Cognitive Functioning and Degree of Psychosis in Schizophrenics given many Electroconvulsive Treatments

By DONALD I. TEMPLER, CAROL F. RUFF and GLORIA ARMSTRONG

**Problem**

Goldman, Gomer, and Templer (2) found that the Bender-Gestalt and Benton Visual Retention Test performances of male chronic schizophrenic in-patients with a history of 50 or more electroconvulsive treatments (ECT) were significantly inferior to those of control patients matched for age, level of education, and race. However, the authors maintained that it cannot be inferred with certainty that ECT causes permanent brain damage since it is possible that schizophrenic patients more likely to receive ECT are those whose psychosis is more severe. It has been reported that patients with the so-called functional psychiatric disorders tend to do poorly on tests of organicity (5).

The purposes of the present research were (i) to replicate the findings of Goldman et al.; (ii) to compare ECT and control patients on the Wechsler Adult Intelligence Scale (WAIS); and (iii) to compare the degree of psychosis of ECT and control patients.

**Method**

Subjects were 14 male and 30 female schizophrenics in Western State Hospital, Hopkinsville, Kentucky. Of these patients 22 had a history of from 40 to 263 ECT with a median number of 58.5. All ECT was administered earlier than seven years ago. The 22 control patients were matched for age, sex, race, and level of education. Table I indicates the extent of the between-groups matching. All 44 patients were administered the WAIS, the Bender-Gestalt, and the Benton (Form C, Administration A). Ten of the ECT patients and 18 of the control patients were able to complete the Minnesota Multiphasic Personality Inventory (MMPI). The Pascal and Suttell (3) method of scoring for deviations on the Bender-Gestalt was employed. Two scoring systems were used for the Benton: (i) the number of correct reproductions or ‘number correct scores’, and (ii) ‘error scores’ consisting of a detailed analysis of specific errors in each figure of each card (1). The interscorer reliability coefficients between the two scorers were .99 ($p < .01$) for the Bender-Gestalt error scores, .97 ($p < .01$) for the Benton error scores, and .95 ($p < .01$) for the Benton number correct scores.

The MMPI was administered so that the scores of ECT and control patients could be compared both on the Schizophrenia (Sc) Scale and on a special Sc-O Scale developed by Watson (4) to differentiate organics from schizophrenics. The unweighted long form of the Sc-O Scale was employed.

Additional procedures for comparing the degree of psychosis of ECT and control patients entailed the blind rating of two experienced clinical psychologists. These psychologists were requested to sort the 44 sets of answers on the Verbal section of the WAIS into the 22 most psychotic and the 22 least psychotic. The two
psychologists were given the following instructions:

'Place the 44 sets of WAIS answers into two categories, with those of the 22 most psychotic patients in one category and those of the 22 least psychotic patients in the other. Consider looseness of associations, peculiar ideation, idiosyncratic responses, and in general the abnormalities than can be subsumed under "schizophrenic thinking". Try to consider extent of thought disorder rather than number of correct answers or level of intelligence displayed. In like fashion, place the Bender-Gestalt reproductions into two categories of the 22 most psychotic and the 22 least psychotic.'

RESULTS

As indicated in Table I, the mean error score on the Bender-Gestalt was 124.27 for the ECT group and 56.82 for the control group ($t = 3.20, p < .01$). The mean Benton error score was 18.48 for the ECT group and 14.82 for the control group ($t = 2.20, p < .05$), and the mean Benton number correct score was 1.29 for the ECT group and 2.18 for the control group ($t = 1.67, p < .05$). On the WAIS, the ECT and control group respective means were 68.50 and 79.72 for Verbal IQ ($t = 2.46, p < .01$), 65.68 and 75.59 for Performance IQ ($t = 2.02, p < .05$), and 65.73 and 76.77 for Full Scale IQ ($t = 2.33, p < .05$).

For the ECT group, the product-moment correlation coefficient between number of ECT received and Bender-Gestalt error score was -.07 (n.s.); between number of ECT and Benton error score, .34 ($p < .10$); between number of ECT and Benton number correct score, .37 ($p < .05$); between number of ECT and Verbal IQ, .10 (n.s.); between number of ECT and Performance IQ, .34 ($p < .10$); between number of ECT and Full Scale IQ, .26 (n.s.).

The mean MMPI Sc Scale score was 40.90 for the 10 ECT patients who completed the MMPI and 36.50 for the 18 control patients who completed the MMPI ($t = .93$, n.s.). In nine instances both the ECT patients and their control patients completed the MMPI. The mean Sc Scale score for these nine ECT patients was 41.78; the mean of the corresponding nine control patients was 35.89 ($t = 1.07$, n.s.). On the MMPI Sc-O Scale (upon which a higher score indicates a greater probability of organicity and a lesser one of schizophrenia), the 10 ECT patients obtained a mean score of 38.00 and the 18 control patients obtained a mean score of 42.11 ($t = 1.51$, n.s.). For the nine cases in which the ECT patients and their controls both completed the MMPI, the mean Sc-O Scales scores were 38.22 and 45.44 respectively ($t = 2.19, p < .05$).

One of the clinical psychologist raters classified 15 of the ECT patients' WAIS protocols and seven of the control patients' protocols into the '22 most psychotic' category ($x^2 = 5.08, p < .02$). The other clinical psychologist classified 16 ECT patients' protocols and 6 control protocols into the '22 most psychotic' category ($x^2 = 9.08, p < .01$).

The Goldmanaal. findings of ECT patients' inferior Benton and Bender-Gestalt performances were replicated in the present study. The ECT patients' performance was also found to be inferior on the WAIS. However, the ECT patients were found to be more psychotic on all eight indices of psychosis—both of the MMPI Sc Scale score comparisons, both of the Sc-O Scale comparisons, both sets of clinical judgments upon the WAIS, and both sets of clinical judgments upon the Bender-Gestalt. The level of significance is beyond the .05 level in three of these comparisons. Furthermore, for the 10 ECT patients who completed the MMPI, the correlation coefficient between number of ECT received and Sc Scale score is .77 ($p < .01$).

However, the greater degree of psychosis of the ECT patients does not rule out organicity. It is conceivable that they could be both organically damaged and more psychotic. In order to equate both groups for degree of psychosis, the 10 ECT patients who completed the MMPI were matched for MMPI Sc Scale score as ci patients. To these ECT is t 9 point ECT and control mean differ and control 

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score as closely as possible with 10 control patients. The mean absolute difference between these ECT and control patients was almost identical, 49.9 and 46.8 respectively. The respective mean difference for these MMPI matched ECT and control patients is 76.9 and 35.9 (t = 2.28, \( p < .05 \)) for Bender-Gestalt error score; 15.9 and 14.0 (t = 1.01, n.s.) for Benton error score; 2.10 and 2.00 (t = 1.00, n.s.) for Benton number correct score; 77.1 and 82.1 (t = .14, n.s.) for Verbal IQ; 78.3 and 79.0 (t = .24, n.s.) for Performance IQ; and 76.1 and 80.1 (t = .78, n.s.) for Full Scale IQ.

It is apparent that the Benton and WAIS performances of ECT and control patients are very similar when degree of psychosis is controlled for. However, even with the two groups so matched for psychopathology, the ECT patients' Bender-Gestalt performance was significantly inferior to that of the control group. It is not certain why such significance was obtained upon a test of perceptual-motor functioning but not upon tests of memory and general intelligence. However, with the 22 ECT patients and their 22 control patients, the greatest level of significance was obtained with the Bender-Gestalt. Such a finding was also reported in the Goldman et al. study. The ECT patients' inferior Bender-Gestalt performance does suggest that ECT causes permanent brain damage.

Acknowledgments

Appreciation is extended to Cyril and Violet Franks for their judgments of the psychotics of WAIS answers and Bender-Gestalt reproductions.

References


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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, 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

<table>
<thead>
<tr>
<th></th>
<th>ECT Group</th>
<th>Control Group</th>
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<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>Age</td>
<td>45.8</td>
<td>4.2</td>
</tr>
<tr>
<td>Years of Education</td>
<td>10.9</td>
<td>2.3</td>
</tr>
<tr>
<td>Years of Hospitalization</td>
<td>19.8</td>
<td>3.6</td>
</tr>
<tr>
<td>Bender Error Score</td>
<td>60.9</td>
<td>31.6</td>
</tr>
<tr>
<td>Benton Error Score</td>
<td>19.2</td>
<td>8.1</td>
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<tr>
<td>Benton No. Correct</td>
<td>2.6</td>
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As indicated for the ECT group Benton error score (t = 1.90, p < . The ECT and Bender-Gest Benton error score correct score .4.

The groups were matched in this years of hospitalization were .28 for the ECT group and .04 .27, and

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1. BENTON, A. L. 2. ERWIN, E. F. -their effect on cognition.
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\(^6\) 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 .13.

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\(^{a5}\). Therefore, one cannot be absolutely positive that the ECT and control groups were equated for degree of pre-ECT psychopathology.

REFERENCES

MALVARIA, THE HOFER-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 (6) Diagnostic test (HOD), a self-rating set of true-false statements, differentiated between mauve and non-mauve producing patients in the same way that it differentiated between schizophrenics and neurotics (8).

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 tranquilizer, 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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Cognitive Functioning in Depressed Geriatric Patients With a History of ECT

Helen M. Pettinati, Ph.D., and Kathryn M. Bonner

Cognitive functioning in depressed geriatric patients, some with a history of ECT, was assessed with the Trail Making B test. Depressed patients over the age of 65 who had had at least one prior series of ECT performed more poorly on the test than did older patients with no history of ECT and younger depressed patients regardless of their ECT history. The groups did not differ in severity of depression. Careful assessment of elderly patients' history of ECT will allow for more informed decisions about the current use of ECT and an understanding of the cognitive status of these patients.

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Electroconvulsive therapy (ECT) is currently a favored treatment for severe depression in the elderly (1, 2). The physiological changes that accompany the aging process can produce an increased sensitivity to pharmacological side effects and toxicity that precludes widespread use of antidepressants in this population (3, 4). Although no long-term adverse effects of ECT have been documented, transient retrograde and anterograde amnesia often occur, especially with bilateral electrode placement (5-7). Fraser (1) reported finding transient deficits in learning and long: recovery time (marked by confusion and disorientation) in older depressed patients following a series of bilateral ECT compared with younger depressed patients. He attributed the longer duration of postictal confusion to the potential cumulative effect of ECT in the elderly.

The temporary deficits in new learning (short-term memory) following recent bilateral ECT have been extensively reported (5). Atteptional deficits (usually associated with depression) and short-term memory deficits (dysfunction usually associated with the ECT amnestic syndrome, anterograde) have been reported for both younger and older depressed patients (8). However, the prolongation in the elderly patient of the acute organic brain syndrome that typically is seen immediately after ECT administration raises the possibility that the depressed elderly patient may react differently to an accepted form of treatment. Thus, older patients often display more prolonged confusion and disorientation than do younger patients.

