ECT: Brain Damage

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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-

TABLE I

Extent of between-group matching and mean Bender-Gestalt, Benton, and WAIS scores for ECT and control groups

	ECT group		Control group	
	Mean	S.D.	Mean	S.D.
Age	43·86	10·99	42·23	8·61
Years of education	9·86	3·47	9·82	3·08
Bender error score	124·17	87·32	56·82	46·17
Benton error score	18·48	5·28	14·82	5·60
Benton no. correct	1·29	1·76	2·18	2·08
WAIS verbal IQ ..	68·50	16·86	79·72	14·67
WAIS performance IQ	65·68	17·67	75·59	14·64
WAIS full scale IQ	65·73	16·87	76·77	14·65

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 $124 \cdot 27$ for the ECT group and $56 \cdot 82$ for the control group ($t = 3 \cdot 20$, $p < \cdot 01$). The mean Benton error score was $18 \cdot 48$ for the ECT group and $14 \cdot 82$ for the control group ($t = 2 \cdot 20$, $p < \cdot 05$), and the mean Benton number correct score was $1 \cdot 29$ for the ECT group and $2 \cdot 18$ for the control group ($t = 1 \cdot 67$, $p < \cdot 05$). On the WAIS, the ECT and control group respective means were $68 \cdot 50$ and $79 \cdot 72$ for Verbal IQ ($t = 2 \cdot 46$, $p < \cdot 01$), $65 \cdot 68$ and $75 \cdot 59$ for Performance IQ ($t = 2 \cdot 02$, $p < \cdot 05$), and $65 \cdot 73$ and $76 \cdot 77$ for Full Scale IQ ($t = 2 \cdot 32$, $p < \cdot 05$).

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

The mean MMPI Sc Scale score was $40 \cdot 90$ for the 10 ECT patients who completed the MMPI and $36 \cdot 50$ for the 18 control patients who completed the MMPI ($t = \cdot 93$, 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 $41 \cdot 78$; the mean of the corresponding nine control patients was $35 \cdot 89$ ($t = 1 \cdot 07$, 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 $38 \cdot 00$ and the 18 control patients obtained a mean score of $42 \cdot 11$ ($t = 1 \cdot 51$, 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 $38 \cdot 22$ and $45 \cdot 44$ respectively ($t = 2 \cdot 19$, $p < \cdot 05$).

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 ($\chi^2 = 5 \cdot 08$, $p < \cdot 02$). The other clinical psychologist classified 16 ECT patients' protocols and 6 control protocols into the '22 most psychotic' category ($\chi^2 = 9 \cdot 08$, $p < \cdot 01$).

One of the clinical psychologists classified 14 ECT patients' and 8 of the control patients' Bender-Gestalt reproductions into the '22 most psychotic' category ($\chi^2 = 3 \cdot 27$, $p < \cdot 10$). The other psychologist classified 15 ECT patients' Bender-Gestalt reproductions and 7 control patients' reproductions into the '22 most psychotic' category ($\chi^2 = 5 \cdot 80$, $p < \cdot 02$).

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 $\cdot 05$ 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 $\cdot 77$ ($p < \cdot 01$).

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

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score as closely as possible with 10 control patients. The mean absolute difference between these ECT and control patients on the Sc Scale is $1\cdot9$ points. The mean Sc Scale scores for the ECT and control patients were almost identical, $40\cdot90$ and $40\cdot80$ respectively. The respective mean difference for these MMPI matched ECT and control patients is $76\cdot9$ and $35\cdot9$ ($t = 2\cdot28$, $p < \cdot05$) for Bender-Gestalt error score; $15\cdot9$ and $14\cdot0$ ($t = 1\cdot01$, n.s.) for Benton error score; $2\cdot10$ and $2\cdot00$ ($t = 1\cdot00$, n.s.) for Benton number correct score; $77\cdot1$ and $82\cdot1$ ($t = \cdot14$, n.s.) for Verbal IQ; $78\cdot3$ and $79\cdot8$ ($t = \cdot24$, n.s.) for Performance IQ; and $76\cdot1$ and $80\cdot1$ ($t = \cdot78$, 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.

REFERENCES

1. BENTON, A. L. (1963). *The Benton Visual Retention Test: Clinical and Experimental Applications*. New York: Psychological Corporation.
2. GOLDMAN, H., GOMER, F. E., and TEMPLER, D. I. (1972). 'Long-term effects of electroconvulsive therapy upon memory and perceptual-motor performance.' *J. clin. Psychol.*, 28, 32–4.
3. PASCAL, G. R., and SUTTELL, B. J. (1951). *The Bender-Gestalt Test*. New York: Paul B. Hoeber.
4. WATSON, C. G. (1971). 'An MMPI scale to separate brain-damaged from schizophrenic men.' *J. consult. clin. Psychol.*, 36, 121–5.
5. YATES, A. J. (1954). 'The validity of some psychological tests of brain damage.' *Psychol. Bull.*, 51, 359–79.

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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 [2, 3, 6, 7, 8, 10, 11, 13]. There appear to be only two investigations that determined the cognitive effects of ECT after a number of months [6, 9]. However, in both of these studies neither control patients nor an adequate number of ECT patients were employed. In the report of Pascal and Zeaman [6], a patient's Wechsler-Bellevue and Rorschach scores before 10 ECT and 7 months afterward were comparable. Stone [9] reported that a patient's Henmon-Nelson Test of Mental Ability score 60 days after the last of 20 ECT was comparable to her score of 7 years earlier.

