

Correspondence

The Leicester ECT Trial: Results in Schizophrenia

DEAR SIR,
We read with interest the recent trial of Brandon *et al* regarding the benefits of ECT on schizophrenic symptomatology (*Journal*, February 1985, 146, 177–183). Their conclusion that the improvement seen was due mainly to the beneficial effect of ECT on schizophrenic symptoms and not on affective symptoms is based on the premise that the Montgomery-Åsberg Schizophrenia Scale (MASS) is exclusively a measure of schizophrenic symptomatology. However, this is not entirely the case, for if the MASS is compared to the Montgomery-Åsberg Depression Scale (MADRS) (Montgomery and Åsberg, 1979), then three items are seen to be identical, namely 'inability to feel', 'sadness', and 'pessimistic thoughts'. One other item on the MASS can also relate to affective psychopathology, namely 'other delusions' in the possible form of 'hypochondriacal delusions'. Thus, in total one third of the twelve items of the MASS may be related to affective features and not to core schizophrenic psychopathology. We thus believe that before the authors of the paper can conclude that ECT has had a beneficial effect on the core schizophrenic features of their patients they must demonstrate an improvement, not just in the total MASS score, but in the eight items of the MASS that are free from affective bias.

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Plasma Immunoglobulins in Depressed and Lithium-treated Patients

DEAR SIR,
DeLisi *et al* (*Journal*, December 1984, 145, 661–666) reported that a small group of patients, principally those with a major depressive disorder,

had low plasma concentrations of IgM although their IgA and IgG plasma concentrations were not significantly different from normal control values. The authors suggested that their results provided evidence for the suppression of immunity in some psychiatric patients.

In our investigation only patients diagnosed to be suffering from a major depressive disorder (Spitzer *et al*, 1978) were studied. These female patients were assessed for the severity of depression by the first 16 items of the Hamilton Rating Scale for depression (Hamilton 1967). Only patients who scored 16 or more on this scale after a 7–10 day drug-free assessment phase were included in this study. Women who volunteered to act as control subjects had no known psychiatric illness nor had taken any medication 7–10 days prior to testing. Their plasma IgA, IgG and IgM concentrations were measured using a radial immunodiffusion technique (Immuno Ltd, Sevenoaks, UK).

In order to establish the clinical significance of any changes in immunoglobulin status, euthymic lithium-treated patients were also studied.

Seventeen female patients (13 unipolar and 4 bipolar) who were being treated with long-term lithium prophylaxis for (mean \pm SEM = 5.7 \pm 0.6 yr) were studied. All patients, at the time of testing, were receiving lithium as their sole psychotropic medication. They had not received any other medication 7–10 days prior to testing. Their mean (\pm SEM) plasma lithium concentration approximately 12 hr after their evening dose was 0.89 \pm 0.04 nmol/l.

No statistically significant difference was noted in the plasma concentrations of IgA and IgG between the two patient groups and the normal controls. However, the plasma levels of IgM (mean \pm SEM mg/dl) of the drug-free, acutely depressed patients (106 \pm 14.5) and lithium-treated patients (116 \pm 8.4) were significantly ($P < 0.01$) lower than in the controls (176 \pm 13.2). Since the controls were significantly ($P < 0.05$) younger (43.1 yrs) than either the depressed patients (61.5 yrs) or the lithium-treated patients (58.5 yrs), then the lowered plasma levels of IgM may be a reflection of age rather than predisposition to affective illness. No statistically significant linear relationship could be established between age and IgM plasma concentrations in the

controls, depressed or lithium-treated patients ($r = 0.24, -0.19$ and -0.26 respectively). The mean (\pm SEM) of the plasma levels of IgM in the unipolar patients (108 ± 7.5) was significantly ($P < 0.02$) lower than the controls. The mean (\pm SEM) of the plasma levels of IgM in the bipolar patients was 142 ± 24.5 . It is of interest, therefore, that the abnormality appears to be associated with the unipolar rather than the bipolar form of the illness. However, no definite conclusion can be drawn because of the small number of bipolar patients studied.