Potentially confounding the clinical observation that cognitive recovery is slow-paced for the depressed aged individual is the likelihood that these patients have already received ECT in previous hospital admissions. There are reports that an extended history of ECT (say, 50 or more treatments) may produce longer-lasting cognitive dysfunction (5, 9). A history of ECT typically aids the clinician in determining how useful ECT may be for treatment during the current admission. However, there is little empirical evidence about the impact of a history of ECT on subsequent recovery time from a current psychiatric illness or on the individual's ability to assimilate side effects of both ECT and medication.

We retrospectively examined the possible effects of past ECT administration in a sample of depressed elderly patients who were scheduled to receive ECT during their current admission. We compared their performance on the Trail Making B test (10), a neuropsychological measure of organic brain dysfunction, with that of younger depressed individuals who also were scheduled for ECT. We took a history of ECT for each patient so that performance related to age could be distinguished from performance related to a history of ECT.

METHOD

The sample for this study was drawn from inpatients at the Carrier Foundation with a diagnosis of major depressive disorder or schizoaffective disorder according to DSM-III criteria who were scheduled to receive ECT and who had agreed to participate in another research study involving drug effects on ECT (11). Twenty patients (13 women and seven men) were age 65 or over, with an average (±SD) age of 69.7±3.76 years. Forty-one patients (29 women and 12 men) were under age 65, with an average age of 41.7±12.43

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years. No patient had received ECT for at least 3 months before the current hospital admission.

As part of an ongoing study comparing bilateral and nondominant unilateral ECT, we administered a test battery designed by one of us (H.M.R) to assess cognitive functioning to each patient 3 to 6 hours before his or her first ECT treatment. The test battery included two measures of attention, one of short-term memory (two verbal and two nonverbal), and of long-term memory (one verbal and one nonverbal). In addition, the Trail Making B test from the Halstead-Reitan Neuropsychological Battery (12) was included to assess confusion and more general cognitive functioning than is measured by the other tests in the battery. Since the Trail Making B test was designed to be sensitive to "organic brain dysfunction" and reflects impairment associated with several different brain locations, it is useful as a screening instrument for the longer-lasting cognitive deficits due to ECT that might be expected in the aged (10).

The Trail Making B test requires basic motor and spatial skills, the ability to count and to follow a complex plan, and cognitive flexibility. Each patient is instructed to connect numbers and letters in sequential order beginning with number 1, then proceeding to letter A, number 2, letter B, and so forth. The patient is interrupted and corrected when an error is made, then instructed to proceed from that point. Total completion time, including time for corrections, is recorded in seconds.

Cognitive functioning can also be affected by severity of depression (13), especially in elderly patients suffering from pseudodementia (14). In addition to making a DSM-III diagnosis, a psychiatrist completed the Hamilton Rating Scale for Depression and the Brief Psychiatric Rating Scale (BPRS). The patients were asked to complete the Beck Depression Inventory and the SCL-90-R (15).

Following data collection, we assessed the patients' history of ECT through chart review. Patients were rated as having had none, one, or two or more previous ECT series, and we noted how long it had been since the last ECT series. Information was based on patient reports at admission, typically corroborated by a relative. We could only verify these reports when the treatment had been previously administered at Carrier, which was the case for approximately 54% of ECT series reported. Three patients (two older and one younger) could not be specific about the actual number of series and were conservatively assigned to having had one prior series. All except two younger patients with a history of ECT gave the specific date of their last ECT series. These two patients were included in all the analyses except the one comparing younger and older patients in time elapsed since last ECT. Electrode placement and the number of treatments per series in prior admissions were largely unrecorded. Chart review was conducted without knowledge of the results of the Trail Making B test, and the reviewer was unaware of the purpose of this study.

RESULTS

Of the 20 older depressed patients, only five (25%) had not received any prior ECT. In contrast, 23 (56%) of the younger depressed patients had no prior ECT. Not surprisingly, older depressed patients were significantly more likely to have received previous ECT treatments ($\chi^2=5.24$, df=1, $p<.02$).

According to psychiatrist ratings, the older patients were not more depressed than the younger ones. On the Hamilton depression scale, older patients had a mean (±SD) of 19.60±7.28 compared with a mean of 20.22±7.24 for younger patients; on the BPRS, older patients scored an average of 36.10±9.23 while younger patients scored an average 37.17±9.00. Older patients rated themselves as less depressed than the younger patients did, with a mean on the Beck inventory of 22.92±14.17 for older patients versus 31.45±12.29 for younger ones ($t=2.28$, df=52, $p<.05$, two-tailed) and means on the SCL-90-R of 1.18±0.66 for older patients and 1.67±0.74 for younger ones ($t=2.28$, df=50, $p<.05$, two-tailed).

The most important findings assessing the effects of age and history of ECT were evaluated by two-way analysis of variance, with the number of seconds required to complete the Trail Making B test as the dependent variable. This analysis, illustrated in figure 1, yielded a significant interaction ($F=3.27$, df=2, 55, $p<.05$). That is, the length of time required to complete the test increased if the patient both was over the age of 65 and had a history of ECT. Specifically, cognitive functioning was more deficient in the depressed patients in the presence of two factors: age and history of ECT.

Two-tailed t tests were conducted on the data measuring performance on the Trail Making B test for both older and younger patients who had received one prior series of ECT; older patients had a mean (±SD) score of 386.40±218.21 seconds versus 231.25±95.63 seconds for the younger patients ($t=1.9$, df=11, $p<.10$). For patients who had received at least two series of ECT, older patients had a mean score on the test of 535.50±326.99 seconds versus 202.10±109.39 seconds for younger patients ($t=4.4$, df=18, $p<.005$). In both cases the older patients required more time to complete the test.

Older patients with no history of ECT had a mean score of 218.40±64.46 seconds, which was significantly less time than that needed by older patients with a history of ECT, who had a mean score of 485.80±296.03 seconds ($t=3.08$, df=18, $p<.01$). The two groups did not differ significantly in mean age (±SD) (69.80±4.09 years versus 69.67±3.79 years, respectively). In addition, older depressed patients with no history of ECT (mean score=218.40 seconds) were more likely to perform in the same way as the younger depressed patients with no prior ECT (mean score=166.61±98.49 seconds) as well as those younger depressed patients with prior ECT (mean=215.06±101.60 seconds). There were no significant

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differences between younger patients with and without a history of ECT in the amount of time needed to complete the B test.

Because there were only five patients over age 65 with no history of ECT, the results, although statistically significant, may capitalize on chance factors. Therefore, we did a regression analysis assessing the relative effects of age and prior ECT on test performance (the dependent variable) for all 61 patients, so that age was treated as a continuous rather than a dichotomous variable. Severity of depression at the time of testing as measured by the Hamilton depression scale was covaried to equate severity of depression across all ages. There still was a significant interaction (F=3.42, df=1, 57, p<.07) between age and prior ECT, indicating that as the patients got older (not necessarily over 65 years) and had a history of ECT, deficits in cognitive functioning became more pronounced.

Although we expected an age differential in cerebral impairment only on the Trail Making B Test, we did a similar analysis on the other tests in the cognitive battery. For all ages, prior ECT did correlate with the short-term verbal memory tests but not with the tests of attention or long-term memory. Significant main effects for prior ECT were found on the paired associates test (F=4.85, df=1, 59, p<.05) and the short story test (F=3.00, df=1, 55, p<.08) from the Wechsler Memory Scale (16). There was no differential effect for the older individual.

Differences in cognitive functioning due to both age and history of ECT are important only if these deficiencies cannot be attributed to more severe depression in the older patient with a history of ECT. A two-way analysis of variance assessing severity of depression at admission (based on the psychiatrist's rating on the Hamilton depression scale) was performed on the sample who had completed the Trail Making B test according to age and amount of prior ECT a patient had received. As illustrated in figure 2, of the six possible groups no one group of patients was significantly more depressed than any other group. The older patients who previously had received ECT were not more depressed on admission than older patients who had no history of ECT or than any of the younger groups of patients.

We performed a regression analysis assessing performance on the Trail Making test by age and the number of months since the last ECT for those patients who reported prior ECT. The number of months since the last ECT series did not account for the results over and above age; therefore there was no relationship between time since the last ECT and the performance on the test. Data were available for 16 of the 18 younger patients and for all 15 of the older patients with a history of ECT. Younger patients had a mean (±SD) of 65.06±92.26 months since last series of ECT and older patients had a mean of 52.20±77.41 months. Thus, both groups averaged at least 4 years
since last ECT and there was no statistical difference between the two groups. Six (40%) of these 15 older patients and seven (44%) of these 16 younger patients had had ECT within a year of our study.

The mean (±SD) number of series of prior ECT reported in the patients' history was not significantly different for younger patients (2.28±1.49) than for older patients (2.87±2.42).

There was a nonsignificant trend reflecting a relationship between the number of prior series of ECT and more deficient performance on the test among the older patients (r=.37, df=13, n.s.) which was directly opposite to and significantly greater than (p<.05) that found in younger patients (r=-.22, df=16, n.s.).

DISCUSSION

In this study, elderly depressed patients with a history of ECT were found to be more likely than similar patients with no history of ECT or younger depressed patients regardless of their history for ECT to show some cognitive dysfunction. The difference cannot be laid to a more serious depression, a more extensive history of ECT, or more recent ECT treatments.

Although this retrospective small-group study must be interpreted cautiously, it suggests that the depressed older patient with a history of ECT may be functioning less efficiently than older depressed patients who have never received ECT. The results, if replicated, suggest longer lasting effects of ECT in elderly patients. There is, however, at least one other possible interpretation of this finding. We were unable to assess electrode placement in prior treatments accurately. Since bilateral ECT is more extensively used, it is possible that the cognitive effects noted in those patients with a history of ECT can be attributed to previous bilateral electrode placement. Although this hypothesis might be dismissed, since younger depressed patients with a similar history did not show this effect (and their chances of having had unilateral placement are probably only slightly greater), it is possible that bilateral ECT has potentially more side effects for the older patient. That possibility would support Fraser's (1) recommendation to use nondominant unilateral ECT to minimize the cognitive side effects of ECT in the elderly.

When one is evaluating treatment alternatives for the older patient, focusing on the patient's history of ECT along with other significant ECT treatment variables such as monitoring seizure length, deciding on unilateral versus bilateral electrode placement and so forth (17) is not unlike the strict monitoring of psychopharmacologic treatments due to such factors as differences in drug half-life and absorption rates for patients of all ages. Thus, the "proper dosage" of ECT may need to be more carefully evaluated for older patients just as special monitoring of psychotropic medications is required with these patients. A history of ECT, therefore, may be an important consideration in evaluating the level of functioning of a depressed older patient and a significant factor in shaping expectations for the course of recovery.

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Cognitive Functioning and Degree of Psychosis in Schizophrenics given many Electroconvulsive Treatments

By DONALD I. TEMPLER, CAROL F. RUFF and GLORIA ARMSTRONG

**Problem**

Goldman, Gomer, and Templer (2) found that the Bender-Gestalt and Benton Visual Retention Test performances of male chronic schizophrenic in-patients with a history of 50 or more electroconvulsive treatments (ECT) were significantly inferior to those of control patients matched for age, level of education, and race. However, the authors maintained that it cannot be inferred with certainty that ECT causes permanent brain damage since it is possible that schizophrenic patients more likely to receive ECT are those whose psychosis is more severe. It has been reported that patients with the so-called functional psychiatric disorders tend to do poorly on tests of organicity (5).

