

ECT: Brain Damage

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LONG-TERM EFFECTS OF ELECTROCONVULSIVE THERAPY UPON MEMORY AND PERCEPTUAL-MOTOR PERFORMANCE

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PROBLEM

This study investigated whether there are memory and perceptual-motor deficits in patients who have had in excess of 50 electroconvulsive treatments (ECT). A number of investigators have explored the effects of ECT upon psychological tests sensitive to organicity. These researchers usually found decreased performance during and shortly after a course of ECT^(1, 2, 6, 7, 8, 10, 11, 12). There appear to be only two investigations that determined the cognitive effects of ECT after a number of months^(4, 5). However, in both of these studies neither control patients nor an adequate number of ECT patients were employed. In the report of Pascal and Zeaman⁽⁴⁾, a patient's Wechsler-Bellevue and Rorschach scores before 10 ECT and 7 months afterward were comparable. Stone⁽⁵⁾ reported that a patient's Henmon-Nelson Test of Mental Ability score 60 days after the last of 20 ECT was comparable to her score of 7 years earlier.

An appropriate generalization is that the evidence as to whether ECT causes permanent cognitive impairment is inconclusive. The studies reported in the literature have not been controlled adequately for the assessment of such impairment. Furthermore, the number of ECT have been far fewer than in the present research.

METHOD

There were 40 male chronic schizophrenic patients in Jefferson Barracks Veterans Administration Hospital. Twenty patients with a history of 50 or more ECT were assigned to the ECT group, and 20 patients with no record of ECT were matched with individual ECT Ss for age (within 5 years), race, and level of education (within 2 years), and were assigned to the control group. Four Ss were eliminated from the ECT group (two refused to participate and two produced no scorable test responses), and their controls also were dropped. The Bender-Gestalt and the Benton Visual Retention Test (Form C, Administration A) were administered satisfactorily to 16 ECT and 16 control Ss. Table 1 indicates the extent of the between-groups matching. The ECT Ss had received from 50 to 219 ECT with a median of 69.5, and there was a range of 10 to 15 years since the last course of ECT.

TABLE 1. EXTENT OF BETWEEN-GROUP MATCHING AND MEAN BENDER-GESTALT AND BENTON SCORES FOR ECT AND CONTROL GROUP

	ECT Group		Control Group	
	Mean	SD	Mean	SD
Age	45.8	4.2	43.6	4.9
Years of Education	10.9	2.3	10.8	2.4
Years of Hospitalization	19.8	3.6	17.3	2.6
Bender Error Score	69.9	31.6	35.9	15.9
Benton Error Score	19.2	8.1	14.3	6.9
Benton No. Correct	2.6	1.8	3.8	2.4

The Bender-tests that reflect systems. The Pa Gestalt designs (1) the number of scores" that consist of card⁽¹⁾. The ir (p < .005) for 1 error scores, and

As indicated for the ECT group Benton error score (t = 1.90, p < .05) ECT group and 3

For the ECT and Bender-Gestalt Benton error score correct score = 4

The groups were investigated mainly important in this years of hospitalization were .28 for Benton number of were .04, .27, and

The significant Bender-Gestalt at course of treatment more, it seems plausible damaging effect of between number of ECT-produced observations of pre-treated⁽⁴⁾.

Nevertheless, permanent brain damage to receive ECT as it has been reported orders tend to do just as completely positive the ECT psychopathology

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The Bender-Gestalt and Benton were selected because they are well established tests that reflect brain pathology and because they have quantitative scoring systems. The Pascal and Suttell⁽⁴⁾ method of scoring for deviations on the Bender-Gestalt designs was employed. Two scoring systems were used for the Benton: (1) the number of correct reproductions or "number correct scores", and (2) "error scores" that consisted of a detailed analysis of specific errors in each figure of each card⁽⁴⁾. The interscorer reliability coefficients between two scorers were .90 ($p < .005$) for the Bender-Gestalt error scores, .97 ($p < .005$) for the Benton error scores, and .94 ($p < .005$) for the Benton number correct scores.