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

METHOD

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

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

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

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5. PASCAL, G. R. and
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The Bender-Gestalt and Benton were selected because they are well established tests that reflect brain pathology and because they have quantitative scoring systems. The Pascal and Suttell [5] method of scoring for deviations on the Bender-Gestalt designs was employed. Two scoring systems were used for the Benton: (1) the number of correct reproductions or "number correct scores", and (2) "error scores" that consisted of a detailed analysis of specific errors in each figure of each card [1]. The interscorer reliability coefficients between two scorers were .90 ($p < .005$) for the Bender-Gestalt error scores, .97 ($p < .005$) for the Benton error scores, and .94 ($p < .005$) for the Benton number correct scores.

RESULTS

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

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

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

CONCLUSIONS

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

Nevertheless, it cannot be inferred with complete certainty that ECT causes permanent brain pathology. It is possible that schizophrenic patients more likely to receive ECT are those whose psychotic symptomatology is more severe. And, it has been reported that patients with the so-called functional psychiatric disorders tend to do poorly on tests of organicity [12]. Therefore, one cannot be absolutely positive that the ECT and control groups were equated for degree of pre-ECT psychopathology.

REFERENCES

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

7. STONE, C. P. Characteristic losses and gains on the Wechsler Memory Scales as applied on psychotic patients before, during, and after a series of electroconvulsive shocks. *Amer. Psychologist*, 1946, *1*, 245.

8. STONE, C. P. Losses and gains on the Alpha group examination as related to electroconvulsive shocks. *J. comp. physiol. Psychol.*, 1947, *40*, 183-189.

9. STONE, C. P. Pre-illness test records compared with performances during and after electroconvulsive shocks. *J. abn. soc. Psychol.*, 1950, *45*, 154-159.

10. SUMMERSKILL, J., SEEMAN, W. and MEALES, O. W. An evaluation of post electroshock confusion with the Reiter apparatus. *Amer. J. Psychiat.*, 1952, *100*, 835-838.

11. WORCHEL, P. and NARCISO, J. C. Electroshock convulsions and memory: The interval between learning and shock. *J. abn. soc. Psychol.*, 1950, *45*, 85-98.

12. YATES, A. J. The validity of some psychological tests of brain damage. *Psychol. Bull.*, 1954, *51*, 359-379.

13. ZAMORA, E. N. and KAELBLING, R. Memory and electro-convulsive therapy. *Amer. J. Psychiat.*, 1965, *122*, 546-554.

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

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PROBLEM

Malvaria is a psychiatric disease proposed by Hoffer and Osmond[7], the criterion for which is simply a mauve chromatograph stain extracted from the urine. Mauve producers were either schizophrenic or displayed features of this diagnosis. Other studies[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[3], but another investigator[4] reported that kryptopyrrol produced the mauve. This substance is unlikely to result from tranquillizers. The Hoffer-Osmond[5] Diagnostic test (HOD), a self-rating set of true-false statements, differentiated between mauve and nonmauve producing patients in the same way that it differentiated between schizophrenics and neurotics[6].

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

METHOD

From the psychiatric ward of a teaching general hospital, 82 patients were obtained, all of whom were examined during the first few days after admission. Only 14 were on any 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.

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 longer 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). 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±12.45

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).
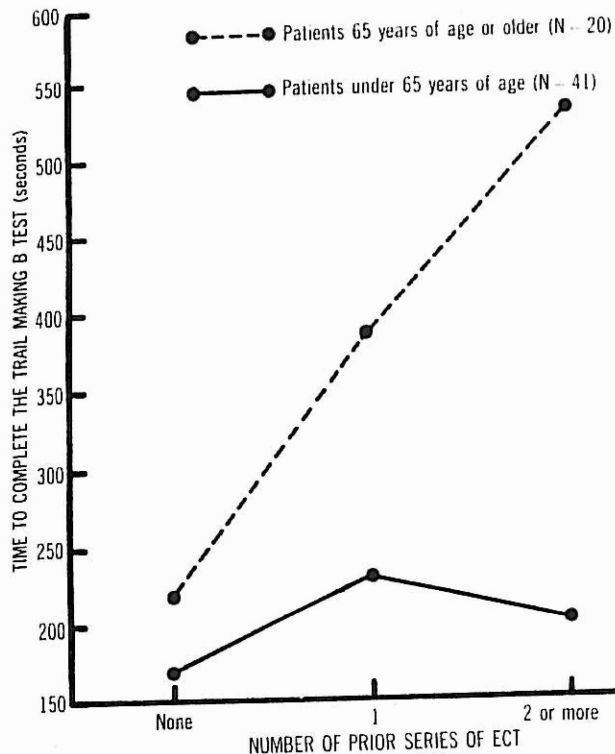
According to psychiatrist ratings, the older patients were not more depressed than the younger ones. On the Hamilton depression scale, older patients had a mean ($\pm$SD) of 19.60 ± 7.28 compared with a mean of 20.22 ± 7.24 for younger patients; on the BPRS, older patients scored an average of 36.10 ± 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) (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$\pm$98.49 seconds) as well as those younger depressed patients with prior ECT (mean=215.06$\pm$101.60 seconds). There were no significant