The purposes of the present research were (i) to replicate the findings of Goldman et al.; (ii) to compare ECT and control patients on the Wechsler Adult Intelligence Scale (WAIS); and (iii) to compare the degree of psychosis of ECT and control patients.

**Method**

Subjects were 14 male and 30 female schizophrenics in Western State Hospital, Hopkinsville, Kentucky. Of these patients 22 had a history of from 40 to 263 ECT with a median number of 58.5. All ECT was administered earlier than seven years ago. The 22 control patients were matched for age, sex, race, and level of education. Table I indicates the extent of the between-groups matching.

All 44 patients were administered the WAIS, the Bender-Gestalt, and the Benton (Form C, Administration A). Ten of the ECT patients and 18 of the control patients were able to complete the Minnesota Multiphasic Personality Inventory (MMPI). The Pascal and Suttell (3) method of scoring for deviations on the Bender-Gestalt was employed. Two scoring systems were used for the Benton: (i) the number of correct reproductions or `number correct scores', and (ii) `error scores' consisting of a detailed analysis of specific errors in each figure of each card (1). The interscorer reliability coefficients between the two scorers were .99 (p < .01) for the Bender-Gestalt error scores, .97 (p < .01) for the Benton error scores, and .95 (p < .01) for the Benton number correct scores.

The MMPI was administered so that the scores of ECT and control patients could be compared both on the Schizophrenia (Sc) Scale and on a special Sc-O Scale developed by Watson (4) to differentiate organics from schizophrenics. The unweighted long form of the Sc-O Scale was employed.

Additional procedures for comparing the degree of psychosis of ECT and control patients entailed the blind rating of two experienced clinical psychologists. These psychologists were requested to sort the 44 sets of answers on the Verbal section of the WAIS into the 22 most psychotic and the 22 least psychotic. The two

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<th>Extent of between-group matching and mean Bender-Gestalt, Benton, and WAIS scores for ECT and control groups</th>
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<td>ECT group</td>
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<tr>
<td>Mean S.D.</td>
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<tr>
<td>Age 43.86 ± 10.99</td>
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<tr>
<td>Years of education 9.86 ± 5.47</td>
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<tr>
<td>Bender error score 124.17 ± 87.32</td>
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<tr>
<td>Benton error score 18.48 ± 5.28</td>
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<tr>
<td>Benton no. correct 1.29 ± 1.76</td>
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<tr>
<td>WAIS verbal IQ 68.50 ± 16.86</td>
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<tr>
<td>WAIS performance IQ 65.68 ± 17.67</td>
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<td>WAIS full scale IQ 65.73 ± 16.87</td>
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Gestalt was employed. Two scoring systems were used for the Benton: (i) the number of correct reproductions or 'number correct scores', and (ii) 'error scores' consisting of a detailed analysis of specific errors in each figure of each card (1). The interscorer reliability coefficients between the two scorers were .99 (p < .01) for the Bender-Gestalt error scores, .97 (p < .01) for the Benton error scores, and .95 (p < .01) for the Benton number correct scores.

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psychologists were given the following instructions:

'Place the 44 sets of WAIS answers into two categories, with those of the 22 most psychotic patients in one category and those of the 22 least psychotic patients in the other. Consider looseness of associations, peculiar ideation, idiosyncratic responses, and in general the abnormalities than can be subsumed under “schizophrenic thinking”. Try to consider extent of thought disorder rather than number of correct answers or level of intelligence displayed. In like fashion, place the Bender-Gestalt reproductions into two categories of the 22 most psychotic and the 22 least psychotic.'

RESULTS

As indicated in Table I, the mean error score on the Bender-Gestalt was 124.27 for the EOT group and 56.82 for the control group ($t = 3.20, p < .01$). The mean Benton error score was 18.48 for the EOT group and 14.82 for the control group ($t = 2.20, p < .05$), and the mean Benton number correct score was 1.29 for the EOT group and 2.18 for the control group ($t = 1.67, p < .05$). On the WAIS, the EOT and control group respective means were 68.50 and 79.72 for Verbal IQ ($t = 2.45, p < .01$), 65.68 and 75.59 for Performance IQ ($t = 2.02, p < .05$), and 65.73 and 76.77 for Full Scale IQ ($t = 2.32, p < .05$).

For the EOT group, the product-moment correlation coefficient between number of EOT received and Bender-Gestalt error score was -.07 (n.s.); between number of EOT and Benton error score, .34 ($p < .10$); between number of EOT and Benton number correct score, .37 ($p < .05$); between number of EOT and Verbal IQ, .10 (n.s.); between number of EOT and Performance IQ, .34 ($p < .10$); between number of EOT and Full Scale IQ, .26 (n.s.).

The mean MMPI Sc Scale score was 40.90 for the 10 EOT patients who completed the MMPI and 36.50 for the 18 control patients who completed the MMPI ($t = .93$, n.s.). In nine instances both the EOT patients and their control patients completed the MMPI. The mean Sc Scale score for these nine EOT patients was 41.78; the mean of the corresponding nine control patients was 35.89 ($t = 1.07$, n.s.). On the MMPI Sc-O Scale (upon which a higher score indicates a greater probability of organicity and a lesser one of schizophrenia), the 10 EOT patients obtained a mean score of 38.00 and the 18 control patients obtained a mean score of 42.11 ($t = 1.51$, n.s.). For the nine cases in which the EOT patients and their controls both completed the MMPI, the mean Sc-O Scales scores were 38.22 and 45.44 respectively ($t = 2.19, p < .05$).

One of the clinical psychologist raters classified 15 of the EOT patients’ WAIS protocols and seven of the control patients’ protocols into the ‘22 most psychotic’ category ($x^2 = 5.08, p < .02$). The other clinical psychologist classified 16 EOT patients’ protocols and 6 control protocols into the ‘22 most psychotic’ category ($x^2 = 4.08, p < .05$).

CONCLUSIONS AND DISCUSSION

The Goldman et al. findings of EOT patients’ inferior Benton and Bender-Gestalt performances were replicated in the present study. The EOT patients’ performance was also found to be inferior on the WAIS. However, the EOT patients were found to be more psychotic on all eight indices of psychoses—both of the MMPI Sc Scale score comparisons, both of the Sc-O Scale comparisons, both sets of clinical judgements upon the WAIS, and both sets of clinical judgements upon the Bender-Gestalt. The level of significance is beyond the .05 level in three of these comparisons. Furthermore, for the 10 EOT patients who completed the MMPI, the correlation coefficient between number of EOT received and Sc Scale score is .77 ($p < .01$).

However, the greater degree of psychosis of the EOT patients does not rule out organicity. It is conceivable that they could be both organically damaged and more psychotic. In order to equate both groups for degree of psychosis, the 10 EOT patients who completed the MMPI were matched for MMPI Sc Scale score as close as possible. The 10 EOT patients all had an MMPI Sc score of 38.90 or less; the 10 control patients all had an MMPI Sc score of 40.90 or less. According to the clinical psychologists, 7 of the 10 EOT patients and 3 of the 10 control patients were more psychotic than this cut-off point ($x^2 = 3.27, p < .10$). The other clinical psychologist classified 15 EOT patients’ Bender-Gestalt reproductions and 6 control patients’ reproductions into the ‘22 most psychotic’ category ($x^2 = 3.08, p < .05$).

One of the clinical psychologists classified 15 of the EOT patients’ WAIS protocols and 8 of the control patients’ Bender-Gestalt reproductions into the ‘22 most psychotic’ category ($x^2 = 5.08, p < .02$). The other clinical psychologist classified 16 EOT patients’ Bender-Gestalt reproductions and 7 control patients’ reproductions into the ‘22 most psychotic’ category ($x^2 = 4.08, p < .05$).

It is apparent that the performance of the EOT patients was very similar to that of the control patients. The EOT patients’ performance was not significantly different from the control patients’ performance upon a test of this nature.

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The mean absolute difference between the ECT and control patients on the Sc Scale was 0.5 points. The mean Sc Scale scores for the ECT and control patients were almost identical, 40.9 and 40.8, respectively. The respective mean difference for these MMPI-matched ECT and control patients is 0.6 and 0.5, respectively, with p < 0.05 for Bender-Gestalt error score; 15.9 and 14.9 for Benton error score; 210 and 200 for Benton number correct score; 78.3 and 79.8 for Verbal IQ; 76.2 and 80.2 for Performance IQ; and 1078 and 780 for Full Scale IQ.

It is apparent that the Benton and WAIS performances of ECT and control patients are very similar when degree of psychosis is controlled for. However, even with the two groups so matched for psychopathology, the ECT patients' Bender-Gestalt performance was significantly inferior to that of the control group. It is not certain why such significance was obtained upon a test of perceptual-motor functioning but not upon tests of memory and general intelligence.

ACKNOWLEDGEMENT

Appreciation is extended to Cyril and Violet Franks for their judgments of the psychoticism of WAIS answers and Bender-Gestalt reproductions.

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

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VA Hospital, Jefferson Barracks, Mo.

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, 3, 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 (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>
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<tr>
<th>Table 1. Extent of Between-Group Matching and Mean Bender-Gestalt and Benton Scores for ECT and Control Group</th>
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As indicated for the ECT group, Benton error scores (r = .90, p < .001) ECT group and 3. For the ECT and Bender-Gestalt, Benton error scores, correct score = .4. The groups with investigators main important in this years of hospitali were .28 for Bent, Benton number c were .04, .27, and .04.

The significant Bender-Gestalt at course of treatment more, it seems the damaging effect of between number of ECT-produced str observation of pre treated.

Nevertheless, permanent brain p to receive ECT at it has been report orders tend to do not; potentially positive the ECT psychopathol

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 method of scoring 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. 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.

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

REFERENCES


MALVARIA, THE HOFER-OSMOND DIAGNOSTIC TEST, AND THE BEHAVIOR OF PATIENTS*
W. J. CRAIG
Queen's University and Kingston (Ontario) Psychiatric Hospital

PROBLEM
Malvaria is a psychiatric disease proposed by Hoffer and Osmond (1), 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 (2) 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 non-mauve 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 (7), and their ward behavior rated on the Nursing Observation of Behavior Scales (8). 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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Cognitive Functioning in Depressed Geriatric Patients With a History of ECT

Helen M. Pettinati, Ph.D., and Kathryn M. Bonner

Cognitive functioning in depressed geriatric patients, some with a history of ECT, was assessed with the Trail Making B test. Depressed patients over the age of 65 who had had at least one prior series of ECT performed more poorly on the test than did older patients with no history of ECT and younger depressed patients regardless of their ECT history. The groups did not differ in severity of depression. Careful assessment of elderly patients' history of ECT will allow for more informed decisions about the current use of ECT and an understanding of the cognitive status of these patients.

