Stewart, D. E., Klompenhouwer, J. L., Kendell, R. E., *et al* (1991) Prophylactic lithium in puerperal psychosis. The experience of three centres. *British Journal of Psychiatry*, **158**, 393–397.

V. O'Keane Section of Perinatal Psychiatry, Mother and Baby Unit, Bethlem Royal Hospital, and Institute of Psychiatry, De Crespigny Park, London SE5 8AF, UK. E-mail: v.o'keane@iop.kcl.ac.uk

Authors' reply: We agree with Dr O'Keane regarding the severity and potentially devastating consequences of post-partum psychosis in women with a history of bipolar disorder and assure her that any negative emphasis she detected in our brief comments regarding prophylactic treatment were indeed unintended. The brief report format did not allow us to discuss this aspect of management at length but we have taken up this issue more fully in our recent editorial (Jones & Craddock, 2005).

We would, however, defend our contention that the decision to commence mood-stabilising (or indeed any) medication in women of child-bearing years should follow a 'very careful weighing up of risks and benefits'. Any medication should be started assuming that the women may become pregnant and future pregnancy and contraception should be actively discussed at the earliest possible opportunity.

We would also argue that the evidence base for the use of prophylaxis in women with bipolar illness in the post-partum period is not as robust as would be ideal. As Dr O'Keane has outlined, the literature does support the use of lithium in this context, although the retrospective (and partially overlapping) studies differed in when lithium was commenced – important as there may be practical problems in achieving therapeutic levels quickly following delivery and the onset of puerperal psychosis is typically in the few days following delivery. In our series of 101 women with post-partum psychosis more than half had an onset on days 1–3 with over a fifth on the first post-partum day (further details available from the authors on request). With regard to other mood stabilisers, there are few data in the literature. A recently published study demonstrated no efficacy for sodium valproate (Wisner *et al*, 2004) and, despite anecdotal reports of the benefit of typical or atypical antipsychotic medication as prophylaxis, there are no data regarding their use in this context.

Finally, it is our experience that women have strong views on the acceptability of taking medication during pregnancy and while breast-feeding. This may account for the fact that out of the 54 women in our study who went on to have a further pregnancy, only six took prophylactic medication in the puerperium (lithium or haloperidol). Although only two went on to have a recurrence of puerperal psychosis, the numbers are clearly too small to draw conclusions regarding the efficacy of prophylaxis.

This is an area, therefore, in which management decisions are not straightforward but the frequency and severity of post-partum episodes in women with bipolar disorder must weigh heavily in the risk–benefit analysis. What is needed, we can all agree, is further research to provide empirical data on which clinicians, women, and their families can base these difficult decisions.

Jones, I. & Craddock, N. (2005) Bipolar disorder and childbirth: the importance of recognising risk. *British Journal of Psychiatry*, **186**, 453–454.

Wisner, K. L., Hanusa, B. H., Peindl, K. S., *et al* (2004) Prevention of postpartum episodes in women with bipolar disorder. *Biological Psychiatry*, **56**, 592–596.

I. Jones, E. Robertson-Blackmore, N. Craddock Section of Perinatal Psychiatry, Department of Psychological Medicine, Cardiff University, Heath Park, Cardiff CF14 4XN, UK. E-mail: jonesirl@Cardiff.ac.uk

Value of measuring suicide intent

The paper by Harriss *et al* (2005) addresses the very relevant issue of measuring suicide intent in the evaluation of future suicide risk. Measuring suicide intent is more useful than measuring the lethality of the attempts (i.e. the degree of danger to life resulting from self-injurious behaviour; Beck *et al*, 1975). Assessing the intent can be particularly useful in situations where there is no correlation between the expected and actual outcome of the method used as may happen in those with a low level of literacy. Accuracy of expectations about the likelihood of dying moderates the relationship between suicide intent and medical lethality (Brown *et al*, 2004).