FIGURE 1. Performance on Trail Making B Test by Age and Amount of Prior ECT

FIGURE 2. Severity of Depression Found on the Hamilton Rating Scale at Time of Admission by Age and Amount of Prior ECT
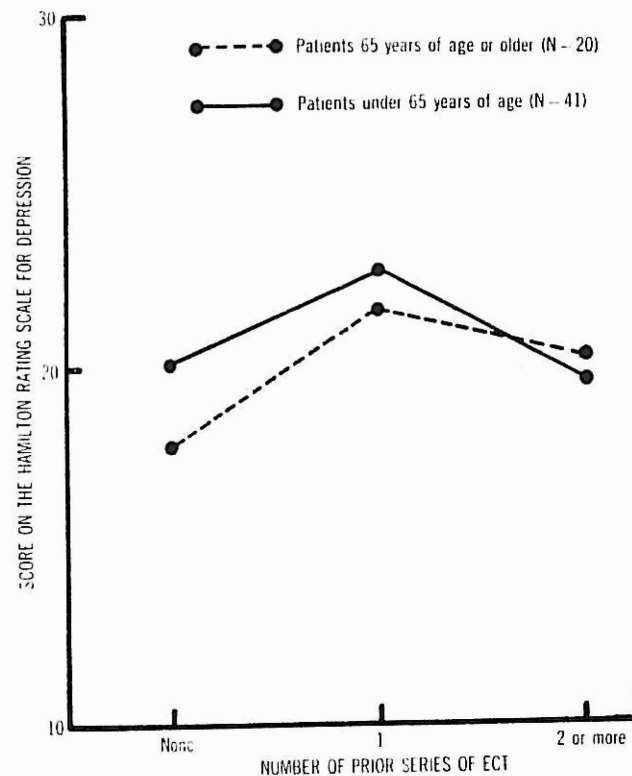
differences between younger patients with and without a history of ECT in the amount of time needed to complete the B test.

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 ± 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.

REFERENCES

1. Fraser RM: ECT and the elderly, in Electroconvulsive Therapy: An Appraisal. Edited by Palmer R. Oxford, Oxford University Press, 1981
2. Weiner RD: The role of electroconvulsive therapy in the treatment of depression in the elderly. J Am Geriatr Soc 30:710–712, 1982
3. Albert M: Geriatric neuropsychology. J Consult Clin Psychol 49:835–850, 1981
4. Salzman C: A primer on geriatric psychopharmacology. Am J Psychiatry 139:67–78, 1982
5. American Psychiatric Association: Task Force Report 14:ECT. Washington, DC, APA, 1978
6. D'Elia G, Raotma H: Is unilateral ECT less effective than bilateral ECT? Br J Psychiatry 126:83–89, 1975
7. Rosenberg J, Pettinati HM: Differential memory complaints after bilateral and unilateral electroconvulsive therapy. Am J Psychiatry (in press)
8. Sternberg ED, Jarvik ME: Memory functions in depression. Arch Gen Psychiatry 33:219–224, 1976
9. Price T: Short- and long-term cognitive effects of ECT, part I: effects on memory. Psychopharmacol Bull 18:81–91, 1982
10. Reitan RM: Validity of the Trail Making Test as an indicator of organic brain damage. Percept Mot Skills 8:271–276, 1958
11. Horne RL, Pettinati HM, Menken M, et al: Dexamethasone in ECT: efficacy for depression and post-ECT amnesia. Biological Psychiatry (in press)
12. Reitan RM: Manual for the Administration of Neuropsychological Test Batteries for Adults and Children. Indianapolis, RM Reitan, 1969
13. McAllister T: Cognitive functioning in the affective disorders. Compr Psychiatry 22:572–586, 1981
14. Wells C: Pseudodementia. Am J Psychiatry 136:895–900, 1979
15. Derogatis LR: SCL-90-R: Administration, Scoring and Procedures Manual I. Baltimore, Clinical Psychometrics Research, 1977
16. Wechsler D: A standardized memory scale for clinical use. J Psychol 19:87–95, 1945
17. Fink M: Convulsive therapy: a risk-benefit analysis. Psychopharmacol Bull 18:110–116, 1982

ECT: Brain Damage

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

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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-

TABLE I

Extent of between-group matching and mean Bender-Gestalt, Benton, and WAIS scores for ECT and control groups

	ECT group		Control group	
	Mean	S.D.	Mean	S.D.
Age	43·86	10·99	42·23	8·61
Years of education	9·86	3·47	9·82	3·08
Bender error score	124·17	87·32	56·82	46·17
Benton error score	18·48	5·28	14·82	5·60
Benton no. correct	1·29	1·76	2·18	2·08
WAIS verbal IQ ..	68·50	16·86	79·72	14·67
WAIS performance IQ	65·68	17·67	75·59	14·64
WAIS full scale IQ	65·73	16·87	76·77	14·65

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 $124 \cdot 27$ for the ECT group and $56 \cdot 82$ for the control group ($t = 3 \cdot 20$, $p < \cdot 01$). The mean Benton error score was $18 \cdot 48$ for the ECT group and $14 \cdot 82$ for the control group ($t = 2 \cdot 20$, $p < \cdot 05$), and the mean Benton number correct score was $1 \cdot 29$ for the ECT group and $2 \cdot 18$ for the control group ($t = 1 \cdot 67$, $p < \cdot 05$). On the WAIS, the ECT and control group respective means were $68 \cdot 50$ and $79 \cdot 72$ for Verbal IQ ($t = 2 \cdot 46$, $p < \cdot 01$), $65 \cdot 68$ and $75 \cdot 59$ for Performance IQ ($t = 2 \cdot 02$, $p < \cdot 05$), and $65 \cdot 73$ and $76 \cdot 77$ for Full Scale IQ ($t = 2 \cdot 32$, $p < \cdot 05$).

