cannot perfectly reflect the incidence of EPS. Owing to the limitation of our dataset (which did not include indications for prescriptions), we cannot exclude the possibility that some patients may have been prescribed antiparkinsonian medication because they had Parkinson's disease, not because they had EPS caused by antipsychotics.

Grover & Kulhara question why we included only 266 GPs in this study. We selected from the GPRD only those patients who had been diagnosed with schizophrenia and prescribed antipsychotics between 1992 and 2000. Therefore 6356 patients who met those requirements and their 266 general practices were included in the study.

Grover & Kulhara raise the possibility that patients might have taken both classes of antipsychotics simultaneously. We examined the effects of switching antipsychotics on antiparkinsonian drug prescribing by classifying patients into two groups. We defined the TA group as patients who had been prescribed typical antipsychotics with no atypical antipsychotic use before the switch, completely stopped typical antipsychotics and subsequently switched to atypical antipsychotics, with no typical antipsychotic use for at least 2 years after the switch. The TT group included patients who were prescribed one typical antipsychotic (e.g. chlorpromazine) then switched to a different typical antipsychotic (e.g. haloperidol), and who never received an atypical antipsychotic during the study period. Therefore, by definition, no patients in our study were receiving a combination of both classes of antipsychotics.

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Treatment of borderline personality disorder

Fonagy & Bateman (2006) hypothesise that a more benign course of borderline personality disorder may partially result from a reduction in iatrogenic harm. They describe people with borderline personality disorder as having 'hyperactive attachment systems' which interfere with the therapeutic relationship and treatment. They describe 'treatment' as being psychosocial treatment or psychotherapy, and attachment figures as therapists.

Many people with borderline personality disorder do not receive psychotherapy but do have contact with psychiatric services – casualty assessments, out-patient contact with generic services, brief crisis admissions and sometimes even prolonged admissions. I am curious as to Fonagy & Bateman's view on the nature of attachments that people with borderline personality disorder have with psychiatric *institutions*, especially when contact with individual workers may be inconsistent. Fonagy & Batemen give advice about how to encourage 'mentalisation' in the context of psychotherapy in order to avoid potential iatrogenic damage but give no advice for other clinical settings.

Clinical teams are well aware of how people with borderline personality disorder may unconsciously 'engineer' situations to re-enact disturbed early life experiences. Now Fonagy & Bateman suggest that although teams are aware of this situation further damage may be done. A 'helpful' intervention may deprive the patient of using or developing other more useful strategies. Fonagy & Bateman suggest that an 'inquisitive and flexible' approach may be useful. The challenge is therefore how this approach should be applied to how clinical teams within *institutions* respond to people with borderline personality disorder.

Fonagy, P. & Bateman, A. (2006) Progress in the treatment of borderline personality disorder. *British Journal of Psychiatry*, **188**, 1–3.

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Authors' reply: We share Dr Mountain's concern that this group of patients is often inadequately managed. Our primary aim in pointing to the iatrogenic consequences of psychotherapy was to illustrate the dangers of intensive interventions or those with poorly defined boundaries. The same concerns for iatrogenic consequences apply to institutional involvement because this is often disrupted by frequent staff changes. Separations and losses of this kind are also iatrogenic. They activate patients' attachment systems, leading them to make unproductive attempts to restabilise their sense of self. Moreover, interactions with institutions often occur at times of personal crisis when the attachment system is already stimulated. Concerns about the patient's state of panic and about reduced mentalising may lead to hospital admission. However, this can become iatrogenic in itself because emotionally charged interactions with staff and other patients may further destabilise the patient, leading them to self-harm or threaten suicide, prolonging hospital admission. We and others (Paris, 2004) recommend that the level of risk for self-harm of patients admitted to hospital should be assessed and documented daily. If there is no reduction in risk, alternative management of the patient in the community should be implemented.