Identifying a cut-off to differentiate between high-intent and low-intent attempts is very difficult. Median scores on the Suicide Intent Scale (SIS) were used by Harriss *et al* (2005) to categorise high-intent and low-intent attempts. Their results showed that women with high intent repeat suicide attempts whereas men with low intent tend to do so. Since there was a gender difference in the median values, the cut-off score used for males (10) was higher than that used for females (8). By virtue of using separate cut-off scores, men were classified as having low intent even if they had similar scores on the SIS to women in the high-intent group, possibly affecting the repetition rates. Quantifying and classifying suicide intent have been approached in different ways by various researchers. Baca-Garcia *et al* (2004) studied the characteristics which influence emergency psychiatrists in decisions to hospitalise after a suicide attempt, and found that a cut-off of 11 on the SIS correctly classified 72% of participants. However the authors clearly acknowledge the advantages of using an extensive clinical checklist over an instrument such as the SIS. Although the SIS was not originally designed to predict repetition of self-harm, it may be possible to identify similar cut-off points to predict the likelihood of repetition of suicide attempts when used with other known risk factors. For any risk assessment to be clinically meaningful it should be based on a composite index which takes into account various factors, including the level of suicide intent, the severity of depression, the degree of hopelessness, the impact of life events and the lethality of the attempt.

Baca-Garcia, E., Diaz-Sastre, C., Resa, E. G., *et al* (2004) Variables associated with hospitalization decisions by emergency psychiatrists after a patient's suicide attempt. *Psychiatric Services*, **55**, 792–797.

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Brown, G. K., Henriques, G. R., Sosdjan, D., *et al* (2004) Suicide intent and accurate expectations of lethality: predictors of medical lethality of suicide attempts. *Journal of Consulting and Clinical Psychology*, **72**, 1170–1174.

Harriss, L., Hawton, K. & Zahl, D. (2005) Value of measuring suicidal intent in the assessment of people attending hospital following self-poisoning or self-injury. *British Journal of Psychiatry*, **186**, 60–66.

C. T. S. Kumar The Maudsley Hospital, Denmark Hill, London SE5 8AZ, UK. E-mail: doctorctskumar@yahoo.co.uk

Free will and volition

Although I agree with Professor Henderson (2005) that we should acknowledge that

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many psychiatric patients have a greater degree of volition, or free will, and hence of moral responsibility, than they are often considered to have, I think that he has made things far too easy for himself.

Professor Henderson has simply assumed that we have free will, at the same time maintaining that 'as brain function comes to be increasingly understood, it is possible that abnormal behaviour will be attributed less to the person's power of choice in regard to action, and more to abnormalities of brain function or genotype'. Both these assumptions are not uncontroversial and would deserve at least some arguments to lend them plausibility. One of many questions which arise here is 'why should only abnormal behaviours be attributed less to the person's power of choice in regard to action and more to abnormal brain function?' Could not normal behaviour equally be attributed less to the free will of the agent and more to normal brain function as we come to understand brain function better? Henderson has given us no reason to think that this could not be the case with normal behaviour as well.

Interestingly Henderson cites Libet *et al* (1999) but curiously omits to mention Libet's famous discovery of a readiness potential arising in the brain some 350 ms before a conscious decision to act is experienced. This finding is usually interpreted as evidence of unconscious initiation of the volitional process, and hence as evidence against freedom of the will. Henderson also quotes Alper (1998): 'Even if human beings are genetically deterministic systems, their behaviour may still be unpredictable and they may still possess free will'. But if our behaviour is unpredictable or random, then we do not have free will, because free will implies that we are autonomous agents who can bring about our actions intentionally.

Alper, J. S. (1998) Genes, free will and criminal responsibility. *Social Science and Medicine*, **46**, 1599–1661.

Henderson, S. (2005) The neglect of volition. *British Journal of Psychiatry*, **186**, 273–274.

Libet, B., Freeman, A. & Sutherland, K. (1999) *The Volitional Brain*. Oxford: Blackwell.