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

The mean MMPI Sc Scale score was $40 \cdot 90$ for the 10 ECT patients who completed the MMPI and $36 \cdot 50$ for the 18 control patients who completed the MMPI ($t = \cdot 93$, 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 $41 \cdot 78$; the mean of the corresponding nine control patients was $35 \cdot 89$ ($t = 1 \cdot 07$, 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 $38 \cdot 00$ and the 18 control patients obtained a mean score of $42 \cdot 11$ ($t = 1 \cdot 51$, 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 $38 \cdot 22$ and $45 \cdot 44$ respectively ($t = 2 \cdot 19$, $p < \cdot 05$).

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 ($\chi^2 = 5 \cdot 08$, $p < \cdot 02$). The other clinical psychologist classified 16 ECT patients' protocols and 6 control protocols into the '22 most psychotic' category ($\chi^2 = 9 \cdot 08$, $p < \cdot 01$).

One of the clinical psychologists classified 14 ECT patients' and 8 of the control patients' Bender-Gestalt reproductions into the '22 most psychotic' category ($\chi^2 = 3 \cdot 27$, $p < \cdot 10$). The other psychologist classified 15 ECT patients' Bender-Gestalt reproductions and 7 control patients' reproductions into the '22 most psychotic' category ($\chi^2 = 5 \cdot 80$, $p < \cdot 02$).

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 $\cdot 05$ 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 $\cdot 77$ ($p < \cdot 01$).

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

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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 ($t = ·14$, n.s.) for Verbal IQ; 78·3 and 79·8 ($t = ·24$, 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 intelli-gence. 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.

REFERENCES

1. BENTON, A. L. (1963). *The Benton Visual Retention Test: Clinical and Experimental Applications.* New York: Psychological Corporation.
2. GOLDMAN, H., GOMER, F. E., and TEMPLER, D. I. (1972). 'Long-term effects of electroconvulsive therapy upon memory and perceptual-motor performance.' *J. clin. Psychol.*, 28, 32–4.
3. PASCAL, G. R., and SUTTELL, B. J. (1951). *The Bender-Gestalt Test.* New York: Paul B. Hoeber.
4. WATSON, C. G. (1971). 'An MMPI scale to separate brain-damaged from schizophrenic men.' *J. consult. clin. Psychol.*, 36, 121–5.
5. YATES, A. J. (1954). 'The validity of some psychological tests of brain damage.' *Psychol. Bull.*, 51, 359–79.

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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[2, 3, 6, 7, 8, 10, 11, 13]. There appear to be only two investigations that determined the cognitive effects of ECT after a number of months[6, 9]. However, in both of these studies neither control patients nor an adequate number of ECT patients were employed. In the report of Pascal and Zeaman[6], a patient's Wechsler-Bellevue and Rorschach scores before 10 ECT and 7 months afterward were comparable. Stone[9] reported that a patient's Henmon-Nelson Test of Mental Ability score 60 days after the last of 20 ECT was comparable to her score of 7 years earlier.

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

METHOD

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

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

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

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The Bender-Gestalt and Benton were selected because they are well established tests that reflect brain pathology and because they have quantitative scoring systems. The Pascal and Suttell[5] method of scoring for deviations on the Bender-Gestalt designs was employed. Two scoring systems were used for the Benton: (1) the number of correct reproductions or "number correct scores", and (2) "error scores" that consisted of a detailed analysis of specific errors in each figure of each card[1]. The interscorer reliability coefficients between two scorers were .90 ($p < .005$) for the Bender-Gestalt error scores, .97 ($p < .005$) for the Benton error scores, and .94 ($p < .005$) for the Benton number correct scores.

RESULTS

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

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

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

CONCLUSIONS

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

Nevertheless, it cannot be inferred with complete certainty that ECT causes permanent brain pathology. It is possible that schizophrenic patients more likely to receive ECT are those whose psychotic symptomatology is more severe. And, it has been reported that patients with the so-called functional psychiatric disorders tend to do poorly on tests of organicity[12]. Therefore, one cannot be absolutely positive that the ECT and control groups were equated for degree of pre-ECT psychopathology.

REFERENCES

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

7. STONE, C. P. Characteristic losses and gains on the Wechsler Memory Scales as applied on psychotic patients before, during, and after a series of electroconvulsive shocks. *Amer. Psychologist*, 1946, *1*, 245.

8. STONE, C. P. Losses and gains on the Alpha group examination as related to electroconvulsive shocks. *J. comp. physiol. Psychol.*, 1947, *40*, 183–189.

9. STONE, C. P. Pre-illness test records compared with performances during and after electroconvulsive shocks. *J. abn. soc. Psychol.*, 1950, *45*, 154-159.

10. SUMMERSKILL, J., SEEMAN, W. and MEALES, O. W. An evaluation of post electroshock confusion with the Reiter apparatus. *Amer. J. Psychiat.*, 1952, *100*, 835-838.

11. WORCHEL, P. and NARCISO, J. C. Electroshock convulsions and memory: The interval between learning and shock. *J. abn. soc. Psychol.*, 1950, *45*, 85-98.

