharmacodynamic effect, but was caused by an aspecific increase in the dopamine blockade in a population which was receiving clozapine at a subtherapeutic dosage. The same effect would probably have been achieved by adding any of the other major tranquillisers or, better still, increasing the dose of clozapine. Bone marrow depression, which is the most dangerous adverse effect of clozapine, is not dose dependent. It would, therefore, be unwise to advocate a polypharmaceutical regimen, which introduces the very unpleasant side-effects secondary to the sulpiride-induced hyperprolactinaemia, instead of just increasing the dose of clozapine.

As a last point it is unlikely that, as suggested in the article, the augmenting effect of sulpiride could be due to a pharmacokinetic interference at the level of the P450 liver metabolism of clozapine. The hepatic metabolism of sulpiride is, in fact, negligible, more than 95% of the compound being excreted unchanged in the urine (Imondi *et al*, 1978).

Buckley P. F. (1996) Treatment of schizophrenia: advances during the decade of the brain. *British Journal of Hospital Medicine*, **11**, 574–580.

https://doi.org/10.1192/bjp.172.5.449c Published online by Cambridge University Press

Imondi, A. R., Alam, A. S., Brennan, J. J., et al (1978) Metabolism of sulpiride in man and rhesus monkeys. *Archives Internationales de Pharmacodynamie et de Therapie*, **232**, 79–91.

Kane, J., Honigfeld, G., Singer, J., et al (1988) Clozapine for treatment-resistant schizophrenia: a double blind comparison with chlorpromazine. *Archives of General Psychiatry*, **45**, 789–796.

Miller, D. D., Fleming, F., Holman, T. L., et al (1994) Plasma clozapine concentrations as a predictor of clozapine response: a follow up study. *Journal of Clinical Psychiatry*, **55**, 117–121.

Shiloh, R., Zemishlany, Z., Aizenberg, D., et al (1997) Sulpiride augmentation in people with schizophrenia partially responsive to clozapine. A double-blind, placebo-controlled study. *British Journal of Psychiatry*, **171**, 569–573.

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Authors' reply: Dr Procopio draws attention to two main points. First, he raises the possibility that under-dosing might be the primary factor associated with the partial response to clozapine observed in our study population prior to the addition of sulpiride to their regimen. We are aware of such possibility, which might account for some of the beneficial effects described. However, we would like to stress our main claim which emphasised the role of the altered serotonin–dopamine receptor occupancy ratio which was achieved by the enhanced D_2 dopaminergic blockade of sulpiride (a selective D_2 antagonist) and could not have been attained (to a similar degree) with higher doses of clozapine (a relatively weak D_2 antagonist). Furthermore, all of our patients have shown an initial response to clozapine, which was later followed by a relatively long and steady non-responsive period. At the same time, some of our patients were unable to tolerate higher doses of clozapine because of troubling side-effects. Moreover, it is of note that clozapine-related seizures appear to be close-related, and high-dose therapy $\geqslant$600 mg/day) is associated with substantially increased risk than are doses of 300–600 mg/day (Devinsky *et al*, 1991). Furthermore, we would like to refer to a similar and substantial clinical improvement which was recently reported with the combination of clozapine and pimozide (Friedman *et al*, 1997) and clozapine–risperidone regimens (Henderson & Goff, 1996) in partial responders to clozapine. Both pimozide and risperidone are relatively potent D_2 blockers and in these cases the mean daily doses of clozapine were 425 and 479 mg, respectively, which are in the same range as in our study (403 mg/day). These studies examined the efficacy of the described combinations in patients who were maintained on clozapine treatment alone for longer periods (8–12 months) before adding either pimozide or risperidone. Hence, it seems that some patients with schizophrenia either partially responsive to clozapine or unable to tolerate higher doses could substantially benefit from enhancing the D_2 dopaminergic blockade.

Devinsky, O., Honigfeld, G. & Patin, J. (1991) Clozapine-related seizures. *Neurology*, **41**, 369–371.