P. Crichton 10 Harley Street, London WIG 9PF, UK. E-mail: Paulcrichton72@aol.com

Author's reply: Dr Crichton's points are most useful. He can be assured that I tried to make the topic as easy as possible for the reader, not for myself. He is correct that I have not considered whether free will really exists, simply choosing to make volition the central topic of the editorial. Yes, what I have said applies just as much to minds free of mental illness. There, biological contributions to behaviour are equally likely to be present. What I wrote deliberately did not consider the unconscious, whether or not its presence might be revealed by readiness potentials preceding an action. We are all aware that psychoanalytic theory has made extensive proposals about unconscious origins for normal behaviour. But psychoanalysis and free will are matters to be considered elsewhere, preferably by philosophers rather than clinicians. For myself, I simply retain an interest in the place of personal responsibility in the presence of mental illness. It has been encouraging that the editorial has already caught the attention of some senior judges and lawyers.

A. S. Henderson 9 Timbarra Crescent, O'Malley, ACT 2606, Australia. E-mail: ashenderson@netspace.net.au

Violence and offending in people with learning disabilities

I found Reed *et al*'s (2004) study fascinating, as it demonstrates the apparently random nature of a forensic label in our patients. It is clearly not to do with risk. I am confused by some of the results. The whole gist of the argument is that the offender group is less violent than their non-offender counterparts. However, it is stated that in the offender group the challenging behaviour diminishes from 0.79 incidents per week to 0.36 and that for the non-offender group from 0.23 to 0.11. This is challenging behaviour generally but this suggests that those in the offender group exhibit greater challenging behaviour throughout their stay than those in the non-offender group. Table 2 states the opposite. I would be interested to see how this inconsistency can be explained.

Reed, S., Russell, A., Xenitidis, K., *et al* (2004) People with learning disabilities in a low secure in-patient unit: comparison of offenders and non-offenders. *British Journal of Psychiatry*, **185**, 499–504.

T. Marshall Community Resource Team for People with a Learning Disability, Ist Floor, The Rutson, High Street, Northallerton, North Yorkshire DL7 8EN, UK. E-mail: Thomas.Marshall@hrpct.nhs.uk

Authors' reply: We would like to point out that we do not maintain that those in the offender group are less violent than their non-offender counterparts. Rather, we conclude that, as stated in the Results section, people in the offender group were significantly more likely to display some types of challenging behaviour but significantly less likely to display others. The results showing a reduction in the frequency of challenging behaviour during admission measured the change in rate of challenging behaviour per person per week by comparing a 4-week baseline period with the last 4 weeks of admission. Thus, these figures do not show the level of challenging behaviour exhibited in each group throughout their stay. The fact that there was no significant between-group difference in the rate of total incidents of challenging behaviour per month is shown correctly in Table 2. We thank Dr Marshall for giving us the opportunity to clarify this point.

S. Reed, D. Murphy, K. Xenitidis, A. Russell Maudsley Centre for Behavioural Disorders, South London and Maudsley NHS Trust and Institute of Psychiatry, London. E-mail: S.Reed@iop.kcl.ac.uk

Escitalopram for social anxiety disorder

We noted the findings of Kasper *et al* (2005) and their conclusion that 'escitalopram was efficacious in treatment of social anxiety disorder' with interest. They reported a difference of 7.3 (P=0.005) on the Liebowitz Social Anxiety Scale (LSAS) from baseline to week 12, favouring escitalopram over placebo. They suggested that this difference was comparable to three previous studies that reported the efficacy of paroxetine in the treatment of social anxiety disorder (Stein *et al*, 1998; Allgulander, 1999; Baldwin *et al*, 1999).

Unfortunately, without the confidence interval (CI), reliable interpretation of the above difference is not possible. Hence we calculated the standardised effect size, which was 0.22 (95% CI 0.01–0.43). Although the lower limit of the CI is not reassuring, by convention, the point estimate of 0.22 can be interpreted as 'small'.

We appreciate that small effect sizes can be clinically relevant, especially if the condition treated is common and the putative treatment is easily available, cheap and without adverse effects. In addition, the given treatment must perform better than

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