RESULTS

As indicated in Table 1, the mean error score on the Bender-Gestalt was 69.9 for the ECT group and 35.9 for the control group ($t = 3.84, p < .001$). The mean Benton error score was 19.2 for the ECT group and 14.3 for the control group ($t = 1.90, p < .05$), and the mean Benton number correct score was 2.6 for the ECT group and 3.8 for the control group ($t = 1.62, p < .10$).

For the ECT group, the product moment correlation between number of ECT and Bender-Gestalt error score was .32 ($p < .15$), between number of ECT and Benton error score .62 ($p < .005$), and between number of ECT and Benton number correct score $-.43$ ($p < .05$).

The groups were not matched on length of hospitalization, a variable that some investigators maintain affects test performance. However, this apparently was not important in this study, since the correlation coefficients between test score and years of hospitalization were not significant. For the ECT group, the coefficients were .28 for Bender-Gestalt error score, .05 for Benton error score, and .05 for Benton number correct score. For the control group, the respective correlations were .04, .27, and .12.

CONCLUSIONS

The significantly greater error scores obtained by the ECT Ss on both the Bender-Gestalt and the Benton after a relatively long time period since the last course of treatment suggest that ECT causes irreversible brain damage. Furthermore, it seems plausible that the cognitive impairment results from the cumulative damaging effect of each treatment, particularly in view of the significant correlations between number of ECT and both Benton number correct and error scores. Such ECT-produced structural changes would be consistent with the common clinical observation of progressive mental deterioration of epileptics, especially if untreated⁽⁴⁾.

Nevertheless, it cannot be inferred with complete certainty that ECT causes permanent brain pathology. It is possible that schizophrenic patients more likely to receive ECT are those whose psychotic symptomatology is more severe. And, it has been reported that patients with the so-called functional psychiatric disorders tend to do poorly on tests of organicity⁽¹³⁾. Therefore, one cannot be absolutely positive that the ECT and control groups were equated for degree of pre-ECT psychopathology.

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MALVARIA, THE HOFFER-OSMOND DIAGNOSTIC TEST, AND THE BEHAVIOR OF PATIENTS*

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PROBLEM

Malvaria is a psychiatric disease proposed by Hoffer and Osmond⁽⁷⁾, the criterion for which is simply a mauve chromatograph stain extracted from the urine. Mauve producers were either schizophrenic or displayed features of this diagnosis. Other studies⁽⁸⁾ were less conclusive, but found these patients to be more disturbed, particularly in their thinking. A considerable relationship has been found between the mauve and ingestion of certain tranquillizers⁽⁹⁾, but another investigator⁽⁴⁾ reported that kryptopyrrol produced the mauve. This substance is unlikely to result from tranquillizers. The Hoffer-Osmond⁽⁷⁾ Diagnostic test (HOD), a self-rating set of true-false statements, differentiated between mauve and nonmauve producing patients in the same way that it differentiated between schizophrenics and neurotics⁽⁶⁾.

If malvaria is truly a valid classification or a consequence of medications reliably and validly given for specific psychiatric disorders, then mauve-producing patients should differ from non-mauve producers in terms of objective ratings of symptoms and behavior such as HOD scores.

METHOD

From the psychiatric ward of a teaching general hospital, 82 patients were obtained; all of whom were examined during the first few days after admission. Only 14 were on any tranquillizer, age ranged from 18 to 55, none was an alcoholic, drug addict, psychopath, brain damaged (as far as was known), or below dull-normal intelligence. Their symptoms were rated on the Wittenborn Psychiatric Rating Scales⁽¹⁾, and their ward behavior rated on the Nursing Observation of Behavior Scales⁽²⁾. These measures were filed for scoring at a later date. The mauve and HOD data were excluded from clinical use, and the results were not even known to this investigator until long after the project was completed. Thus, all sets of data were separated to prevent experimental bias as the project proceeded.

*The data were gathered from the psychiatric wards of the University Hospital, Saskatoon, with support from Canadian Mental Health Grants. Analysis was assisted by the Medical Research Council.

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