Friedman, J., Ault, K. & Powchik, P. (1997) Pimozide augmentation for the treatment of schizophrenic patients who are partial responders to clozapine. *Biological Psychiatry*, **42**, 522–523.

Henderson, D. C. & Goff, D. C. (1996) Risperidone as an adjunct to clozapine therapy in chronic schizophrenics. *Journal of Clinical Psychiatry*, **57**, 395–397.

R. Shiloh, Z. Zemishlany, D. Aizenberg, A. Weizman Gehah Mental Health Center, PO Box 102, Petach-Tikua 49100, Israel

Amisulpride in schizophrenia

Sir: We read with interest the editorial by Thomas & Lewis (1998) on atypical antipsychotics, and value their review of these drugs which have significantly affected the management of schizophrenia. However, we were surprised to note the omission of amisulpride in their consideration of atypical antipsychotics, despite its being mentioned in their introduction. After extensive use in France, amisulpride has only recently become available in the UK and has been the focus of several papers in the *Journal* (Boyer *et al*, 1995; Loo *et al*, 1997; Speller *et al*, 1997).

Thomas & Lewis comment that the atypical antipsychotics have not been shown to benefit primary negative symptoms in schizophrenia, and certainly the majority of studies dealing with this issue have been subject to considerable confounding variables (such as simultaneous improvement in positive symptoms and extrapyramidal side-effects; King, 1998)

Amisulpride would appear to be one of the few antipsychotic drugs which has been studied with consideration of these pitfalls (Boyer *et al*, 1995; Loo *et al*, 1997) and the findings support a positive outcome with primary negative symptoms. Speller *et al* (1997) found no such improvement over the course of one year, but given that their sample had a median age of 63 years and duration of illness of 36 years, the lack of response was perhaps not surprising.

We would suggest that the positive results of the amisulpride studies merit further examination, given that negative symptomatology is for many patients the most debilitating aspect of their illness. Or could Euroscepticism be influencing our approach to the drug treatment of schizophrenia?

Boyer, P., Lecrubier, Y., Puech, A. J., et al (1995) Treatment of negative symptoms of schizophrenia with amisulpride. *British Journal of Psychiatry*, **166**, 68–72.

Loo, H., Poirier-Littre, M.-F., Theron, M., et al (1997) Amisulpride versus placebo in the medium-term treatment of the negative symptoms of schizophrenia. *British Journal of Psychiatry*, **170**, 18–22.

King, D. J. (1998) Atypical antipsychotics and the negative symptoms of schizophrenia. *Advances in Psychiatric Treatment*, **4**, 53–61.

Speller, J. C., Barnes, T. R. E., Curson, D. A., et al (1997) One-year, low-dose neuroleptic study of in-patients with chronic schizophrenia characterised by persistent negative symptoms. Amisulpride v. haloperidol. *British Journal of Psychiatry*, **171**, 564–568.

Thomas, C. S. & Lewis, S. (1998) Which atypical antipsychotic? *British Journal of Psychiatry*, **172**, 106–109.

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Systematic does not necessarily mean comprehensive

Sir: The recent review of brain abnormality in schizophrenia (Lawrie & Abukmeil, 1998) is described as systematic. The reviewers identified studies by a "computerised literature search from 1986 to June 1996 with Medline on CD-ROM using the search terms 'MRI' and 'schizophrenia' ". Journals were also hand-searched and reference lists scrutinised. There are important problems with this search. It is not enough simply to state that a CD-ROM system has been searched over a designated period. It should be made explicit exactly which disk issues were searched. Not to do so makes replication of the review impossible and causes the resulting product to stray from being systematic at all.

The search was systematic but not comprehensive. We replicated Lawrie & Abukmeil's electronic search on the January 1998 SilverPlatter edition of Medline, requesting that citations be retrieved only from between 1986 and June 1996; 187