12. YATES, A. J. The validity of some psychological tests of brain damage. *Psychol. Bull.*, 1954, *51*, 359-379.

13. ZAMORA, E. N. and KAELBLING, R. Memory and electro-convulsive therapy. *Amer. J. Psychiat.*, 1965, *122*, 546-554.

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

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PROBLEM

Malvaria is a psychiatric disease proposed by Hoffer and Osmond[7], the criterion for which is simply a mauve chromatograph stain extracted from the urine. Mauve producers were either schizophrenic or displayed features of this diagnosis. Other studies[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[3], but another investigator[4] reported that kryptopyrrol produced the mauve. This substance is unlikely to result from tranquillizers. The Hoffer-Osmond[5] Diagnostic test (HOD), a self-rating set of true-false statements, differentiated between mauve and nonmauve producing patients in the same way that it differentiated between schizophrenics and neurotics[6].

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

METHOD

From the psychiatric ward of a teaching general hospital, 82 patients were obtained, all of whom were examined during the first few days after admission. Only 14 were on any 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.

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 longer 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). 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±12.45

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).
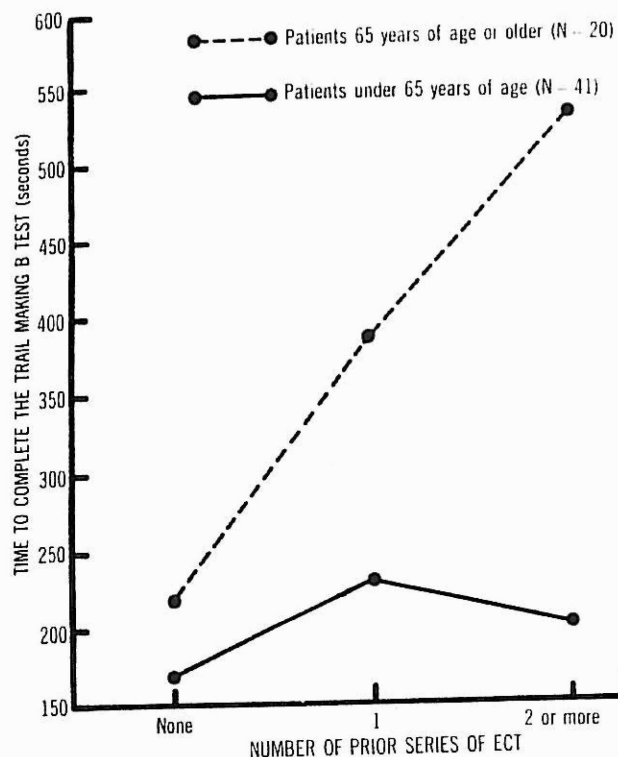
According to psychiatrist ratings, the older patients were not more depressed than the younger ones. On the Hamilton depression scale, older patients had a mean ($\pm$SD) of 19.60 ± 7.28 compared with a mean of 20.22 ± 7.24 for younger patients; on the BPRS, older patients scored an average of 36.10 ± 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) (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

FIGURE 1. Performance on Trail Making B Test by Age and Amount of Prior ECT

FIGURE 2. Severity of Depression Found on the Hamilton Rating Scale at Time of Admission by Age and Amount of Prior ECT
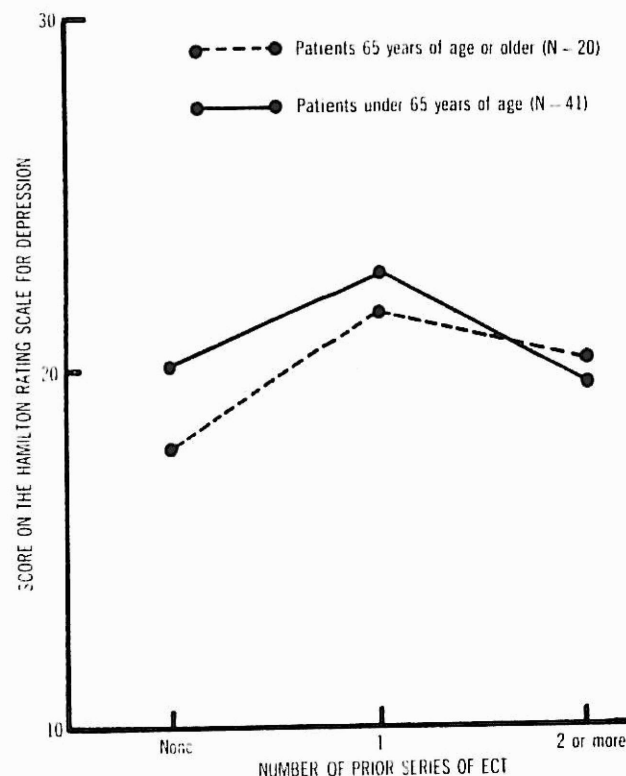
differences between younger patients with and without a history of ECT in the amount of time needed to complete the B test.

