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.

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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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