ECT: Brain Damage

(9)

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[2, 3, 6, 7, 8, 10, 11, 13]. There appear to be only two investigations that determined the cognitive effects of ECT after a number of months[6, 9]. 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[6], a patient's Wechsler-Bellevue and Rorschach scores before 10 ECT and 7 months afterward were comparable. Stone[9] 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

Ss 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

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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[5] 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[1]. 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[12]. Therefore, one cannot be absolutely positive that the ECT and control groups were equated for degree of pre-ECT psychopathology.

References

1. Benton, A. L. *The Revised Benton Visual Retention Test: Clinical and Experimental Applications.* New York: Psychological Corporation, 1963.
2. Erwin, E. F. and Hampe, E. Assessment of perceptual-motor changes following electroshock treatment. *Percept. mot. Skills*, 1966, *22*, 1970.
3. Kendall, B. S., Mills, W. B. and Thale, T. Comparison of two methods of electroshock in their effect on cognitive functions. *J. consult. Psychol.*, 1956, *20*, 423-429.
4. Nielson, J. M. *A Textbook of Clinical Neurology.* New York: Paul B. Hoeber, 1951.
5. Pascal, G. R. and Suttell, B. J. *The Bender-Gestalt Test.* New York: Grune & Stratton, 1951.
6. Pascal, G. R. and Zeaman, J. B. Measurement of some effects of electroconvulsive therapy on the individual patient. *J. abn. soc. Psychol.*, 1951, *46*, 104-115.

7. STONE, C. P. Characteristic losses and gains on the Wechsler Memory Scales as applied on psychotic patients before, during, and after a series of electroconvulsive shocks. *Amer. Psychologist*, 1946, *1*, 245.
8. STONE, C. P. Losses and gains on the Alpha group examination as related to electroconvulsive shocks. *J. comp. physiol. Psychol.*, 1947, *40*, 183-189.
9. STONE, C. P. Pre-illness test records compared with performances during and after electroconvulsive shocks. *J. abn. soc. Psychol.*, 1950, *45*, 154-159.
10. SUMMERSKILL, J., SEEMAN, W. and MEALES, O. W. An evaluation of post electroshock confusion with the Reiter apparatus. *Amer. J. Psychiat.*, 1952, *100*, 835-838.
11. WORCHEL, P. and NARCISO, J. C. Electroshock convulsions and memory: The interval between learning and shock. *J. abn. soc. Psychol.*, 1950, *45*, 85-98.
12. YATES, A. J. The validity of some psychological tests of brain damage. *Psychol. Bull.*, 1954, *51*, 359-379.
13. ZAMORA, E. N. and KAELBLING, R. Memory and electro-convulsive therapy. *Amer. J. Psychiat.*, 1965, *122*, 546-554.

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[6] 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[3], but another investigator[4] reported that kryptopyrrol produced the mauve. This substance is unlikely to result from tranquillizers. The Hoffer-Osmond[5] 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 tranquilliser, 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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SUPPLEMENTUM 77

CEREBRAL CHANGES FOLLOWING ELECTRICALLY INDUCED CONVULSIONS

An Experimental Study on Cats

BY

HANS HARTELIUS

EJNAR MUNKSGAARD
COPENHAGEN 1952

considerably greater possibilities of noting changes in the nerve cells than of discerning a more or less diffuse elective dropping out of cells. The period during which such positive findings may be anticipated appears to be fairly limited; the survival times fixed in the present investigation *i.e.*, 24, 48 and 96 hours and 8 days therefore appear to be suitable. They were actually chosen on the basis of the aforegoing considerations.

In earlier studies on ECT in animals, the changes were also found in those cases in which they could be observed to be distributed throughout the brain but to be somewhat more marked in the frontal parts of the cortex.

Against the background of the aforegoing considerations, my search for irreversible nerve cell changes was focused on two phenomena, *i.e.*, shadow cells and neuronophagia, and the study was confined to the frontal parts of the brain.

A two-grade scale was used to evaluate the shadow cells. The first grade (slight) denotes that the cell damage was possibly irreversible; the second grade (more severe) denotes that the damage was probably irreversible.

The following schema shows how seldom such changes were found at the unbiased examination of specimens from the gyrus sigmoideus anterior, gyrus sigmoideus posterior and gyrus lateralis (*i.e.*, from the frontal parts).

Group	No. of shadow cells: possibly irreversible damage	No. of shadow cells: probably irreversible damage
C	5	
A	6	2
B	7	3

If the extremely large number of nerve cells examined several hundred in each specimen is taken into account, the very small figures are remarkable.

True neuronophagia seemed to be still more rare. It was not observed in any of the control animals and could only be suspected on seven occasions in the animals subjected to ECT. This phenomenon should not be confused with so-called satellitosis. (Reference is also made to the photomicrographs.)

A number of earlier workers have reported disappearance of cells and acellular areas in the cerebral cortex of the experimental animals. I was therefore particularly interested in ascertaining whether such phenomena were present. I found that the cytoarchitecture consistently exhibited some irregularity. The nerve cells were assembled in groups, these groups appearing to have relationship to the vascular architecture. This irregularity seemed to be greater in the deeper layers of the cortex. It is therefore, in all probability, likely to be extremely difficult to determine the possible occurrence of small acellular areas or of an elective dropping out of cells. This has also been pointed out by Scholz[91], among others.

At the unbiased examination, I was unable to find any large necrotic area Single, small areas with *suspected* dropping out of cells were observed sporadically in animals subjected to larger series (11 16) of ECT's and with a long survival time (group B). In only one specimen could this finding be considered as definite. In several cases, however, the suspicion could not be regarded altogether unfounded. Finally, the following facts may be mentioned. It w a question of a few cells in 7 specimens out of the total 282 examined; t phenomenon was not found in any of the control animals. It was seen especial in group B.

The question of whether or not irreversible damage to the nerve cells m occur in association with ECT must therefore be answered in the affirmativ This is the first conclusion to be drawn from the observations reported. T changes found were not, however, extensive; they affected only a small minor of the nerve cells and occurred principally in those animals given the larg series of ECT's. On the other hand, only a very small proportion of the c in the cerebral cortex were examined in the individual animal. In absol figures, the number of damaged nerve cells in the whole cortex should considerably greater. There is, however, no reason to anticipate a lar proportion of damaged cells in other sections of the cortex. On the contra it is possible that they would be less than in the frontal parts.

With regard to the animals given less intensive treatment *i.e.*, 4 EC only it may be concluded that it was not possible to demonstrate any i versible nerve cell damage of any consequence.

The following statement may also be made. It appears almost impossi in the case of animals surviving for a longer period after a series of EC to recognize a dropping out of cells of this type and on the aforementio scale. In my opinion, such cell changes can only be recognized if the mic scopical examination is made in the course of the pathological process, during the days immediately following the ECT. This emphasizes the neces of choosing a suitable survival time for the animals in such neuropatholog experiments.

CHAPTER 17

Considerations on the Pathogenesis of the Cerebral Changes

In the present experiments, the most easily discernible changes in the b of the animals subjected to ECT were found in the vascular system. In e case the blood vessels were dilated and filled with blood. Since this also ap to the control animals, this phenomenon was not considered to be correl