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

REFERENCES

1. Fraser RM: ECT and the elderly, in Electroconvulsive Therapy: An Appraisal. Edited by Palmer R. Oxford, Oxford University Press, 1981
2. Weiner RD: The role of electroconvulsive therapy in the treatment of depression in the elderly. J Am Geriatr Soc 30:710–712, 1982
3. Albert M: Geriatric neuropsychology. J Consult Clin Psychol 49:835–850, 1981
4. Salzman C: A primer on geriatric psychopharmacology. Am J Psychiatry 139:67–78, 1982
5. American Psychiatric Association: Task Force Report 14:ECT. Washington, DC, APA, 1978
6. D'Elia G, Raotma H: Is unilateral ECT less effective than bilateral ECT? Br J Psychiatry 126:83–89, 1975
7. Rosenberg J, Pettinati HM: Differential memory complaints after bilateral and unilateral electroconvulsive therapy. Am J Psychiatry (in press)
8. Sternberg ED, Jarvik ME: Memory functions in depression. Arch Gen Psychiatry 33:219–224, 1976
9. Price T: Short- and long-term cognitive effects of ECT, part I: effects on memory. Psychopharmacol Bull 18:81–91, 1982
10. Reitan RM: Validity of the Trail Making Test as an indicator of organic brain damage. Percept Mot Skills 8:271–276, 1958
11. Horne RL, Pettinati HM, Menken M, et al: Dexamethasone in ECT: efficacy for depression and post-ECT amnesia. Biological Psychiatry (in press)
12. Reitan RM: Manual for the Administration of Neuropsychological Test Batteries for Adults and Children. Indianapolis, RM Reitan, 1969
13. McAllister T: Cognitive functioning in the affective disorders. Compr Psychiatry 22:572–586, 1981
14. Wells C: Pseudodementia. Am J Psychiatry 136:895–900, 1979
15. Derogatis LR: SCL-90-R: Administration, Scoring and Procedures Manual I. Baltimore, Clinical Psychometrics Research, 1977
16. Wechsler D: A standardized memory scale for clinical use. J Psychol 19:87–95, 1945
17. Fink M: Convulsive therapy: a risk-benefit analysis. Psychopharmacol Bull 18:110–116, 1982

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 (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 tetanic 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 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 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-

ECT: Brain Damage

(17)

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-

TABLE I

Extent of between-group matching and mean Bender-Gestalt, Benton, and WAIS scores for ECT and control groups

	ECT group		Control group	
	Mean	S.D.	Mean	S.D.
Age	43·86	10·99	42·23	8·61
Years of education	9·86	3·47	9·82	3·08
Bender error score	124·17	87·32	56·82	46·17
Benton error score	18·48	5·28	14·82	5·60
Benton no. correct	1·29	1·76	2·18	2·08
WAIS verbal IQ ..	68·50	16·86	79·72	14·67
WAIS performance IQ	65·68	17·67	75·59	14·64
WAIS full scale IQ	65·73	16·87	76·77	14·65

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 $124 \cdot 27$ for the ECT group and $56 \cdot 82$ for the control group ($t = 3 \cdot 20$, $p < \cdot 01$). The mean Benton error score was $18 \cdot 48$ for the ECT group and $14 \cdot 82$ for the control group ($t = 2 \cdot 20$, $p < \cdot 05$), and the mean Benton number correct score was $1 \cdot 29$ for the ECT group and $2 \cdot 18$ for the control group ($t = 1 \cdot 67$, $p < \cdot 05$). On the WAIS, the ECT and control group respective means were $68 \cdot 50$ and $79 \cdot 72$ for Verbal IQ ($t = 2 \cdot 46$, $p < \cdot 01$), $65 \cdot 68$ and $75 \cdot 59$ for Performance IQ ($t = 2 \cdot 02$, $p < \cdot 05$), and $65 \cdot 73$ and $76 \cdot 77$ for Full Scale IQ ($t = 2 \cdot 32$, $p < \cdot 05$).

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

The mean MMPI Sc Scale score was $40 \cdot 90$ for the 10 ECT patients who completed the MMPI and $36 \cdot 50$ for the 18 control patients who completed the MMPI ($t = \cdot 93$, 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 $41 \cdot 78$; the mean of the corresponding nine control patients was $35 \cdot 89$ ($t = 1 \cdot 07$, 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 $38 \cdot 00$ and the 18 control patients obtained a mean score of $42 \cdot 11$ ($t = 1 \cdot 51$, n.s.). For the nine cases in which the ECT patients and their controls both completed the MMPI, the mean Sc-O Scale scores were $38 \cdot 22$ and $45 \cdot 44$ respectively ($t = 2 \cdot 19$, $p < \cdot 05$).

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 ($\chi^2 = 5 \cdot 08$, $p < \cdot 02$). The other clinical psychologist classified 16 ECT patients' protocols and 6 control protocols into the '22 most psychotic' category ($\chi^2 = 9 \cdot 08$, $p < \cdot 01$).

One of the clinical psychologists classified 14 ECT patients' and 8 of the control patients' Bender-Gestalt reproductions into the '22 most psychotic' category ($\chi^2 = 3 \cdot 27$, $p < \cdot 10$). The other psychologist classified 15 ECT patients' Bender-Gestalt reproductions and 7 control patients' reproductions into the '22 most psychotic' category ($\chi^2 = 5 \cdot 80$, $p < \cdot 02$).

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 $\cdot 05$ 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 $\cdot 77$ ($p < \cdot 01$).

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

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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 ($t = 2\cdot28$, $p < \cdot05$) for Bender-Gestalt error score; 15·9 and 14·0 ($t = 1\cdot01$, n.s.) for Benton error score; 2·10 and 2·00 ($t = 1\cdot00$, n.s.) for Benton number correct score; 77·1 and 82·1 ($t = \cdot14$, n.s.) for Verbal IQ; 78·3 and 79·8 ($t = \cdot24$, n.s.) for Performance IQ; and 76·1 and 80·1 ($t = \cdot78$, 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.

REFERENCES

1. BENTON, A. L. (1963). *The Benton Visual Retention Test: Clinical and Experimental Applications.* New York: Psychological Corporation.
2. GOLDMAN, H., GOMER, F. E., and TEMPLER, D. I. (1972). 'Long-term effects of electroconvulsive therapy upon memory and perceptual-motor performance.' *J. clin. Psychol.*, 28, 32-4.
3. PASCAL, G. R., and SUTTELL, B. J. (1951). *The Bender-Gestalt Test.* New York: Paul B. Hoeber.
4. WATSON, C. G. (1971). 'An MMPI scale to separate brain-damaged from schizophrenic men.' *J. consult. clin. Psychol.*, 36, 121-5.
5. YATES, A. J. (1954). 'The validity of some psychological tests of brain damage.' *Psychol. Bull.*, 51, 359-79.