(Am J Psychiatry 141:49–52, 1984)

Electroconvulsive therapy (ECT) is currently a favored treatment for severe depression in the elderly (1, 2). The physiological changes that accompany the aging process can produce an increased sensitivity to pharmacological side effects and toxicity that precludes widespread use of antidepressants in this population (3, 4). Although no long-term adverse effects of ECT have been documented, transient retrograde and anterograde amnesia often occur, especially with bilateral electrode placement (5–7). Fraser (1) reported finding transient deficits in learning and recovery time (marked by confusion and disorientation) in older depressed patients following a series of bilateral ECT compared with younger depressed patients. He attributed the longer duration of postictal confusion to the potential cumulative effect of ECT in the elderly.

The temporary deficits in new learning (short-term memory) following recent bilateral ECT have been extensively reported (5). Attributable deficits (usually associated with depression) and short-term memory deficits (dysfunction usually associated with the ECT amnestic syndrome, anterograde) have been reported for both younger and older depressed patients (8). However, the prolongation in the elderly patient of the acute organic brain syndrome that typically is seen immediately after ECT administration raises the possibility that the depressed elderly patient may react differently to an accepted form of treatment. Thus, older patients often display more prolonged confusion and disorientation than do younger patients.

Potentially confounding the clinical observation that cognitive recovery is slow-paced for the depressed aged individual is the likelihood that these patients have already received ECT in previous hospital admissions. There are reports that an extended history of ECT (say, 50 or more treatments) may produce longer-lasting cognitive dysfunction (5, 9). A history of ECT typically aids the clinician in determining how useful ECT may be for treatment during the current admission. However, there is little empirical evidence about the impact of a history of ECT on subsequent recovery time from a current psychiatric illness or on the individual's ability to assimilate side effects of both ECT and medication.

We retrospectively examined the possible effects of past ECT administration in a sample of depressed elderly patients who were scheduled to receive ECT during their current admission. We compared their performance on the Trail Making B test (10), a neuropsychological measure of organic brain dysfunction, with that of younger depressed individuals who also were scheduled for ECT. We took a history of ECT for each patient so that performance related to age could be distinguished from performance related to a history of ECT.

METHOD

The sample for this study was drawn from inpatients at the Carrier Foundation with a diagnosis of major depressive disorder or schizoaffective disorder according to DSM-III criteria who were scheduled to receive ECT and who had agreed to participate in another research study involving drug effects on ECT (11). Twenty patients (13 women and seven men) were age 65 or over, with an average (±SD) age of 69.7 ± 3.76 years. Forty-one patients (29 women and 12 men) were under age 65, with an average age of 41.7 ± 12.45 years.

Received July 29, 1982; revised Dec. 27, 1982, and March 8, 1983; accepted April 4, 1983. From the Research Division, Carrier Foundation, Belle Meade, NJ. Address reprint requests to Dr. Pettinati, Research Division, Carrier Foundation, Belle Mead, NJ 08502.

This research was supported by the Carrier Foundation. The authors thank the medical and research staffs of the Carrier Foundation, especially Frederick J. Evans, Ph.D., Kenneth S. Mathisen, Ph.D., Joanne Rosenberg, M.S., Marie Mager, Kathleen Meyers, M.S., and Leslie Martin for their assistance.

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years. No patient had received ECT for at least 3 months before the current hospital admission.

As part of an ongoing study comparing bilateral and nondominant unilateral ECT, we administered a test battery designed by one of us (H.M.P.) to assess cognitive functioning to each patient 3 to 6 hours before his or her first ECT treatment. The test battery included two measures of attention, four of short-term memory (two verbal and two nonverbal), and two of long-term memory (one verbal and one nonverbal). In addition, the Trail Making B test from the Halstead-Reitan Neuropsychological Battery (12) was included to assess confusion and more general cognitive functioning than is measured by the other tests in the battery. Since the Trail Making B test was designed to be sensitive to “organic brain dysfunction” and reflects impairment associated with several different brain locations, it is useful as a screening instrument for the longer-lasting cognitive deficits due to ECT that might be expected in the aged (10).

The Trail Making B test requires basic motor and spatial skills, the ability to count and to follow a complex plan, and cognitive flexibility. Each patient is instructed to connect numbers and letters in sequential order beginning with number 1, then proceeding to letter A, number 2, letter B, and so forth. The patient is interrupted and corrected when an error is made, then instructed to proceed from that point. Total completion time, including time for corrections, is recorded in seconds.

Cognitive functioning can also be affected by severity of depression (13), especially in elderly patients suffering from pseudodementia (14). In addition to making a DSM-III diagnosis, a psychiatrist completed the Hamilton Rating Scale for Depression and the Brief Psychiatric Rating Scale (BPRS). The patients were asked to complete the Beck Depression Inventory and the SCL-90-R (15).

Following data collection, we assessed the patients’ history of ECT through chart review. Patients were rated as having had none, one, or two or more previous ECT series, and we noted how long it had been since the last ECT series. Information was based on patient reports at admission, typically corroborated by a relative. We could only verify these reports when the treatment had been previously administered at Carrier, which was the case for approximately 54% of ECT series reported. Three patients (two older and one younger) could not be specific about the actual number of series and were conservatively assigned to having had one prior series. All except two younger patients with a history of ECT gave the specific date of their last ECT series. These two patients were included in all the analyses except the one comparing younger and older patients in time elapsed since last ECT. Electrode placement and the number of treatments per series in prior admissions were largely unrecorded. Chart review was conducted without knowledge of the results of the Trail Making B test, and the reviewer was unaware of the purpose of this study.

RESULTS

Of the 20 older depressed patients, only five (25%) had not received any prior ECT. In contrast, 23 (56%) of the younger depressed patients had no prior ECT. Not surprisingly, older depressed patients were significantly more likely to have received previous ECT treatments ($\chi^2=5.24$, df=1, $p<.02$).

According to psychiatrist ratings, the older patients were not more depressed than the younger ones. On the Hamilton depression scale, older patients had a mean ($\pm$SD) of 19.60±7.28 compared with a mean of 20.22±7.24 for younger patients; on the BPRS, older patients scored an average of 36.10±9.23 while younger patients scored an average 37.17±9.00. Older patients rated themselves as less depressed than the younger patients did, with a mean on the Beck inventory of 22.92±14.17 for older patients versus 31.45±12.29 for younger ones ($t=2.28$, df=52, $p<.05$, two-tailed) and means on the SCL-90-R of 1.18±0.66 for older patients and 1.67±0.74 for younger ones ($t=2.28$, df=50, $p<.05$, two-tailed).

The most important findings assessing the effects of age and history of ECT were evaluated by two-way analysis of variance, with the number of seconds required to complete the Trail Making B test as the dependent variable. This analysis, illustrated in figure 1, yielded a significant interaction ($F=3.27$, df=2, 55, $p<.05$). That is, the length of time required to complete the test increased if the patient both was over the age of 65 and had a history of ECT. Specifically, cognitive functioning was more deficient in the depressed patients in the presence of two factors: age and history of ECT.

Two-tailed $t$ tests were conducted on the data measuring performance on the Trail Making B test for both older and younger patients who had received one prior series of ECT; older patients had a mean ($\pm$SD) score of 386.40±218.21 seconds versus 231.25±95.63 seconds for the younger patients ($t=1.9$, df=11, $p<.10$). For patients who had received at least two series of ECT, older patients had a mean score on the test of 535.50±326.99 seconds versus 202.10±109.39 seconds for younger patients ($t=4.4$, df=18, $p<.005$). In both cases the older patients required more time to complete the test.

Older patients with no history of ECT had a mean score of 218.40±64.46 seconds, which was significantly less time than that needed by older patients with a history of ECT, who had a mean score of 485.80±296.03 seconds ($t=3.08$, df=18, $p<.01$). The two groups did not differ significantly in mean age ($\pm$SD) of 69.80±4.09 years versus 69.67±3.79 years, respectively. In addition, older depressed patients with no history of ECT (mean score=218.40 seconds) were more likely to perform in the same way as the younger depressed patients with no prior ECT (mean score=166.61±98.49 seconds) as well as those younger depressed patients with prior ECT (mean=215.06±101.60 seconds). There were no significant
differences between younger patients with and without a history of ECT in the amount of time needed to complete the B test.

Because there were only five patients over age 65 with no history of ECT, the results, although statistically significant, may capitalize on chance factors. Therefore, we did a regression analysis assessing the relative effects of age and prior ECT on test performance (the dependent variable) for all 61 patients, so that age was treated as a continuous rather than a dichotomous variable. Severity of depression at the time of testing as measured by the Hamilton depression scale was covaried to equate severity of depression across all ages. There still was a significant interaction ($F=3.42, \text{df}=1, 57, p<.07$) between age and prior ECT, indicating that as the patients got older (not necessarily over 65 years) and had a history of ECT, deficits in cognitive functioning became more pronounced.

Although we expected an age differential in cerebral impairment only on the Trail Making B Test, we did a similar analysis on the other tests in the cognitive battery. For all ages, prior ECT did correlate with the short-term verbal memory tests but not with the tests of attention or long-term memory. Significant main effects for prior ECT were found on the paired associates test ($F=4.85, \text{df}=1, 59, p<.05$) and the short story test ($F=3.00, \text{df}=1, 55, p<.08$) from the Wechsler Memory Scale (16). There was no differential effect for the older individual.

Differences in cognitive functioning due to both age and history of ECT are important only if these deficiencies cannot be attributed to more severe depression in the older patient with a history of ECT. A two-way analysis of variance assessing severity of depression at admission (based on the psychiatrist's rating on the Hamilton depression scale) was performed on the sample who had completed the Trail Making B test according to age and amount of prior ECT a patient had received. As illustrated in figure 2, of the six possible groups no one group of patients was significantly more depressed than any other group. The older patients who previously had received ECT were not more depressed on admission than older patients who had no history of ECT or than any of the younger groups of patients.

We performed a regression analysis assessing performance on the Trail Making test by age and the number of months since the last ECT for those patients who reported prior ECT. The number of months since the last ECT did not account for the results over and above age; therefore there was no relationship between time since the last ECT and the performance on the test. Data were available for 16 of the 18 younger patients and for all 15 of the older patients with a history of ECT. Younger patients had a mean ($\pm SD$) of 65.06±92.26 months since last series of ECT and older patients had a mean of 52.20±77.41 months. Thus, both groups averaged at least 4 years.
COGNITIVE FUNCTIONING IN DEPRESSED GERIATRIC PATIENTS

since last ECT and there was no statistical difference between the two groups. Six (40%) of these 15 older patients and seven (44%) of these 16 younger patients had had ECT within a year of our study.

The mean (±SD) number of series of prior ECT reported in the patients' history was not significantly different for younger patients (2.28±1.49) than for older patients (2.87±2.42).

There was a nonsignificant trend reflecting a relationship between the number of prior series of ECT and more deficient performance on the test among the older patients (r=.37, df=13, n.s.) which was directly opposite to and significantly greater than (p<.05) that found in younger patients (r=-.22, df=16, n.s.).

DISCUSSION

In this study, elderly depressed patients with a history of ECT were found to be more likely than similar patients with no history of ECT or younger depressed patients regardless of their history for ECT to show some cognitive dysfunction. The difference cannot be laid to a more serious depression, a more extensive history of ECT, or more recent ECT treatments.

