CORRESPONDENCE

Orwell lives

Branton & Brookes (2010) end their excellent article with the statement 'This [the changes to the Mental Health Act 1983] could lead to new services, offering compulsory treatment to individuals hitherto unlikely to be held liable to detention'.

'Offering compulsory treatment'? Compulsory treatment isn't 'offered'. Compulsion is removing a person's right to make treatment decisions. It is the denial of personal autonomy. It is replacing an individual's view as to what is in their 'best interest', including the best interest of their own health (e.g. the balance between therapeutic and adverse effects of medication), with the (no doubt well-meaning) opinions of others. It is used even when people are capable of making treatment decisions for themselves.

Compulsion may be necessary but we should never forget, or use language to minimise, its impact on the individuals concerned.

Branton T, Brookes G (2010) Definitions and criteria: the 2007 amendments to the Mental Health Act 1983. *Advances in Psychiatric Treatment* **16**: 161–7.

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CORRECTION

Correction

Cookson J (2010) Seeing the evidence: learning from images in neuroscience. *Advances in Psychiatric Treatment* **16**: 82–85.

The author would like to make the following post-publication clarifications to his article, published in March issue of *Advances*.

On p. 83, the caption to Fig. 1 should read:

> Kandel's model for the cellular and molecular basis of declarative memory formation in the hippocampus. The image combines information gained from his studies of giant marine snail neurons and studies of vertebrate hippocampus. Artist: Philip Wilson.

On p. 84, the fourth paragraph in the left-hand column should end:

> The cellular mechanisms involved in long-term potentiation have also been implicated in hypotheses about the pathophysiology of depression. This was first discussed in detail by Reid & Stewart (2001; see also Zaman & Zaman 2001). The image illustrates a role for dopamine in signalling salience for memory. In the snail, serotonin plays a role similar to that postulated for dopamine in Fig. 1, potentiating glutamate synapses. Harmer *et al* (2009) argue that serotonin plays a part in determining the affective salience of cues, which has relevance for depression. The role of CPEB may cast light on the pathogenesis of prion diseases.

The reference list on p. 84 should include:

> Harmer CJ, Goodwin GM, Cowen PJ (2009) Why do antidepressants take so long to work? A cognitive neuropsychological model of antidepressant drug action. *British Journal of Psychiatry* **195**: 102–8.
>
> Reid IC, Stewart CA (2001) How antidepressants work. New perspectives on the pathophysiology of depressive disorder. *British Journal of Psychiatry* **178**: 299–303.

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