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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 [2, 3, 6, 7, 8, 10, 11, 13]. There appear to be only two investigations that determined the cognitive effects of ECT after a number of months [6, 9]. However, in both of these studies neither control patients nor an adequate number of ECT patients were employed. In the report of Pascal and Zeaman [6], a patient's Wechsler-Bellevue and Rorschach scores before 10 ECT and 7 months afterward were comparable. Stone [9] reported that a patient's Henmon-Nelson Test of Mental Ability score 60 days after the last of 20 ECT was comparable to her score of 7 years earlier.

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

METHOD

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

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

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

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2. ERWIN, E. F. and
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3. KENDALL, B. S., N
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4. NIELSON, J. M. *A
5. PASCAL, G. R. and
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The Bender-Gestalt and Benton were selected because they are well established tests that reflect brain pathology and because they have quantitative scoring systems. The Pascal and Suttell[5] method of scoring for deviations on the Bender-Gestalt designs was employed. Two scoring systems were used for the Benton: (1) the number of correct reproductions or "number correct scores", and (2) "error scores" that consisted of a detailed analysis of specific errors in each figure of each card[1]. The interscorer reliability coefficients between two scorers were .90 ($p < .005$) for the Bender-Gestalt error scores, .97 ($p < .005$) for the Benton error scores, and .94 ($p < .005$) for the Benton number correct scores.

RESULTS

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

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

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

CONCLUSIONS

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

Nevertheless, it cannot be inferred with complete certainty that ECT causes permanent brain pathology. It is possible that schizophrenic patients more likely to receive ECT are those whose psychotic symptomatology is more severe. And, it has been reported that patients with the so-called functional psychiatric disorders tend to do poorly on tests of organicity[12]. Therefore, one cannot be absolutely positive that the ECT and control groups were equated for degree of pre-ECT psychopathology.

REFERENCES

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

7. STONE, C. P. Characteristic losses and gains on the Wechsler Memory Scales as applied on psychotic patients before, during, and after a series of electroconvulsive shocks. *Amer. Psychologist*, 1946, *1*, 245.

8. STONE, C. P. Losses and gains on the Alpha group examination as related to electroconvulsive shocks. *J. comp. physiol. Psychol.*, 1947, *40*, 183-189.

9. STONE, C. P. Pre-illness test records compared with performances during and after electroconvulsive shocks. *J. abn. soc. Psychol.*, 1950, *45*, 154-159.

10. SUMMERSKILL, J., SEEMAN, W. and MEALES, O. W. An evaluation of post electroshock confusion with the Reiter apparatus. *Amer. J. Psychiat.*, 1952, *100*, 835-838.

11. WORCHEL, P. and NARCISO, J. C. Electroshock convulsions and memory: The interval between learning and shock. *J. abn. soc. Psychol.*, 1950, *45*, 85-98.

12. YATES, A. J. The validity of some psychological tests of brain damage. *Psychol. Bull.*, 1954, *51*, 359-379.

13. ZAMORA, E. N. and KAELBLING, R. Memory and electro-convulsive therapy. *Amer. J. Psychiat.*, 1965, *122*, 546-554.

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

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PROBLEM

Malvaria is a psychiatric disease proposed by Hoffer and Osmond[7], the criterion for which is simply a mauve chromatograph stain extracted from the urine. Mauve producers were either schizophrenic or displayed features of this diagnosis. Other studies[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[3], but another investigator[4] reported that kryptopyrrol produced the mauve. This substance is unlikely to result from tranquillizers. The Hoffer-Osmond[5] Diagnostic test (HOD), a self-rating set of true-false statements, differentiated between mauve and nonmauve producing patients in the same way that it differentiated between schizophrenics and neurotics[6].

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

METHOD

From the psychiatric ward of a teaching general hospital, 82 patients were obtained, all of whom were examined during the first few days after admission. Only 14 were on any 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.

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1. CRAIG, W. J.
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2. CRAIG, W. J
   26, 91-97.
3. ELLMAN, G.
   Psychiat., 1968,
4. IRVINE, D. C
   human urine an
5. KELM, H. T
6. HOFFER, A.
7. HOFFER, A.
   39, 335-66.
8. O'REILLY, P
   1965, *111*, 741

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 longer 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). 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 ($\pm$SD) age of 69.7$\pm$3.76 years. Forty-one patients (29 women and 12 men) were under age 65, with an average age of 41.7$\pm$12.45