Although this retrospective small-group study must be interpreted cautiously, it suggests that the depressed older patient with a history of ECT may be functioning less efficiently than older depressed patients who have never received ECT. The results, if replicated, suggest longer lasting effects of ECT in elderly patients. There is, however, at least one other possible interpretation of this finding. We were unable to assess electrode placement in prior treatments accurately. Since bilateral ECT is more extensively used, it is possible that the cognitive effects noted in those patients with a history of ECT can be attributed to previous bilateral electrode placement. Although this hypothesis might be dismissed, since younger depressed patients with a similar history did not show this effect (and their chances of having had unilateral placement are probably only slightly greater), it is possible that bilateral ECT has potentially more side effects for the older patient. That possibility would support Fraser's (1) recommendation to use nondominant unilateral ECT to minimize the cognitive side effects of ECT in the elderly.

When one is evaluating treatment alternatives for the older patient, focusing on the patient's history of ECT along with other significant ECT treatment variables such as monitoring seizure length, deciding on unilateral versus bilateral electrode placement and so forth (17) is not unlike the strict monitoring of psychopharmacologic treatments due to such factors as differences in drug half-life and absorption rates for patients of all ages. Thus, the "proper dosage" of ECT may need to be more carefully evaluated for older patients just as special monitoring of psychotropic medications is required with these patients. A history of ECT, therefore, may be an important consideration in evaluating the level of functioning of a depressed older patient and a significant factor in shaping expectations for the course of recovery.

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The author reviews reports of neuropathology resulting from electroconvulsive therapy in experimental animals and humans. Although findings of petechial hemorrhage, gliosis, and neuronal loss were well established in the decade following the introduction of ECT, they have been generally ignored since then. ECT produces characteristic EEG changes and severe retrograde amnesia, as well as other more subtle effects on memory and learning. The author concludes that ECT results in brain disease and questions whether doctors should offer brain damage to their patients.

A 32-YEAR-OLD WOMAN who had received 21 ECT treatments stated 5 years later,

"One of the results of the whole thing is that I have no memory of what happened in the year to year and a half prior to my shock treatments. The doctor assured me that it was going to come back and it never has. I don't remember a bloody thing. I couldn't even find my way around the town I lived in for three years. If I walked into a building I didn't even know where I was. I could barely find my way around my own house. I could sew and knit before, but afterward I could no more comprehend a pattern to sew than the man in the moon." (1, p. 22)

By 1928, 10 years before the introduction of electroconvulsive therapy, it was known that accidental death by cardiac arrest could result from as little as 70 to 80 milliamperes in the human (2). It was also known in this early period that voltage applied to the head, as in legal electrocution, produced hemorrhage and rupture of cranial contents. Ugo Cerletti (3) demonstrated that electricity in the range of 100 volts and 200 milliamperes is rarely fatal when the current path is confined to the head, but does evoke a grand mal seizure marked by a stereotyped succession of events. A tonic muscular contraction, the "electric spasm," is followed after a latency of seconds by unconsciousness, a high voltage paroxysmal spike and sharp-wave discharge, and a clonic convulsion. Upon recovery of consciousness the subject is left with a transient acute brain syndrome, a high likelihood of permanent brain damage, and greater retrograde amnesia than is seen in any other form of head injury.

BRAIN DAMAGE IN EXPERIMENTAL ANIMALS

Before examining the premise that ECT damages human brains, a brief discussion of the lesions produced in animals by electrically induced convulsions is worthwhile. The many reports on this subject indicate that petechial hemorrhages scattered throughout both white and gray matter and concentrated in the path of the current are the most consistent finding. If animals are sacrificed after a delay of days or weeks following a convulsive series, hemosiderin pigment in phagocytes remains as evidence of vascular insult. Proliferation of glial cells, neuronal changes, and dropout are also commonly reported.

In 1938, the year of the first use of ECT on a human being, Lucio Bini, Cerletti's collaborator, reported "widespread and severe" brain damage in dogs with mouth to rectum electrode placement (4). At least seven subsequent animal studies employing conventional cranial electrodes supported his findings (5-11). These culminated in the exhaustive controlled experiment by Hans Hartelius in 1952 (12). This researcher found discernible vascular, glial, and neuronal changes in cats subjected to a maximum of 16 shocks. The animals were not paralyzed but were protected from physical injury during the seizure. Damage was slight but consistent, and the author concluded: "The question of whether or not irreversible damage to the nerve cells may occur in association with ECT must therefore be answered in the affirmative." Furthermore, by examination of unlabeled slides alone Hartelius was able to correctly recognize 8 of 8 slides from shocked animals as well as 8 of 8 controls. Although he considered many of the vascular and glial changes to be reversible, there was no mistaking the brain of a shocked animal for that of a control.

Since that time, ECT in humans has been modified through the use of oxygen and muscle paralysis to reduce the incidence of bone fractures. Although it is believed that these modifications also reduce brain...
damage, there are no animal studies to support this idea. On the contrary, recent work in England by Meldrum and associates (13, 14) on status epilepticus in primates suggests that the overexcited neuron by itself may be an important factor in seizure damage, especially in the hippocampus.

HUMAN BRAIN DAMAGE

Let us turn now to the neuropathological findings in humans who died during or shortly after ECT. As in lower animals, bleeding is the most frequent non-specific tissue response to injury and the one seen most often after electric shock. The first autopsy study in this country revealed brain damage identical to that seen in experimental animals. Alpers and Hughes (15) described the brains of 2 women who had received 62 and 6 shocks, respectively. The first woman's seizures had been suppressed by curare. Both brains showed hemorrhagic lesions around small blood vessels, rarefaction of tissue, and gliosis.

Throughout the 1940s similar reports continued to call attention to brain changes after ECT, including cases in which oxygen and curare had been administered (16). In 1948 Riese (17) added 2 more autopsy studies to the growing list and commented, "In all observations of sudden death after electric shock reported so far, petechial hemorrhages, cellular changes and some glial proliferation stand out prominently, as an almost constant whole."

Pathologists were especially interested in cases that discriminated between the direct effect of electricity and the mechanical and hypoxic effects secondary to convulsive motor activity. In 1953 Larsen reported on a 45-year-old man who had been given 4 electroshocks in the course of 5 days. The ECT did not induce any convulsions. The subject died from pneumonia 36 hours after the fourth electroshock. At autopsy fresh subarachnoid hemorrhage was found in the upper part of the left motor region—"at the site where an electrode had been applied" (18).

In 1957 Impastato summarized 254 electroshock fatalities. Brain damage was the leading cause of death in persons under 40 years of age, and nearly one-fifth of all cerebral deaths were hemorrhagic (19).

Some physicians were alarmed by the evidence of human brain damage. In 1959 Allen reported 18 cases in which he had found signs and symptoms of neurological sequelae following ECT. He concluded, "It is probable that some damage, which may be reversible but is often irreversible, is inseparable from this form of treatment," and called for "more serious consideration" of the entire procedure (20).

In 1963 McKegney and associates (21) reported the case of a 23-year-old man who became comatose 15 minutes after a single shock. The significance of this case was twofold: first, a complete physical and neurological examination was reportedly normal prior to ECT, and second, the ECT technique was contemporaneous and impeccable. The patient had received .6 mg of atropine, 16 mg of succinylcholine (Anectine), and forced oxygenation pre- and post-shock. ECT parameters were conventional, i.e., 130 volts for .3 seconds. Four days later a brain biopsy showed diffuse degeneration of neurons with hyperplasia of astrocytes. The young man never regained consciousness and at autopsy 2 months later evidence of old hemorrhage was found in the brain. This was the last detailed report in the English-language literature.

The damaging effects of ECT on the brain are thoroughly documented. All told, there have been 21 reports of neuropathology in humans (22-36). It is interesting that, despite the importance of a negative finding, there has not been a single detailed report of a normal human brain after shock.

ELECTROENCEPHALOGRAPHIC EFFECTS OF ECT

Like other insults to the brain, ECT produces EEG abnormalities. Diffuse slowing in the delta and theta range, increased voltage, and dysrhythmic activity are seen in all patients immediately following a series of bilateral ECT and, according to Blaurock and associates (37), may persist more than 6 months in 30% of the cases. Such slowing suggests damage to the thalamus.

Sutherland and associates (38) showed that the side of the brain shocked with unilateral ECT could be predicted by double-blind assessment of EEG tracings. The seizure thresholds of the hippocampus and other temporal lobe structures are the lowest in the brain; considerable interest has centered recently around "kindling," or seizure induction by subthreshold stimulation of these areas in animals (39). The induction of a permanent epileptic disorder following ECT in humans was first reported in 1942 and other reports followed (40).

MEMORY LOSS

ECT is a common cause of severe retrograde amnesia, i.e., destruction of memories of events prior to an injury. The potency of ECT as an amnestic exceeds that of severe closed head injury with coma. It is surpassed only by prolonged deficiency of thiamine pyrophosphate, bilateral temporal lobectomy, and the accelerated dementias, such as Alzheimer's.

After ECT it takes 5 to 10 minutes just to remember who you are, where you are, and what day it is. In the first weeks after a full course, retrograde and, to a lesser extent, anterograde amnesia are evident to the casual observer. But as time passes compensation occurs. As in other forms of brain injury, the subject is often oblivious to the residual deficit. Unless specific memories essential to daily living are discovered to be unavailable the victim may never know for sure the extent of memory loss. Unless sensitive tests for spon-
Cognitive Functioning and Degree of Psychosis in Schizophrenics given many Electroconvulsive Treatments

By DONALD I. TEMPLER, CAROL F. RUFF and GLORIA ARMSTRONG

PROBLEM

Goldman, Gomer, and Templer (2) found that the Bender-Gestalt and Benton Visual Retention Test performances of male chronic schizophrenic in-patients with a history of 50 or more electroconvulsive treatments (ECT) were significantly inferior to those of control patients matched for age, level of education, and race. However, the authors maintained that it cannot be inferred with certainty that ECT causes permanent brain damage since it is possible that schizophrenic patients more likely to receive ECT are those whose psychosis is more severe. It has been reported that patients with the so-called functional psychiatric disorders tend to do poorly on tests of organicity (5).

The purposes of the present research were (i) to replicate the findings of Goldman et al.; (ii) to compare ECT and control patients on the Wechsler Adult Intelligence Scale (WAIS); and (iii) to compare the degree of psychosis of ECT and control patients.

METHOD

Subjects were 14 male and 30 female schizophrenics in Western State Hospital, Hopkinsville, Kentucky. Of these patients 22 had a history of from 40 to 263 ECT with a median number of 58.5. All ECT was administered earlier than seven years ago. The 22 control patients were matched for age, sex, race, and level of education. Table I indicates the extent of the between-groups matching.

All 44 patients were administered the WAIS, the Bender-Gestalt, and the Benton (Form C, Administration A). Ten of the ECT patients and 12 of the control patients were able to complete the Minnesota Multiphasic Personality Inventory (MMPI). The Pascal and Suttell (3) method of scoring for deviations on the Bender-Gestalt was employed. Two scoring systems were used for the Benton: (i) the number of correct reproductions or 'number correct scores', and (ii) 'error scores' consisting of a detailed analysis of specific errors in each figure of each card (1). The interscorer reliability coefficients between the two scorers were ·99 (p < ·01) for the Bender-Gestalt error scores, ·97 (p < ·01) for the Benton error scores, and ·95 (p < ·01) for the Benton number correct scores.