The results presented here indicate that depressive patients and lithium-treated patients have lowered plasma IgM concentrations: IgA and IgG concentrations appear to be relatively normal and confirm the results of DeLisi *et al.*

It is worthwhile to note that the pattern of abnormality is present in the euthymic lithium-treated patients, which suggests that we are dealing with a trait associated with vulnerability to affective disorder, particularly unipolar illness.

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Lithium Potentiation of Imipramine in Treatment Resistant Depression

DEAR SIR,

There have been a number of recent reports about the rapid improvement in treatment resistant depression brought about by the addition of lithium carbonate to an antidepressant drug treatment to which the patient has not initially responded (DeMontigny *et al.*, 1981; Nelson & Byck, 1982; Price *et al.*, 1983; DeMontigny *et al.*, 1983; Heninger *et al.*, 1983; Louie & Meltzer, 1984). As this regimen may prove to be useful in some treatment refractory depressed patients we wish to report the first 3 such patients in whom we used lithium potentiation. All 3 patients showed a dramatic, sustained improvement.

Patient 1 was a 52-year old divorced salesman who had a 30 year history of bipolar disorder. He had had 6 admis-

sions. There was no family history of psychiatric disorder. He was admitted with a major depressive episode of 7 weeks duration. His Hamilton depression score was 23 and he was a DST non-suppressor. He was treated with imipramine up to 350 mg a day for 6 weeks without improvement. The imipramine was reduced to 300 mg a day and lithium carbonate 300 mg t.d.s. was added. After 6 days he began to improve and during the next 2 weeks he made a complete recovery. Follow-up two months later revealed that he had remained well and had found a job.

Patient 2 was a 32-year old married woman with a 7 year history of bipolar disorder. She had had 3 admissions. There was no family history of psychiatric disorder. She was admitted with a major depressive episode with psychotic features of 19 months duration. Her Hamilton depression score was 35 and she was a suppressor on the DST. She had earlier had a brief partial remission following a course of 12 ECTs. She had subsequently failed to respond to trials of several tricyclic antidepressants in adequate dosage both alone and in combination with MAOI's and stelazine 30 mg a day. During all this time she remained on lithium carbonate 1,500 mg a day.

Because of the acute suicidal risk she was on 'eye contact' for several weeks. She received a second course of 16 bilateral ECTs which led to a 40% improvement. As she still had depressive hallucinations she was started on Melleril 100 mg four times a day along with imipramine 100 mg four times a day. A month later she was no better. We added lithium carbonate 300 mg t.d.s. with dramatic effect as after 3 days she was significantly improved and over the next 10 days made a 95% improvement. She left hospital on these medications and follow-up 2 months later revealed that she had returned to work.

Patient 3 was a 26-year old single male newspaper reporter. He had a 7 year history of bipolar-II disorder and had had 2 admissions. His father had received ECT for depression. He was admitted with a 5 month history of a major depressive episode with melancholia which had not responded to two courses of 6 ECTs nor to adequate trials of tricyclic and MAOI antidepressants in addition to lithium carbonate 1,500 mg a day. He had a Hamilton depression score of 17 and was a suppressor on the DST.

He was started on imipramine which over 6 weeks was increased to 400 mg daily. He showed no improvement but when lithium carbonate 300 mg t.d.s. was added there was a dramatic effect and within 4 days he was 80% better and over the next week made a complete recovery. He was discharged on these medications though the imipramine was reduced to 300 mg daily. A two month follow-up revealed that he had remained well and had obtained another newspaper reporter job.

All 3 patients were admitted to our research ward after their previous medications had been slowly withdrawn either in their own hospital or as an outpatient. Two were referred as they were treatment refractory. On our ward they were medication-free for at least four weeks while they were investigated. During this time they remained depressed and all 3 were also subsequently refractory to at least one