(2005): 'the clinical treatment of young people identified as being at high risk of developing a psychotic disorder, particularly the use of neuroleptics, should be provided only in the context of a research trial, where standards of informed consent and monitoring are highest'.

Nevertheless, there remain worries about trials in poorer countries. Ethical committees often do not have the same level of independence as they do elsewhere, financial inducements may lead to covert or overt pressures, and there is even sometimes a nationalistic element (e.g. if country X can recruit 100 patients, we must not recruit fewer than 200). This somewhat macho mentality may be behind comments such as that by Khanna *et al* (2005) that the symptoms of mania in the patients seen were 'substantially more severe than those of patients with bipolar disorder participating in trials elsewhere', implying that only countries that can be successful in persuading these 'difficult' patients to take part should be chosen.

We note that the Indian Council of Medical Research has now decided to audit clinical trials systematically to ensure that national recommendations are followed (Mudur, 2005) and the outcome of this will be followed closely. For our part, we have made changes to our refereeing procedure, and have been asking assessors to examine more closely the ethical aspects of papers that are submitted. We shall also be using our new group of international editors (in the case of India this will be Dr Vikram Patel) to advise on ethics both generally and with regard to specific papers, attempting as much as possible to take account of the need for 'autonomy, beneficence, non-maleficence and justice . . . and care ethics' summarised by Bloch & Green's (2006) recent paper.

**Bloch, S. & Green, S. A. (2006)** An ethical framework for psychiatry. *British Journal of Psychiatry*, **188**, 7–12.

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## Antiparkinsonian prescription and extrapyramidal symptoms

Park *et al* (2005) cite the results of clinical trials as evidence supporting their hypothesis that the use of antiparkinsonian drugs in schizophrenia is an indication of extrapyramidal symptoms (EPS). This may be true for clinical trials (most of which include young adults with no comorbidity) but may not hold true for their observational study, in which other factors such as prescribing habits and comorbidity may affect the reason for prescription of antiparkinsonian drugs. As the mean age of their sample was 48.6 years, which falls within the range in which Parkinson's disease often develops, some patients could have been receiving antiparkinsonian drugs for the illness *per se*. Although this is mentioned as a limitation of the study, it has an adverse impact on the central hypothesis. Since decrements and increments in antiparkinsonian medication followed expectations from changes in antipsychotics (Tran *et al*, 1997), the results could well reflect the prescribing pattern of the general practitioners (GPs) rather than be true evidence for the presence of EPS.

One of the main limitations of the study is the lack of data regarding the reason for switching antipsychotics. As it is mandatory to submit data of all major illnesses (presumably including Parkinson's disease), any indication for prescribing or altering medication and any adverse drug reaction to the General Practice Research Database (GPRD; Walley & Mantgani, 1997), the data could have been provided and would have helped in the interpretation of the results. Furthermore, during the period studied more than 400 GPs provided data to GPRD but data from only 266 were analysed. It is not clear why the data from some GPs were excluded.

Park *et al* (2005) classified their study population as those switched from typical to atypical antipsychotics (TA group) and those switched from typical to different typical antipsychotics (TT group). However, when we add up the total figures provided (3% and 99% were receiving atypicals and typicals respectively in 1992, which changed to 47% and 70% in 2000), it appears that some patients were receiving a combination of both classes of antipsychotics. This could have influenced the trend for prescribing antiparkinsonian drugs.

**Park, S., Ross-Degnan, D., Adams, A. S., *et al* (2005)** Effect of switching antipsychotics on antiparkinsonian medication use in schizophrenia: population-based study. *British Journal of Psychiatry*, **187**, 137–142.

**Tran, P. V., Hamilton, S. H., Kuntz, A. J., *et al* (1997)** Double-blind comparison of olanzapine versus risperidone in the treatment of schizophrenia and other psychotic disorders. *Journal of Clinical Psychopharmacology*, **174**, 15–22.

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**Authors' reply:** We agree with the comments of Grover & Kulhara on the lack of information about the specific reasons for the prescription of antiparkinsonian drugs in our observational study. We have stated that such prescribing might have been influenced by factors other than the occurrence of EPS. However, previous naturalistic studies have shown that the use of antiparkinsonian medication was highly correlated with clinical indices of EPS when patients were prescribed antipsychotics (Barak *et al*, 2002; Bobes *et al*, 2003; Montes *et al*, 2003). In addition, the sudden change in the incidence of antiparkinsonian drug use following introduction of atypical antipsychotics in the entire population (not just among patients who switched type of antipsychotic therapy) makes it unlikely that physician prescribing habits were a strong alternative explanation for our findings.

Since we observed the same patients over time in the analysis of drug switching, changes in antiparkinsonian drug prescribing following the switch could be explained by the differential effects of antipsychotics on EPS.

Nevertheless, antiparkinsonian drug prescribing is only a marker of EPS and



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