The MMPI was administered so that the scores of ECT and control patients could be compared both on the Schizophrenia (Sc) Scale and on a special Sc-O Scale developed by Watson (4) to differentiate organics from schizophrenics. The unweighted long form of the Sc-O Scale was employed.

Additional procedures for comparing the degree of psychosis of ECT and control patients entailed the blind rating of two experienced clinical psychologists. These psychologists were requested to sort the 44 sets of answers on the Verbal section of the WAIS into the 22 most psychotic and the 22 least psychotic. The two
psychologists were given the following instructions:

'Place the 44 sets of WAIS answers into two categories, with those of the 22 most psychotic patients in one category and those of the 22 least psychotic patients in the other. Consider looseness of associations, peculiar ideation, idiosyncratic responses, and in general the abnormalities than can be subsumed under "schizophrenic thinking". Try to consider extent of thought disorder rather than number of correct answers or level of intelligence displayed. In like fashion, place the Bender-Gestalt reproductions into two categories of the 22 most psychotic and the 22 least psychotic.'

RESULTS

As indicated in Table I, the mean error score on the Bender-Gestalt was 124.27 for the ECT group and 56.82 for the control group (t = 3.20, p < .01). The mean Benton error score was 18.48 for the ECT group and 14.62 for the control group (t = 2.20, p < .05), and the mean Benton number correct score was 1.29 for the ECT group and 2.18 for the control group (t = 1.67, p < .05). On the WAIS, the ECT and control group respective means were 68.50 and 79.72 for Verbal IQ (t = 2.46, p < .01), 65.68 and 75.59 for Performance IQ (t = 2.02, p < .05), and 65.73 and 76.77 for Full Scale IQ (t = 2.32, p < .05).

For the ECT group, the product-moment correlation coefficient between number of ECT received and Bender-Gestalt error score was .07 (n.s.); between number of ECT and Benton error score, .34 (p < .10); between number of ECT and Benton number correct score, .37 (p < .05); between number of ECT and Verbal IQ, .10 (n.s.); between number of ECT and Performance IQ, .34 (p < .10); between number of ECT and Full Scale IQ, .26 (n.s.).

The mean MMPI Sc Scale score was 40.90 for the 10 ECT patients who completed the MMPI and 36.50 for the 18 control patients who completed the MMPI (t = .93, n.s.). In nine instances both the ECT patients and their control patients completed the MMPI. The mean Sc Scale score for these nine ECT patients was 41.78; the mean of the corresponding nine control patients was 55.89 (t = 1.07, n.s.). On the MMPI Sc-O Scale (upon which a higher score indicates a greater probability of organicity and a lesser one of schizophrenia), the 10 ECT patients obtained a mean score of 38.00 and the 18 control patients obtained a mean score of 42.11 (t = 1.51, n.s.). For the nine cases in which the ECT patients and their controls both completed the MMPI, the mean Sc Scale scores were 38.22 and 45.44 respectively (t = 2.19, p < .05).

One of the clinical psychologist raters classified 15 of the ECT patients' WAIS protocols and seven of the control patients' protocols into the '22 most psychotic' category (χ² = 5.08, p < .02). The other psychologist classified 16 ECT patients' protocols and 6 control protocols into the '22 most psychotic' category (χ² = 9.08, p < .01).

CONCLUSIONS AND DISCUSSION

The Goldman et al. findings of ECT patients' inferior Benton and Bender-Gestalt performances were replicated in the present study. The ECT patients' performance was also found to be inferior on the WAIS. However, the ECT patients were found to be more psychotic on all eight indices of psychoses—both of the MMPI Sc Scale score comparisons, both of the Sc-O Scale comparisons, both sets of clinical judgements upon the WAIS, and both sets of clinical judgements upon the Bender-Gestalt. The level of significance is beyond the .05 level in three of these comparisons. Furthermore, for the 10 ECT patients who completed the MMPI, the correlation coefficient between number of ECT received and Sc Scale score is .77 (p < .01).

However, the greater degree of psychosis of the ECT patients does not rule out organicity. It is conceivable that they could be both organically damaged and more psychotic. In order to equate both groups for degree of psychosis, the 10 ECT patients who completed the MMPI were matched for MMPI Sc Scale score and 10 ECT patients and 10 control patients were matched for MMPI Sc Scale score. The mean MMPI Sc Scale score of the 10 ECT patients matched for MMPI Sc Scale score was 49.00 and the mean MMPI Sc Scale score of the 10 control patients matched for MMPI Sc Scale score was 41.00 (t = 1.94, n.s.).
BY DONALD I. TEMPLER, CAROL F. RUFF AND GLORIA ARMSTRONG

score as closely as possible with 10 control patients. The mean absolute difference between these ECT and control patients on the Sc Scale is 1.9 points. The mean Sc Scale scores for the ECT and control patients were almost identical, 40.90 and 40.80 respectively. The respective mean difference for these MMPI matched ECT and control patients is 76.9 and 35.9 (t = 2.28, p < .05) for Bender-Gestalt error score; 15.9 and 14.0 (t = 1.01, n.s.) for Benton error score; 2.10 and 2.00 (t = 1.00, n.s.) for Benton number correct score; 77.1 and 82.1 (t = 1.14, n.s.) for Verbal IQ; 78.3 and 79.9 (t = 0.24, n.s.) for Performance IQ; and 76.1 and 80.1 (t = 0.78, n.s.) for Full Scale IQ.

It is apparent that the Benton and WAIS performances of ECT and control patients are very similar when degree of psychosis is controlled for. However, even with the two groups so matched for psychopathology, the ECT patients' Bender-Gestalt performance was significantly inferior to that of the control group. It is not certain why such significance was obtained upon a test of perceptual-motor functioning but not upon tests of memory and general intelligence. However, with the 22 ECT patients and their 22 control patients, the greatest level of significance was obtained with the Bender-Gestalt. Such a finding was also reported in the Goldman et al. study. The ECT patients' inferior Bender-Gestalt performance does suggest that ECT causes permanent brain damage.

ACKNOWLEDGEMENT

Appreciation is extended to Cyril and Violet Franks for their judgments of the psychoticism of WAIS answers and Bender-Gestalt reproductions.

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

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VA Hospital, Jefferson Barracks, Mo.

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. Therefore, there appear to be only two investigations that determined the cognitive effects of ECT after a number of months. 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 (7) 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

<table>
<thead>
<tr>
<th></th>
<th>ECT Group</th>
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<th>Control Group</th>
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<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
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<td>35.9</td>
<td>15.9</td>
</tr>
<tr>
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<td>8.1</td>
<td>14.3</td>
<td>6.9</td>
</tr>
<tr>
<td>Benton No. Correct</td>
<td>2.6</td>
<td>1.8</td>
<td>3.8</td>
<td>2.4</td>
</tr>
</tbody>
</table>

As indicated for the ECT group, Benton error score (t = 1.90, p < .05) and ECT group and 3 for the ECT and Bender-Gestalt. Benton error score and correct score −.4

The groups were not controlled adequately for the assessment of such impairment. Furthermore, the number of ECT have been far fewer than in the present research.

The significance of the Bender-Gestalt or course of treatment more, it seems plausible damaging effect of between number of ECT-produced str observation of pr treated.

Nevertheless, permanent brain damage to receive ECT at it has been report orders tend to do I lately positive the ECT psychopathol

1. BENTON, A. L. 2
4. NIELSON, J. M. A
5. PASCAL, G. R. and the individual patient's performance.
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. 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.

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\(^2(2)\). 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 (1). 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 (2) 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 non-mauve 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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Cognitive Functioning in Depressed Geriatric Patients With a History of ECT

Helen M. Pettinati, Ph.D., and Kathryn M. Bonner

Cognitive functioning in depressed geriatric patients, some with a history of ECT, was assessed with the Trail Making B test. Depressed patients over the age of 65 who had had at least one prior series of ECT performed more poorly on the test than did older patients with no history of ECT and younger depressed patients regardless of their ECT history. The groups did not differ in severity of depression. Careful assessment of elderly patients' history of ECT will allow for more informed decisions about the current use of ECT and an understanding of the cognitive status of these patients.

(Am J Psychiatry 141:49-52, 1984)

Electroconvulsive therapy (ECT) is currently a favored treatment for severe depression in the elderly (1, 2). The physiological changes that accompany the aging process can produce an increased sensitivity to pharmacological side effects and toxicity that precludes widespread use of antidepressants in this population (3, 4). Although no long-term adverse effects of ECT have been documented, transient retrograde and anterograde amnesia often occur, especially with bilateral electrode placement (5–7). Fraser (1) reported finding transient deficits in learning and long-term memory following bilateral ECT compared with younger depressed patients. He attributed the longer duration of postictal confusion to the potential cumulative effect of ECT in the elderly.

The temporary deficits in new learning (short-term memory) following recent bilateral ECT have been extensively reported (5). Attentional deficits (usually associated with depression) and short-term memory deficits (dysfunction usually associated with the ECT amnestic syndrome, anterograde) have been reported for both younger and older depressed patients (8). However, the prolongation in the elderly patient of the acute organic brain syndrome that typically is seen immediately after ECT administration raises the possibility that the depressed elderly patient may react differently to an accepted form of treatment. Thus, older patients often display more prolonged confusion and disorientation than do younger patients.

Potentially confounding the clinical observation that cognitive recovery is slow-paced for the depressed aged individual is the likelihood that these patients have already received ECT in previous hospital admissions. There are reports that an extended history of ECT (say, 50 or more treatments) may produce longer-lasting cognitive dysfunction (5, 9). A history of ECT typically aids the clinician in determining how useful ECT may be for treatment during the current admission. However, there is little empirical evidence about the impact of a history of ECT on subsequent recovery time from a current psychiatric illness or on the individual's ability to assimilate side effects of both ECT and medication.

We retrospectively examined the possible effects of past ECT administration in a sample of depressed elderly patients who were scheduled to receive ECT during their current admission. We compared their performance on the Trail Making B test (10), a neuropsychological measure of organic brain dysfunction, with that of younger depressed individuals who also were scheduled for ECT. We took a history of ECT for each patient so that performance related to age could be distinguished from performance related to a history of ECT.

METHOD

The sample for this study was drawn from inpatients at the Carrier Foundation with a diagnosis of major depressive disorder or schizoaffective disorder according to DSM-III criteria who were scheduled to receive ECT and who had agreed to participate in another research study involving drug effects on ECT (11). Twenty patients (13 women and seven men) were age 65 or over, with an average (±SD) age of 69.7±3.76 years. Forty-one patients (29 women and 12 men) were under age 65, with an average age of 41.7±12.43
years. No patient had received ECT for at least 3 months before the current hospital admission.

As part of an ongoing study comparing bilateral and nondominant unilateral ECT, we administered a test battery designed by one of us (H.M.P.) to assess cognitive functioning to each patient 3 to 6 hours before his or her first ECT treatment. The test battery included two measures of attention, four of short-term memory (two verbal and two nonverbal), and two of long-term memory (one verbal and one nonverbal). In addition, the Trail Making B test from the Halstead-Reitan Neuropsychological Battery (12) was included to assess confusion and more general cognitive functioning than is measured by the other tests in the battery. Since the Trail Making B test was designed to be sensitive to "organic brain dysfunction" and reflects impairment associated with several different brain locations, it is useful as a screening instrument for the longer-lasting cognitive deficits due to ECT that might be expected in the aged (10).

