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.

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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.

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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.

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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.

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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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other options. We compared the above effect size with the effect sizes for the three studies quoted above. These were 0.83 (95% CI 0.53–1.13), 1.36 (95% CI 0.90–1.80) and 0.38 (95% CI 0.14–0.61), respectively.

We then looked at the number needed to treat (NNT) based on the responders as per the Clinical Global Impression – Improvement (CGI–I) scores. The NNT for the study by Kasper *et al* (2005) is 7 (95% CI 4–20) and for the comparative studies, 4 (95% CI 3–6), 2 (95% CI 2–3) and 3 (95% CI 3–4), respectively. van der Linden *et al* (2000) reported a meta-analysis of the effectiveness of serotonin reuptake inhibitors (SSRIs) in the treatment of social anxiety disorder. They found a collective NNT of 4 (responders on CGI–I) and a mean effect size for all SSRIs of 1.0 (the SSRI/placebo difference at end-point on the LSAS). None of the ten SSRI studies in the meta-analysis included escitalopram.

It is tempting to suggest that the placebo response in the study of Kasper *et al* (2005) was high and distorts results. However, if randomisation is presumed to have been successful, an equivalent placebo effect would have occurred in the escitalopram group. The impressive *P* values reported by Kasper *et al* (2005) are likely to be because their study was overpowered and they used analysis of covariance (ANCOVA) which is known to have greater statistical power.

Based on our analysis, among the different SSRI medications escitalopram is less likely to be effective in the treatment of social anxiety disorder. We suggest that *P* values can mislead and should not be interpreted as measures of magnitude of effect.

Allgulander, C. (1999) Paroxetine in social anxiety disorder: a randomized placebo-controlled study. *Acta Psychiatrica Scandinavica*, **100**, 193–198.

Baldwin, D., Bobes, J., Stein, D. J., *et al* (1999) Paroxetine in social phobia/social anxiety disorder. Randomised, double-blind, placebo-controlled study. Paroxetine Study Group. *British Journal of Psychiatry*, **175**, 120–126.

Kasper, S., Stein, D. J., Loft, H., *et al* (2005) Escitalopram in the treatment of social anxiety disorder: randomised, placebo-controlled, flexible-dosage study. *British Journal of Psychiatry*, **186**, 222–226.

Stein, M. B., Liebowitz, M. R., Lydiard, R. B., *et al* (1998) Paroxetine treatment of generalised social phobia (social anxiety disorder) – a randomised controlled trial. *JAMA*, **26**, 707–713.

van der Linden, G. J., Stein, D. J. & van Balkom, A. J. (2000) The efficacy of the selective serotonin reuptake inhibitors for social anxiety disorder (social phobia): a meta-analysis of randomized controlled trials. *International Clinical Psychopharmacology* (suppl.), **2**, S15–S23.

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Authors' reply: We thank Drs Lele and Joglekar for drawing our attention to the absence of the 95% CIs for the primary efficacy end-point (treatment effect measured as the difference in the Liebowitz Social Anxiety Scale (LSAS) scores from baseline) in our article on the treatment of social anxiety disorder with escitalopram (Kasper *et al*, 2005). The treatment difference between escitalopram and placebo was 7.3 (95% CI 2.2–12.4) with a standardised effect size of 0.30 (95% CI 0.09–0.51).

When comparing the results of this trial with the literature we looked at the size of the effect of the active treatment, that is, the adjusted change from baseline in LSAS scores, not the standardised effect size. These values are 33.0 (Allgulander, 1999), 29.4 (Baldwin *et al*, 1999) and 30.5 (Stein *et al*, 1998), which are comparable to the 34.5 change in our study with escitalopram (Kasper *et al*, 2005). The main difference between these studies is the placebo response, which was largest in our study.

In interpreting differences in placebo response rate (and hence standardised effect sizes) it is important to recognise differences in study design. One of the paroxetine studies (Allgulander, 1999) was a small (n=92) single-centre trial with a 40% placebo withdrawal rate (compared with 18% for paroxetine) and patients were also required to have been treated for at least 2 weeks. These factors may be responsible for the small placebo effect with the last observation carried forward (LOCF) analysis. In the studies of Allgulander (1999) and Stein *et al* (1998) patients were not excluded if they had comorbid depression, which was the case in our study. Finally, in our escitalopram study the mean baseline LSAS scores in the placebo and treatment groups (95.5 and 96.3) were higher than in the paroxetine studies (70.4 and 78.5 in Allgulander, 1999; 78.0 and 83.5 in Stein *et al*, 1998; and 86.1 and 87.6 in Baldwin *et al*, 1999).

We would like to emphasise the appropriate powering of our study. ANCOVA is overpowered if the distribution is skewed but our data are fairly normally distributed. Allgulander (1999) state that their data were skewed and non-parametric tests were used.

In line with the results of our study additional recent data (Lader *et al*, 2004) confirm the efficacy of escitalopram in social anxiety disorder. In a 24-week study the placebo response was 43.4 compared with 60.8 with 20 mg escitalopram and 53.1 with 20 mg paroxetine (mean change from baseline). The treatment difference (observed cases) between escitalopram and placebo was 17.4 (95% CI 11.5–23.2) with a standardised effect size of 0.77 (95% CI 0.51–1.03). The treatment difference for escitalopram and paroxetine (observed cases) was 7.71 (95% CI 2.0–13.4) in favour of escitalopram with a standardised effect size of 0.34 (95% CI 0.09–0.59). After 12 weeks the number needed to treat (NNT) based on the responders as per Clinical Global Impression – Improvement (CGI–I $\leqslant$2, LOCF) scores for Kasper *et al* (2005) was 6.4 (95% CI 4–19) and 4.8 (95% CI 3–10) for Lader *et al* (2004). To judge a single medication based on the NNT it is necessary to consider all available studies and, based on the evidence published in the literature, we therefore do not agree with the statement of Drs Lele and Joglekar that paroxetine is superior to escitalopram for the treatment of social anxiety disorder.

Declaration of interest

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