makes the interpretation of any resulting phenomenology extremely difficult.

It is also clear from the original publication by Singleton *et al* that no specific assessment for PTSD was carried out, although validated and reliable instruments for this exist (e.g. the Clinical Assessment for PTSD, Blake *et al* (1995) or the Posttraumatic Stress Symptoms Interview (PSSI) or Posttraumatic Stress Symptoms Self-Report (PSS–SR), Foa *et al* (1993)). The authors did a partial screen for a few recognised PTSD symptoms, such as re-experiencing and avoidance, but there was no systematic assessment of the condition that would have allowed them to diagnose the full disorder. It should be recognised that PTSD is a major psychiatric disorder that constitutes a serious burden for the individual and for society (Kessler, 2000). A diagnosis of PTSD has implications in terms of assessing the individual's risk and in terms of treatment recommendations. It is important that the term post-traumatic stress should not be confused or conflated with the term 'post-traumatic stress disorder'. The description of post-traumatic stress made by Coid *et al* cannot be evaluated without deconstructing more precisely what this means. As there are now well-recognised instruments to assess PTSD and lifetime experience of traumatic events in a range of settings, without these being used then terms such as post-traumatic stress should be avoided.

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Specialist care for prisoners?

In his recent editorial on mental health in prisons Dr Reed (2003) urges, understandably and in most cases correctly, that the quicker that patients with psychosis are transferred to specialist psychiatric care, the better.

However, there are prisoners with schizophrenia, willing to take medication, who survive reasonably comfortably in the prison milieu. Their great fear is that they will be transferred to a special psychiatric hospital; 'nutted off' in prisonspeak. They have reason to fear a transfer, for it effectively exchanges a finite sentence for an indefinite one. In the case of those serving a life sentence, it means their fate is in the hands of a mental health review tribunal rather than the Parole Board, the latter, they believe, being less cautious in recommending discharge. As an ex-member of both organisations, I would agree with them.

So, while prison is obviously bad for people with mental illness, hospital is sometimes worse.

Reed, J. (2003) Mental health care in prisons. *British Journal of Psychiatry*, **182**, 287–288.

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Consent and treatment in prisons

I read the article by Earthrowl *et al* (2003) with interest. The issue of providing treatment to prisoners, who are frequently incapable of consenting, will not be unfamiliar to psychiatrists providing mental health care in these establishments. Although the authors correctly state that there is no legislative framework for providing treatment for mental disorders in prisons, this may be slightly disingenuous. The current legislative framework that provides for the treatment of mental disorders, namely the Mental Health Act 1983, is clear that prison health-care wings are not hospitals. It follows that any treatment that is administered forcibly must be consistent with common law. Separate legislation is therefore unnecessary.

They also appear to have overlooked recent guidance on this matter. The Department of Health (2002) in collaboration with the Prison Service has set out, in detail, good practice guidelines for providing care to both competent and incapacitated adult prisoners. These outline circumstances in which prisoners who lack capacity can receive treatment. We have found this very helpful in developing protocols for treatment in the prisons we visit.

The development of policies and protocols will assist in establishing who, when and in which circumstances incapacitated prisoners may be treated and allow us to be more confident when making these difficult decisions.

Department of Health (2002) *Seeking Consent: Working with People in Prison*. London: Department of Health.

Earthrowl, M., O'Grady, J. & Birmingham, L. (2003) Providing treatment to prisoners with mental disorders: development of a policy. Selective literature review and expert consultation exercise. *British Journal of Psychiatry*, **182**, 299–302.

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Authors' reply: We fail to understand Dr Qurashi's comment that 'separate legislation is therefore unnecessary'. Our paper sets out a *policy* for providing treatment to people with mental disorder based on common law (Earthrowl *et al*, 2003). We are not proposing separate legislation.

Dr Qurashi also mentions that we appear to have overlooked recent guidance from the Department of Health (2002). The Department of Health guidelines were produced in July 2002, after our paper was accepted for publication.

These guidelines provide guidance on establishing capacity but, in our opinion, they do not tackle the practical issues relating to the management of prisoners with mental disorder in any great detail, they do not deal with the ethical issues surrounding the provision of an equivalent service in prisons adequately and detailed guidance on making a concerted effort to obtain treatment under the Mental Health Act in hospital before proceeding with treatment under common law is lacking. In our view, these are serious omissions.