The Trail Making B test requires basic motor and spatial skills, the ability to count and to follow a complex plan, and cognitive flexibility. Each patient is instructed to connect numbers and letters in sequential order beginning with number 1, then proceeding to letter A, number 2, letter B, and so forth. The patient is interrupted and corrected when an error is made, then instructed to proceed from that point. Total completion time, including time for corrections, is recorded in seconds.

Cognitive functioning can also be affected by severity of depression (13), especially in elderly patients suffering from pseudodementia (14). In addition to making a DSM-III diagnosis, a psychiatrist completed the Hamilton Rating Scale for Depression and the Brief Psychiatric Rating Scale (BPRS). The patients were asked to complete the Beck Depression Inventory and the SCL-90-R (15).

Following data collection, we assessed the patients' history of ECT through chart review. Patients were rated as having had none, one, or two or more previous ECT series, and we noted how long it had been since the last ECT series. Information was based on patient reports at admission, typically corroborated by a relative. We could only verify these reports when the treatment had been previously administered at Carrier, which was the case for approximately 54% of ECT series reported. Three patients (two older and one younger) could not be specific about the actual number of series and were conservatively assigned to having had one prior series. All except two younger patients with a history of ECT gave the specific date of their last ECT series. These two patients were included in all the analyses except the one comparing younger and older patients in time elapsed since last ECT. Electrode placement and the number of treatments per series in prior admissions were largely unrecorded. Chart review was conducted without knowledge of the results of the Trail Making B test, and the reviewer was unaware of the purpose of this study.

RESULTS

Of the 20 older depressed patients, only five (25%) had not received any prior ECT. In contrast, 23 (56%) of the younger depressed patients had no prior ECT. Not surprisingly, older depressed patients were significantly more likely to have received previous ECT treatments ($\chi^2=5.24, \text{df}=1, p<.02$).

According to psychiatrist ratings, the older patients were not more depressed than the younger ones. On the Hamilton depression scale, older patients had a mean ($\pm$SD) of 19.60±7.28 compared with a mean of 20.22±7.24 for younger patients; on the BPRS, older patients scored an average of 36.10±9.23 while younger patients scored an average 37.17±9.00. Older patients rated themselves as less depressed than the younger patients did, with a mean on the Beck inventory of 22.92±14.17 for older patients versus 31.45±12.29 for younger ones (t=2.28, df=52, p<.05, two-tailed) and means on the SCL-90-R of 1.18±0.66 for older patients and 1.67±0.74 for younger ones (t=2.28, df=50, p<.05, two-tailed).

The most important findings assessing the effects of age and history of ECT were evaluated by two-way analysis of variance, with the number of seconds required to complete the Trail Making B test as the dependent variable. This analysis, illustrated in figure 1, yielded a significant interaction ($F$=3.27, df=2, 55, p<.05). That is, the length of time required to complete the test increased if the patient both was over the age of 65 and had a history of ECT. Specifically, cognitive functioning was more deficient in the depressed patients in the presence of two factors: age and history of ECT.

Two-tailed t tests were conducted on the data measuring performance on the Trail Making B test for both older and younger patients who had received one prior series of ECT; older patients had a mean ($\pm$SD) score of 386.40±218.21 seconds versus 231.25±95.63 seconds for the younger patients (t=1.9, df=11, p<.10). For patients who had received at least two series of ECT, older patients had a mean score on the test of 535.50±326.99 seconds versus 202.10±109.39 seconds for younger patients (t=4.4, df=18, p<.005). In both cases the older patients required more time to complete the test.

Older patients with no history of ECT had a mean score of 218.40±64.46 seconds, which was significantly less time than that needed by older patients with a history of ECT, who had a mean score of 485.80±296.03 seconds (t=3.08, df=18, p<.01). The two groups did not differ significantly in mean age ($\pm$SD) 69.80±4.09 years versus 69.67±3.79 years, respectively. In addition, older depressed patients with no history of ECT (mean score=218.40 seconds) were more likely to perform in the same way as the younger depressed patients with no prior ECT (mean score=166.61±98.49 seconds) as well as those younger depressed patients with prior ECT (mean=215.06±101.60 seconds). There were no significant
Differences between younger patients with and without a history of ECT in the amount of time needed to complete the B test. Because there were only five patients over age 65 with no history of ECT, the results, although statistically significant, may capitalize on chance factors. Therefore, we did a regression analysis assessing the relative effects of age and prior ECT on test performance (the dependent variable) for all 61 patients, so that age was treated as a continuous rather than a dichotomous variable. Severity of depression at the time of testing as measured by the Hamilton depression scale was covaried to equate severity of depression across all ages. There still was a significant interaction $F=3.42$, $df=1$, 57, $p<.07$ between age and prior ECT, indicating that as the patients got older (not necessarily over 65 years) and had a history of ECT, deficits in cognitive functioning became more pronounced.

Although we expected an age differential in cerebral impairment only on the Trail Making B Test, we did a similar analysis on the other tests in the cognitive battery. For all ages, prior ECT did correlate with the short-term verbal memory tests but not with the tests of attention or long-term memory. Significant main effects for prior ECT were found on the paired associates test ($F=4.85$, $df=1$, 59, $p<.05$) and the short story test ($F=3.00$, $df=1$, 55, $p<.08$) from the Wechsler Memory Scale (16). There was no differential effect for the older individual.

Differences in cognitive functioning due to both age and history of ECT are important only if these deficiencies cannot be attributed to more severe depression in the older patient with a history of ECT. A two-way analysis of variance assessing severity of depression at admission (based on the psychiatrist's rating on the Hamilton depression scale) was performed on the sample who had completed the Trail Making B test according to age and amount of prior ECT a patient had received. As illustrated in figure 2, of the six possible groups no one group of patients was significantly more depressed than any other group. The older patients who previously had received ECT were not more depressed on admission than older patients who had no history of ECT or than any of the younger groups of patients.

We performed a regression analysis assessing performance on the Trail Making test by age and the number of months since the last ECT for those patients who reported prior ECT. The number of months since the last ECT series did not account for the results over and above age; therefore there was no relationship between time since the last ECT and the performance on the test. Data were available for 16 of the 18 younger patients and for all 15 of the older patients with a history of ECT. Younger patients had a mean ($\pm$SD) of $65.06\pm92.26$ months since last series of ECT and older patients had a mean of $52.20\pm77.41$ months. Thus, both groups averaged at least 4 years
since last ECT and there was no statistical difference between the two groups. Six (40%) of these 15 older patients and seven (44%) of these 16 younger patients had had ECT within a year of our study.

The mean (±SD) number of series of prior ECT reported in the patients’ history was not significantly different for younger patients (2.28±1.49) than for older patients (2.87±2.42).

There was a nonsignificant trend reflecting a relationship between the number of prior series of ECT and more deficient performance on the test among the older patients (r=.37, df=13, n.s.) which was directly opposite to and significantly greater than (p<.03) that found in younger patients (r=-.22, df=16, n.s.).

DISCUSSION

In this study, elderly depressed patients with a history of ECT were found to be more likely than similar patients with no history of ECT or younger depressed patients regardless of their history for ECT to show some cognitive dysfunction. The difference cannot be laid to a more serious depression, a more extensive history of ECT, or more recent ECT treatments.

Although this retrospective small-group study must be interpreted cautiously, it suggests that the depressed older patient with a history of ECT may be functioning less efficiently than older depressed patients who have never received ECT. The results, if replicated, suggest longer lasting effects of ECT in elderly patients. There is, however, at least one other possible interpretation of this finding. We were unable to assess electrode placement in prior treatments accurately. Since bilateral ECT is more extensively used, it is possible that the cognitive effects noted in those patients with a history of ECT can be attributed to previous bilateral electrode placement. Although this hypothesis might be dismissed, since younger depressed patients with a similar history did not show this effect (and their chances of having had unilateral placement are probably only slightly greater), it is possible that bilateral ECT has potentially more side effects for the older patient. That possibility would support Fraser’s (1) recommendation to use nondominant unilateral ECT to minimize the cognitive side effects of ECT in the elderly.

When one is evaluating treatment alternatives for the older patient, focusing on the patient’s history of ECT along with other significant ECT treatment variables such as monitoring seizure length, deciding on unilateral versus bilateral electrode placement and so forth (17) is not unlike the strict monitoring of psychopharmacologic treatments due to such factors as differences in drug half-life and absorption rates for patients of all ages. Thus, the “proper dosage” of ECT may need to be more carefully evaluated for older patients just as special monitoring of psychotropic medications is required with these patients. A history of ECT, therefore, may be an important consideration in evaluating the level of functioning of a depressed older patient and a significant factor in shaping expectations for the course of recovery.

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Shock Treatment, Brain Damage, and Memory Loss: A Neurological Perspective

BY JOHN FRIEDBERG, M.D.

The author reviews reports of neuropathology resulting from electroconvulsive therapy in experimental animals and humans. Although findings of petechial hemorrhage, gliosis, and neuronal loss were well established in the decade following the introduction of ECT, they have been generally ignored since then. ECT produces characteristic EEG changes and severe retrograde amnesia, as well as other more subtle effects on memory and learning. The author concludes that ECT results in brain disease and questions whether doctors should offer brain damage to their patients.

A 32-YEAR-OLD WOMAN who had received 21 ECT treatments stated 5 years later,

One of the results of the whole thing is that I have no memory of what happened in the year to year and a half prior to my shock treatments. The doctor assured me that it was going to come back and it never has. I don't remember a bloody thing. I couldn't even find my way around the town I lived in for three years. If I walked into a building I didn't even know where I was. I could barely find my way around my own house. I could sew and knit before, but afterward I could no more comprehend a pattern to sew than the man in the moon. (1, p. 22)

By 1928, 10 years before the introduction of electroconvulsive therapy, it was known that accidental death by cardiac arrest could result from as little as 70 to 80 milliamperes in the human. It was also known in this early period that voltage applied to the head, as in legal electrocution, produced hemorrhage and rupture of cranial contents. Ugo Cerletti demonstrated that electricity in the range of 100 volts and 200 milliamperes is rarely fatal when the current path is confined to the head, but does evoke a grand mal seizure marked by a stereotyped succession of events. A tonic muscular contraction, the "electric spasm," is followed after a latency of seconds by unconsciousness, a high voltage paroxysmal spike and sharp-wave discharge, and a clonic convulsion. Upon recovery of consciousness the subject is left with a transient acute brain syndrome, a high likelihood of permanent brain damage, and greater retrograde amnesia than is seen in any other form of head injury.

BRAIN DAMAGE IN EXPERIMENTAL ANIMALS

Before examining the premise that ECT damages human brains, a brief discussion of the lesions produced in animals by electrically induced convulsions is worthwhile. The many reports on this subject indicate that petechial hemorrhages scattered throughout both white and gray matter and concentrated in the path of the current are the most consistent finding. If animals are sacrificed after a delay of days or weeks following a convulsive series, hemosiderin pigment in phagocytes remains as evidence of vascular insult. Proliferation of glial cells, neuronal changes, and dropout are also commonly reported.

