BJPsych | The British Journal of Psychiatry (2010) 197, 499–503

Correspondence

Edited by Kiriakos Xenitidis and Colin Campbell

Contents

Disappearance of Henry Maudsley

In 1991, I published a paper entitled 'Whatever happened to Henry Maudsley?',[1] in which I had deduced that the most likely reason for his sudden and inexplicable disappearance was the onset of an attack of clinical depression.

I further deduced that his malady was primarily precipitated by the death of his wife, although secondary factors were at work of which there are two main ones. The first was that his was a childless marriage so that the loss of his wife resulted in the loss of his only emotional prop; second, his father had behaved in an identical way when his wife, Maudsley's mother, had died.

It was only after the publication of my paper that I realised that my explanation, although certainly feasible, was based on mainly circumstantial evidence, so that, instead of solving the enigma of Maudsley's disappearance, I had complicated it. But it was too late; I had no option but to rest my case. And this is how the position would be today if serendipity had not taken a hand in the game.

It happened that while researching material about the Victorian alienists, I came across a paper, previously unknown to me, by Dr Thomas Walmsley concerning Sir James Crichton-Browne, probably the doyen of psychiatrists at that time.

Dr Walmsley refers in this paper to the occasion when Sir James delivered the first Maudsley lecture to the Royal Medico-Psychological Association in 1920. It is in this paper that Sir James 'recalled the optimistic and energetic Henry Maudsley with whom he had been friendly in the 1860s. With some feeling [the use of this expression is important in that any demonstration of emotion in public at that time would have been considered infra dig], he contrasted the morose and reclusive Maudsley of later years.'[2]

I remember that at this point I emitted a whoop, a mélange of joy and relief – my supposition as to the disappearance of Henry Maudsley had been vindicated!

1 Rollin H. Whatever happened to Henry Maudsley? In *150 Years of British Psychiatry, 1841–1991* (eds GE Berrios, H Freeman): 351–8. Gaskell, 1991.

2 Walmsley T. Crichton-Browne's biological psychiatry. *Psychiatr Bull* 2003; **27**: 20–2.

Henry R. Rollin, c/o Royal College of Psychiatrists, 17 Belgrave Square, London SW1X 8PG, UK. Email: serian@rcpsych.ac.uk

doi: 10.1192/bjp.197.6.499

Serotonin and the mode of action of electroconvulsive therapy

The French philosopher Pierre Charron wrote that the true science and study of man is man. Professor Yatham and colleagues deserve commendation for their application of modern brain imaging techniques to study the mode of action of electroconvulsive therapy (ECT) in living patients with depression.[1] The final assertion that their findings may put to rest the controversy about the role of brain serotonin in mediating the antidepressant effects of ECT may, however, be premature.

The authors suggested a common mode of action among ECT and antidepressant drugs, that is, the down-regulation of brain 5-HT_2 receptors. There is, however, evidence to question the overlap between the mode of action of ECT and antidepressant drugs that target serotonin. Selective serotonin reuptake inhibitors (SSRIs) inhibit the serotonin transporter. The gene that encodes the serotonin transporter has a promoter region that contains a polymorphism, and the allelic status of this polymorphism is associated with the probability of both improvement and recovery with an SSRI. The allelic status of this polymorphism is not associated with the outcome of ECT.[2] A proportion of patients with depression treated successfully with an SSRI will experience transient relapse during acute tryptophan depletion, which in turn depletes serotonin. This is not observed in patients with depression treated successfully with ECT.[3]

There is also evidence to suggest more of an overlap between the mode of action of ECT and antidepressant drugs that target catecholamines. A history of failure to recover with an SSRI during the index episode has no bearing on the probability of remission from unipolar non-psychotic major depression with subsequent ECT; in contradistinction, such a failure with bupropion, which does not target serotonin at all, or a heterocyclic antidepressant is associated with a reduced probability of remission with subsequent ECT. The only known allelic status that is associated with the outcome of ECT in patients with depression concern polymorphisms believed to affect the concentration of dopamine in the forebrain.[4] Modern brain imaging techniques have also been applied to study the effects of ECT on brain dopamine: binding to the D_2 receptor in the rostral anterior cingulate, an area of the brain implicated in the pathophysiology of depressive illness, fell by 25% over a course of bilateral ECT, a finding compatible with an increase in the availability of dopamine.[5]

None of these observations on its own disproves the hypothesis suggested by the authors. Nevertheless, these observations too concern living patients with depression treated by ECT, and together cast doubt on the central role of brain serotonin in the mode of action of ECT in major depression.

1 Yatham LN, Liddle PF, Lam RW, Zis AP, Stoessl AJ, Sossi V, et al. Effect of electroconvulsive therapy on brain 5-HT_2 receptors in major depression. *Br J Psychiatry* 2010; **196**: 474–9.

2 Rasmussen KG, Black JL. Serotonin transporter gene status and electroconvulsive therapy outcomes: a retrospective analysis of 83 patients. *J Clin Psychiatry* 2009; **70**: 92–4.

3 Cassidy F, Murry E, Weiner RD, Carroll BJ. Lack of relapse with tryptophan depletion after successful treatment with ECT. *Am J Psychiatry* 1997; **154**: 1151–2.

4 Domschke K, Zavorotnyy M, Diemer J, Nitsche S, Hohoff C, Baune BT, et al. COMT val 158met influence on electroconvulsive therapy response in major depression. *Am J Med Genet Part B* 2009; **153B**: 286–90.

5 Saijo T, Takano A, Suhara T, Arakawa R, Okumura M, Ichimiya T, et al. Electroconvulsive therapy decreases dopamine D_2 receptor binding in the anterior cingulate in patients with depression: a controlled study using