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Post-ECT Cognitive Defect and Elevation of Blood Pressure

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SUMMARY **There is evidence that ECT causes a breakdown of the blood-brain barrier through the rise of blood pressure it induces. Using the Benton test of cognitive function the day before and three hours after ECT treatment in 27 depressed patients, impairment of function was highly correlated with the maximum rise of systolic BP during the treatment.**

Cerebro-vascular permeability to marker substances during induced epileptic seizures has been studied in experimental animals for more than 40 years. The results show that there is an enhanced penetration of the marker through the blood-brain barrier following prolonged seizures however induced. More recently, Bolwig *et al* (1977a), using the protein horseradish peroxidase as the marker substance, reported staining of brain tissue in some animals following a single electrically induced convulsion. This staining was positively related to the mean arterial BP and the rise of BP associated with the seizure. Spinal cord transection was found to abolish completely both the rise in BP and the associated staining. These investigators argued that the mechanism for these changes was the acute hypertension and not the seizure. Consistent with this view, Westergaard and Bronstedt (1974) demonstrated an increase in permeability of the blood-brain barrier to protein, specifically during acute hypertension induced by metaraminol.

In further investigations, Bolwig *et al* (1977b) have extended the study to psychiatric patients receiving ECT. Using somewhat different methods, these workers demonstrated an increase in the blood-to-brain transfer of the test substance during the short seizure induced by ECT. They presented evidence that the change in permeability was the result of an increase in cerebral blood flow.

In view of the possibility that the breakdown of the blood-brain barrier may account for impaired cognitive function following ECT, it was decided to examine the relation between the rise in BP associated with ECT and impairment of learning and immediate memory after the administration of the treatment.

Method

The group of patients investigated consisted of 17 women and 10 men, aged between 21 and 63 years (median = 35) who had been prescribed a course of ECT for the relief of primary depressive illness. The energy of the stimulus was 25 to 30 joules, the patients were under sodium thiopentone anaesthesia (200–350 mg) and received the muscle relaxant succinyl choline (25–35 mg). In all cases, the patients were judged by the attending psychiatrist to have had a modified grand mal seizure.

Systolic blood pressure was measured before and after the administration of the anaesthetic, after the muscle relaxant and at frequent intervals after the end of the convulsion. Between 8 and 12 readings were taken over a period of 3 to 5 minutes following the convulsion, thereby ensuring that peak elevation of BP had been attained and the BP was returning to the pre-ECT basal level.

Cognitive assessment was carried out with the Revised Benton Visual Retention Test, using forms C and D and administration A, as described by Benton (1963), and with the Inglis Paired Associate Learning Test (Inglis, 1959) forms A and B. Pre-treatment assessments of subjects were made in the afternoon preceding the day on which ECT was administered (in the morning). Post-treatment assessments were

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made 3 to 4 hours after the ECT, using parallel forms of the two tests. The order of presentation of the parallel forms was alternated from subject to subject, to eliminate systematic order effects.

Results

The results of a paired t-test comparing scores on the Benton before and after ECT showed that performance on this test of immediate memory of visual material is impaired by ECT ($t = 3.2$, $p = 0.01$, 2-tailed). There was no impairment on the Inglis test. On the contrary, 11/18 patients (61 per cent) showed improvement on retest compared with 6/27 patients (22 per cent) who showed improvement on the Benton. Thus, both in terms of mean level of performance and total number of individuals showing impaired performance, the Benton test was found to be sensitive to the adverse effect of ECT when testing was carried out within 3 to 4 hours after a single treatment. Because of the insensitivity of the Inglis test, its use was discontinued for the last 9 patients in this series.

Given a test of cognitive function sensitive to the effect of ECT, it was now possible to examine the relationship between rise in BP after ECT and degree of cognitive impairment. The measure of BP rise was the difference between post-anaesthetic level of systolic BP and the maximum level attained after the administration of the ECT (range 10–88 mm Hg, median = 43 mm Hg). A highly significant correlation between BP rise and the pre-ECT minus post-ECT error score on the Benton test was obtained ($r = -0.64$, $p = 0.0005$, 1-tailed). The correlations between age and these two variables was not significant. Not surprisingly, in view of the apparent insensitivity of the Inglis test to cognitive changes produced by ECT, the results with this test did not show a significant relation to rise of blood pressure.

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