In 1938, the year of the first use of ECT on a human being, Lucio Bini, Cerletti's collaborator, reported "widespread and severe" brain damage in dogs with mouth to rectum electrode placement (4). At least seven subsequent animal studies employing conventional cranial electrodes supported his findings (5–11). These culminated in the exhaustive controlled experiment by Hans Hartelius in 1952 (12). This researcher found discernible vascular, glial, and neuronal changes in cats subjected to a maximum of 16 shocks. The animals were not paralyzed but were protected from physical injury during the seizure. Damage was slight but consistent, and the author concluded: "The question of whether or not irreversible damage to the nerve cells may occur in association with ECT must therefore be answered in the affirmative." Furthermore, by examination of unlabeled slides alone Hartelius was able to correctly recognize 8 of 8 slides from shocked animals as well as 8 of 8 controls. Although he considered many of the vascular and glial changes to be reversible, there was no mistaking the brain of a shocked animal for that of a control.

Since that time, ECT in humans has been modified through the use of oxygen and muscle paralysis to reduce the incidence of bone fractures. Although it is believed that these modifications also reduce brus...
damage, there are no animal studies to support this idea. On the contrary, recent work in England by Mel- 
drum and associates (13, 14) on status epilepticus in 
primates suggests that the overexcited neuron by itself 
may be an important factor in seizure damage, espe-
cially in the hippocampus.

HUMAN BRAIN DAMAGE

Let us turn now to the neuropathological findings in 
humans who died during or shortly after ECT. As in 
lower animals, bleeding is the most frequent non-
specific tissue response to injury and the one seen 
most often after electric shock. The first autopsy study 
in this country revealed brain damage identical to that 
seen in experimental animals. Alpers and Hughes (15) 
described the brains of 2 women who had received 62 
and 6 shocks, respectively. The first woman's seizures 
were induced by curare. Both brains showed 
hemorrhagic lesions around small blood vessels, rare-
faction of tissue, and gliosis.

Throughout the 1940s similar reports continued to 
call attention to brain changes after ECT, including 
cases in which oxygen and curare had been adminis-
tered (16). In 1948 Riese (17) added 2 more autopsy 
studies to the growing list and commented, "In all ob-
servations of sudden death after electric shock report-
ed so far, petechial hemorrhages, cellular changes and 
some glial proliferation stand out prominently, as an 
almost constant whole."

Pathologists were especially interested in cases that 
discriminated between the direct effect of electricity 
and the mechanical and hypoxic effects secondary to 
convulsive motor activity. In 1953 Larsen reported on 
a 45-year-old man who had been given 4 electroshocks 
in the course of 5 days. The ECT did not induce any 
convulsions. The subject died from pneumonia 36 
hours after the fourth electroshock. At autopsy fresh 
subarachnoid hemorrhage was found in the upper part 
of the left motor region—"at the site where an elec-
trode had been applied" (18).

In 1957 Impastato summarized 254 electroshock fa-
talities. Brain damage was the leading cause of death 
in persons under 40 years of age, and nearly one-fifth 
of all cerebral deaths were hemorrhagic (19).

Some physicians were alarmed by the evidence of 
human brain damage. In 1959 Allen reported 18 cases 
in which he had found signs and symptoms of neuro-
logical sequelae following ECT. He concluded, "It is 
probable that some damage, which may be reversible 
but is often irreversible, is inseparable from this form 
of treatment," and called for "more serious consid-
eration" of the entire procedure (20).

In 1963 McKechnie and associates (21) reported the 
case of a 23-year-old man who became comatose 15 
minutes after a single shock. The significance of this 
case was twofold: first, a complete physical and neuro-
logical examination was reportedly normal prior to 
ECT, and second, the ECT technique was contempo-
rary and impeccable. The patient had received .6 mg of 
atropine, 16 mg of succinylcholine (Anectine), and 
forced oxygenation pre- and post-shock. ECT param-
ters were conventional, i.e., 150 volts for .3 seconds. 
Four days later a brain biopsy showed diffuse degener-
ation of neurons with hyperplasia of astrocytes. The 
young man never regained consciousness and at au-
topsy 2 months later evidence of old hemorrhage was 
found in the brain. This was the last detailed report in 
the English-language literature.

The damaging effects of ECT on the brain are thor-
oughly documented. All told, there have been 21 re-
ports of neuropathology in humans (22-36). It is inter-
esting that, despite the importance of a negative find-
ing, there has not been a single detailed report of a 
normal human brain after shock.

ELECTROENCEPHALOGRAPHIC EFFECTS OF ECT

Like other insults to the brain, ECT produces EEG 
abnormalities. Diffuse slowing in the delta and theta 
range, increased voltage, and dysrhythmic activity are 
seen in all patients immediately following a series of 
bilateral ECT and, according to Blaurock and associ-
ates (37), may persist more than 6 months in 30% of 
the cases. Such slowing suggests damage to the thala-
mus.

Sutherland and associates (38) showed that the side 
of the brain shocked with unilateral ECT could be pre-
dicted by double-blind assessment of EEG tracings.

The seizure thresholds of the hippocampus and oth-
er temporal lobe structures are the lowest in the brain; 
considerable interest has centered recently around 
"kindling," or seizure induction by subthreshold stim-
ulation of these areas in animals (39). The induction of 
a permanent epileptic disorder following ECT in hu-
mans was first reported in 1942 and other reports fol-
lowed (40).

MEMORY LOSS

ECT is a common cause of severe retrograde am-
nesia, i.e., destruction of memories of events prior to 
an injury. The potency of ECT as an amnestic exceeds 
that of severe closed head injury with coma. It is sur-
passed only by prolonged deficiency of thiamine pyro-
phosphate, bilateral temporal lobectomy, and the ac-
celerated dementias, such as Alzheimer's.

After ECT it takes 5 to 10 minutes just to remember 
who you are, where you are, and what day it is. In the 
first weeks after a full course, retrograde and, to a less-
er extent, anterograde amnesia are evident to the cas-
ual observer. But as time passes compensation occurs. 
As in other forms of brain injury, the subject is often 
oblivious to the residual deficit. Unless specific memo-
ries essential to daily living are discovered to be un-
available the victim may never know for sure the ex-
tent of memory loss. Unless sensitive tests for spon-

A NEUROLOGICAL PERSPECTIVE

Although some studies have purported to show improvement of learning ability after ECT, not one used sham ECT as a control and few used any controls at all. In regard to more general intellectual ability, a study in 1973 (54) showed that the performance on the Bender Gestalt perceptual motor test of 20 institutionalized subjects who had received 50 or more ECT treatments 10 to 15 years before testing was significantly impaired compared to the performance of 20 carefully matched control subjects who had not received ECT. The authors inferred that ECT had caused permanent brain damage.

MECHANISM OF ACTION OF ECT

The mechanism of action of ECT can now be summarized on the basis of evidence accumulated since its introduction. Penfield and Perot showed in the 1950s that memory traces may be evoked by direct electrical stimulation of the temporal lobe cortex, and nowhere else (55). Scoville and Milner (56) discovered that bilateral hippocampal resection utterly abolished the ability to remember any new material, resulting in a catastrophic inability to learn. From numerous studies of the neuropathology of the amnestic-confabulatory syndrome of Korsakoff it is known that the mammillary bodies, the dorsal median nuclei of the thalamus, and the gray matter surrounding the third ventricle and aqueduct are essential to the general memory process. All of these critical brain structures are just beneath the thin squamous plate of the temporal bone, within seven centimeters of the electrodes, in the direct path and highest density of the current during ECT.

CONCLUSIONS

From a neurological point of view ECT is a method of producing amnesia by selectively damaging the temporal lobes and the structures within them. When it was first introduced it was only one of several methods of producing brain damage employed in psychiatry, including insulin coma (1927), camphor and pentylenetetrazol (Metrazol) injections (1933), and prefrontal lobotomy (1935). It is the only such method from that era still used on a large scale. It is highly unlikely that ECT, if critically examined, would be found acceptable by today’s standards of safety.

From a neurological point of view ECT produces a form of brain disease, with an estimated incidence of new cases in the range of 100,000 per year (57). Many psychiatrists are unaware that ECT causes brain damage and memory loss because numerous authorities and a leading psychiatric textbook (58) deny these...
facts. Others, who know of its effects, argue that the interruption of unpleasant states of mind is worth the damage. Some are beginning to give the client a truly informed choice, although most state laws still allow ECT to be imposed if the doctor feels that "good cause" exists.

Assuming free and fully informed consent, it is well to reaffirm the individual's right to pursue happiness through brain damage if he or she so chooses. But we might ask ourselves whether we, as doctors sworn to the Hippocratic Oath, should be offering it.

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DISCUSSION


Current Perspectives on ECT: A Discussion

BY FRED H. FRANKEL, M.B.CH.B., D.P.M.

MY COMMENTS about each of the preceding papers on ECT reflect my personal views and not those of the former Massachusetts Task Force on ECT (1) or the American Psychiatric Association's Task Force on ECT. Space limitations preclude a complete discussion of each of the papers, but I will attempt to address at least some of their important or controversial points.

ECT: A NEUROLOGICAL PERSPECTIVE

In my view, Dr. Friedberg has weakened his position by the manner in which he has gathered the evidence to support it. The questions he raises are relevant; they have been asked by many others. However, he has attempted to answer them with data that have been carelessly culled from the literature and frequently reported inaccurately.

Dr. Friedberg's evidence against ECT is arranged in four main sections. He reports on the neuropathological findings in experimental animals subjected to electrically induced convulsions, and in humans who have died during a course of ECT or within weeks or months afterward. He then reports studies and subjective accounts of memory loss and, finally, EEG changes. He concludes that "from a neurological point of view ECT is a method of producing amnesia by selectively damaging the temporal lobes and the structures within them." He states that "ECT produces a form of brain disease" of epidemic proportions and asks why doctors, who are sworn to the Hippocratic Oath, are offering it.

Dr. Friedberg has listed 58 references in his article. I have examined 25 of these and selected those which seemed especially relevant to his argument. In presenting his evidence for brain damage in animals exposed to electrical currents, Dr. Friedberg states: "In 1938, the year of the first use of ECT on a human being, Lucio Bini, Cerletti's collaborator, reported 'widespread and severe' brain damage in dogs. At least seven subsequent animal studies confirmed his findings." The following quotation is from the 5th reference:

We have so far employed exclusively the method of Vi"ale, which consists of passing the street current (120 volt) for a very short time (1/15 to 1/20 second) through the entire body of the animal with one of two electrodes (carbons from a voltaic arc) in the mouth and the other in the rectum.

With this method we succeeded in producing constantly typical epileptic attacks in dogs. During the passage of the current the animal howls and has a violent tonic spasm with opisthotonus which lasts several seconds after the application of current to the brain.

With this method we succeeded in producing constantly typical epileptic attacks in dogs. During the passage of the current the animal howls and has a violent tonic spasm with opisthotonus which lasts several seconds after the application of current to the brain.