Correspondence

EDITED BY LOUISE HOWARD

Does electroconvulsive therapy lead to changes in cerebral structure?

Sir: Some retrospective imaging studies have reported an association between a history of electroconvulsive therapy (ECT) and cerebral change, particularly affecting the lateral ventricles and/or cerebral cortex (Weinberger *et al*, 1979; Calloway *et al*, 1981; Andreasen *et al*, 1990). We report the results of a small study of the acute cerebral effects of ECT using recently developed highly sensitive image registration and subtraction techniques applied to high-resolution magnetic resonance imaging brain scans.

A prospective study was carried out in which four ECT-naïve patients with depression underwent 3D high-resolution magnetic resonance imaging less than one week prior to the start of bilateral ECT and again within 24 hours of the administration of first treatment. One of the patients was also scanned six weeks after his first ECT. All examinations were performed on a Picker 1.0 T HPQ system using rf spoiled volume acquisition (TR=21 ms, TE=6 ms, flip angle=35°, 1.6 mm nominally isotropic resolution).

Accurate registrations were performed between the baseline and follow-up scans by using an image registration technique producing alignment of images to a fraction of a voxel, typically less than 0.01 mm in each linear dimension (Hajnal *et al*, 1995*a*). Hajnal *et al* (1995*b*) have demonstrated how subtraction of such aligned images can result in virtually complete cancellation of signals from unchanged structures, thereby providing an unambiguous null condition against which much smaller changes to the brain can be detected than has been possible in previous ECT neuroimaging studies.

The anatomical images and the registered subtraction images (n=4) did not reveal any significant difference in cerebral structure following ECT, either within 24 hours or after six weeks. We conclude that use of one of the most sensitive techniques for detecting cerebral change offers no evidence that ECT leads to acute structural brain changes.

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B. K. Puri, A. Oatridge, N. Saeed Robert Steiner MR Unit, Imperial College School of Medicine, Hammersmith Hospital, Du Cane Road, London W12 0HS

J. E. Ging East Surrey Priority Care NHS Trust, East Surrey Hospital, Canada Avenue, Redhill, Surrey RH1 5RH

H. M. McKee Department of Psychiatry, Charing Cross Hospital, Fulham Palace Road, London W6 8RF

S. K. Lekh East Surrey Priority Care NHS Trust, Redhill & Reigate Community Mental Health Team, Shaw's Corner, Blackborough Road, Reigate, Surrey RH2 7DG

J. V. Hajnal Robert Steiner MR Unit, Imperial College School of Medicine, Hammersmith Hospital, Du Cane Road, London W12 0HS

Risperidone-induced rabbit syndrome

Sir: Rabbit syndrome is an uncommon side-effect of neuroleptic treatment. This syndrome consists of tongue-sparing movements of the mouth, usually after long-term treatment (Schwartz *et al*, 1995). Rabbit syndrome has also been reported, in some cases, as an acute complication of therapy (Todd *et al*, 1983). We describe a case of rabbit syndrome in a patient treated with risperidone.

In March 1989, Mrs G., a 68-year-old with no past history of psychiatric disorder, was admitted to the psychiatric unit because of recent symptoms of acute anxiety and paranoid delusions. She was treated with haloperidol and thioridazine but she developed side-effects. She continued treatment with fluphenazine and benzhexol. Four months later she developed a depressive episode and mianserin 30 mg/day was added. Her condition quickly improved. In August 1996 she had a relapse. In September 1996 we stopped the fluphenazine and started risperidone 4 mg/day. Mianserin was continued in the same dose and full remission was achieved again. Four months later involuntary movements of the mouth appeared. The movements were mainly in the jaw, rapid and on a vertical axis, without lingual involvement. Fine tremor of the head was also present. No other extrapyramidal signs were present. Rabbit syndrome was diagnosed and benzhexol 10 mg/day was started orally. One week later the tremor of the head disappeared and two weeks after beginning treatment the movements of the mouth resolved completely. She continues with the same medication, and during follow-up has been free of clinical extrapyramidal symptoms and signs.

Rabbit syndrome is an iatrogenic syndrome induced by neuroleptics. It has not been reported with other psychotropic drugs, such as anxiolytics, sedative hypnotics or antidepressants (Casey, 1992). In most of the case reports the majority of patients with rabbit syndrome also had other Parkinsonian signs such as tremor, rigidity or bradykinesia. Some authors reported this syndrome as a *forme fruste* of drug-induced parkinsonism (Casey, 1992). In spite of that, treatment with dopamine agonists is not effective in rabbit syndrome. In the present case, rabbit syndrome appeared four months after treatment with risperidone and improved