Declaration of interest

J.O. is a member of the Department of Health Prison Expert Group.

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Department of Health (2002) *Seeking Consent: Working with People in Prison*. London: Department of Health.

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Amisulpride-induced mania in a patient with schizophrenia

Numerous case reports of atypical antipsychotics inducing hypomanic/manic symptoms have been published; most concern the use of risperidone and olanzapine (Aubry *et al*, 2000), but quetiapine (Benazzi, 2001) and ziprasidone (Lu *et al*, 2002) have also been implicated. A literature search using Medline and PubMed revealed no such reports associated with amisulpride. Although the manufacturer has accumulated a small number of reports of manic symptoms developing during amisulpride treatment, a recent internal review concluded that no causality could be established (Sanofi-Synthelabo, personal communication, 2002). I report a case of amisulpride-induced mania.

A 17-year-old female with a 4-year history of schizophrenia was commenced on amisulpride for persistent negative symptoms. It was cross-titrated with olanzapine, over a 4-week period, to 400 mg. She continued taking citalopram 20 mg, which had been started 6 months previously on the basis that her negative symptoms could be secondary to a masked depression. On commencement of amisulpride her negative symptoms, as rated on the Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1982), rapidly and linearly improved. Her mood, however, continued to rise and by 3 months she had developed a manic episode without psychotic features. She exhibited insomnia, hyperactivity, distractibility, disinhibition and an abnormally and persistently elevated mood that continued despite the immediate cessation of citalopram. There was no evidence of substance misuse or akathisia. These features improved after halving the amisulpride to 200 mg and re-introducing olanzapine 15 mg. They fully remitted within days of stopping the amisulpride. No other concomitant medication was used. The delay in development of overt manic symptoms may reflect having to overcome a baseline SANS score of 68.

The mechanism of action of mood changes induced by atypical antipsychotics is unknown, with speculation centring exclusively on a 5-HT_{2a}:D_2 economy. Lane *et al* (1998) argue that a higher ratio will increase frontal dopamine release, whereas others point to the combined blockade enhancing the ability of 5-HT_{1a} to release frontal dopamine (Ichikawa *et al*, 2001). These theories do not explain the manicogenic effects of amisulpride, which has no serotonin affinity. I propose that the ability of low doses of amisulpride to differentially block presynaptic D_2 and D_3 autoreceptors enhances dopamine transmission in the frontal cortex and can lead to the development of manic symptoms in susceptible subjects. Presumably this mechanism contributes to its antidepressant efficacy, for which it is used in many countries. The theory implies induction of manic features at low doses only.

Declaration of interest

B.P.M. works for ORYGEN, which has received an unrestricted educational grant from Sanofi-Synthelabo.

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Changing use of ECT

I would like to point out a couple of facts about the decline in electroconvulsive therapy (ECT) use not mentioned by Eranti & McLoughlin (2003) in their recent editorial.

The use of ECT without consent has not declined at all since 1985. There were 3362 people given ECT without their consent under section 58 of the Mental Health Act 1983 in England and Wales in the 2-year period 1985–87, 4454 in 1987–89 and 4463 in 1999–2001, with little change in the years between (Mental Health Act Commission, 1988–2002).

It was the 1970s that saw the greatest decline in ECT use, from an estimated 60 000 courses in Great Britain in 1972 to 30 000 in 1979 (Pippard & Ellam, 1981).

The decline in ECT use over the past 20 years or so has been marked by regional variations. While in England ECT use fell fairly steadily during the 1980s, in Scotland it remained fairly constant during the 1980s and early 1990s and then fell by about a half in the mid-1990s (Freeman *et al*, 2000). In the East Anglian region ECT use actually increased during the 1980s (Pippard, 1992).

Author: Col. 1: written permission for Sanofi-Synthelabo needed

I think it is hard to reconcile these facts with the authors' suggestion that new drugs, improvements in patient care and better appreciation of the indications for ECT are responsible for the decline in ECT; although this would be the most respectable explanation for the decline in use of a treatment which is still described as safe, effective and life-saving – especially since the textbook indications for its use have changed little over the past two or three decades. Is it really the case that fewer people need ECT nowadays – or was it given needlessly to large numbers of people in the recent past? Since no research into the reasons for the decline in the use of ECT has been done, it remains impossible to answer this question with any certainty.

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