Although patients may seem to be enacting past experiences in their interactions with clinical teams, in our view it is not useful to consider these as hapless repetition of past patterns or as acts that respond to or compensate for past hurts; rather they should be viewed as the only solution available to restore a sense of integrity, continuity and coherence. The provision of a highly integrated model of psychiatric care in a structured institutional environment that aims to offer consistent, coherent and thoughtful psychological care with a relationship focus, organised in a patient-oriented flexible manner with individualised care plans, is likely to be most helpful. Out-patient treatment, discharge from an in-patient unit or referral following a casualty visit should be considered in

https://doi.org/10.1192/bjp.188.5.493-a Published online by Cambridge University Press

this context if services are to present a stable and coherent view of the patient's subjective world that may be adopted (internalised) as part of the self-image of the patient's mind. In our view this is the critical change in the treatment of borderline personality disorder.

Declaration of interest

The authors are in receipt of a grant from the Borderline Personality Disorder Foundation to support a randomised controlled trial of intensive out-patient psychotherapy.

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Neurobehavioural characteristics and relapse in addiction

The systematic review by Dom *et al* (2005) of studies using behavioural decision-making tasks and/or neuroimaging techniques to investigate orbitofrontal cortex functioning in substance use disorders was comprehensive. Our research article 'Risk-taking on tests sensitive to ventromedial prefrontal cortex dysfunction predicts early relapse in alcohol dependency' (Bowden-Jones *et al*, 2005) was published simultaneously and, because of its relevance to the review, we considered it important to bring it to readers' attention.

We not only used most of the neuropsychological tests mentioned by Dom *et al* but, more importantly, rated participants on both the Rogers Cambridge Gamble Task (RCGT; Rogers *et al*, 1999) and the Iowa Gambling Task (IGT; Bechara *et al*, 1994), and on the Barratt Impulsivity Scale and two personality scales: the Structured Clinical Interview for DSM–III–R (Spitzer *et al*, 1989) and the Dimensional Assessment of Personality Pathology–Basic Questionnaire (Livesley & Jackson, 2002).

The 21 participants in our study were in-patients in a residential detoxification unit and we were therefore able to carry out tests at 21 days post-detoxification in the knowledge that they had been substance-free during that period. They were followed up for 3 months post-discharge.

The six patients who relapsed early were significantly younger and more impulsive on the Barratt Impulsivity Scale, they sampled significantly more cards from the bad decks on the IGT and consistently risked more points across all odds on the RCGT. Hence people who had recently undergone detoxification were more likely to relapse within 3 months if they made more choices on a gambling task in which the immediate reward was large but the long-term consequences were disadvantageous.

It is unlikely that these findings reflect alcohol-induced brain damage because these people showed no impairments on a memory test sensitive to the early stages of dementia and on tests of dorsolateral prefrontal cortex functioning, which is particularly affected by long-term alcoholism.

Our results are consistent with the hypothesis that a dysfunctional orbitofrontal prefrontal cortex mediates the inability to resist the impulse to drink. This may lead a person to relapse after treatment despite the ultimately deleterious effects and despite the many hours of psychological input associated with a rehabilitation programme.

Relapse after detoxification is an area in need of further research. If it has a biological basis we need simple tests that are able to predict vulnerability to relapse and treatment programmes which are able to identify those patients at greater risk.

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Authors' reply: The findings of Bowden-Jones *et al* (2005) add to the accumulating evidence that impairments on decision-making tasks are an important characteristic of people with substance use and possibly other addictive disorders. The finding that those alcohol-dependent people that performed poorly on behavioural tasks were at higher risk of relapse is a nice demonstration of the 'myopia' for the future that is reflected by poor task performance. This is in line with other recent studies, including that of Goudriaan *et al* (2006), which showed that relapse among gamblers was associated with behavioural (but not self-reported) measures of impulsivity. Furthermore, Paulus *et al* (2005) reported that methamphetamine-dependent people with low prefrontal activation during a decision-making task relapsed significantly more frequently than those with greater activation. Together with the results of Bowden-Jones *et al* (2005), these findings represent an important new line of investigation.

Identification of distinctive neurobehavioural characteristics may allow detection of those people with addictions that are more vulnerable to relapse. Neurobehavioural (endophenotypic) characteristics may prove to be better for the identification of high-risk patients than traditional clinical (phenotypical) variables.

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Paulus, M. P., Tapert, S. F. & Schuckit, M. A. (2005) Neural activation patterns of methamphetamine-dependent subjects during decision making predict relapse. *Archives of General Psychiatry*, **62**, 761–768.

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