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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).
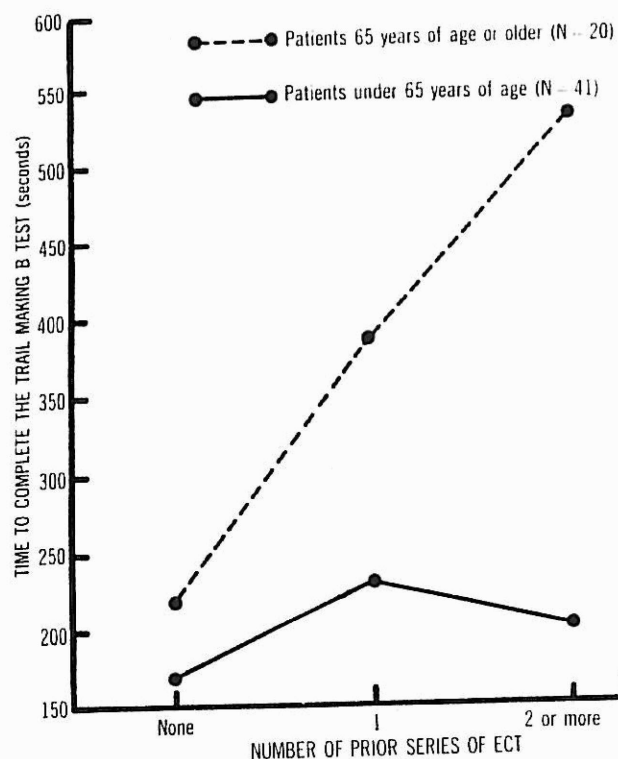
According to psychiatrist ratings, the older patients were not more depressed than the younger ones. On the Hamilton depression scale, older patients had a mean (±SD) of 19.60±7.28 compared with a mean of 20.22±7.24 for younger patients; on the BPRS, older patients scored an average of 36.10±9.23 while 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

FIGURE 1. Performance on Trail Making B Test by Age and Amount of Prior ECT

FIGURE 2. Severity of Depression Found on the Hamilton Rating Scale at Time of Admission by Age and Amount of Prior ECT
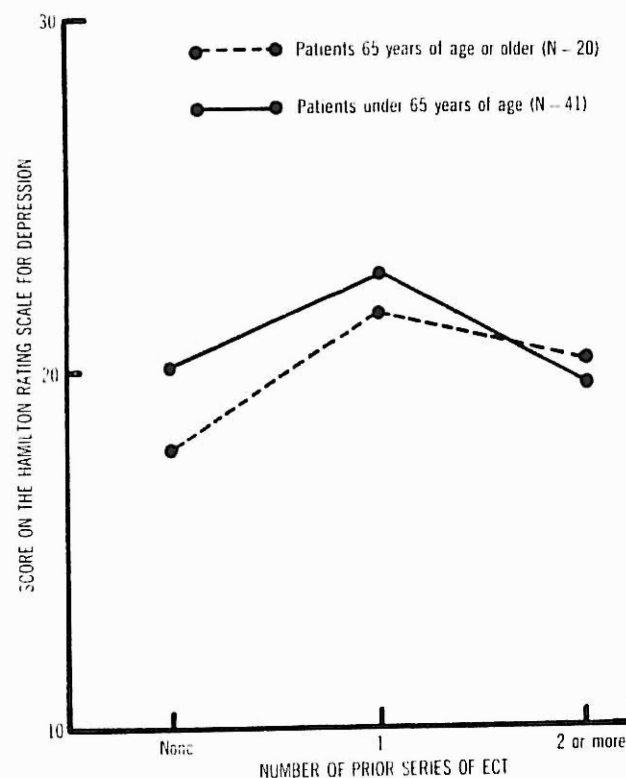

differences between younger patients with and without a history of ECT in the amount of time needed to complete the B test.

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

REFERENCES

1. Fraser RM: ECT and the elderly, in Electroconvulsive Therapy: An Appraisal. Edited by Palmer R. Oxford, Oxford University Press, 1981
2. Weiner RD: The role of electroconvulsive therapy in the treatment of depression in the elderly. J Am Geriatr Soc 30:710–712, 1982
3. Albert M: Geriatric neuropsychology. J Consult Clin Psychol 49:835–850, 1981
4. Salzman C: A primer on geriatric psychopharmacology. Am J Psychiatry 139:67–78, 1982
5. American Psychiatric Association: Task Force Report 14:ECT. Washington, DC, APA, 1978
6. D'Elia G, Raotma H: Is unilateral ECT less effective than bilateral ECT? Br J Psychiatry 126:83–89, 1975
7. Rosenberg J, Pettinati HM: Differential memory complaints after bilateral and unilateral electroconvulsive therapy. Am J Psychiatry (in press)
8. Sternberg ED, Jarvik ME: Memory functions in depression. Arch Gen Psychiatry 33:219–224, 1976
9. Price T: Short- and long-term cognitive effects of ECT, part I: effects on memory. Psychopharmacol Bull 18:81–91, 1982
10. Reitan RM: Validity of the Trail Making Test as an indicator of organic brain damage. Percept Mot Skills 8:271–276, 1958
11. Horne RL, Pettinati HM, Menken M, et al: Dexamethasone in ECT: efficacy for depression and post-ECT amnesia. Biological Psychiatry (in press)
12. Reitan RM: Manual for the Administration of Neuropsychological Test Batteries for Adults and Children. Indianapolis, RM Reitan, 1969
13. McAllister T: Cognitive functioning in the affective disorders. Compr Psychiatry 22:572–586, 1981
14. Wells C: Pseudodementia. Am J Psychiatry 136:895–900, 1979
15. Derogatis LR: SCL-90-R: Administration, Scoring and Procedures Manual I. Baltimore, Clinical Psychometrics Research, 1977
16. Wechsler D: A standardized memory scale for clinical use. J Psychol 19:87–95, 1945
17. Fink M: Convulsive therapy: a risk-benefit analysis. Psychopharmacol Bull 18:110–116, 1982

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 (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 tetanic 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 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 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-

taneous recall of personal preshock data are employed, no one else will know either.

The memory loss following ECT generally follows Ribot's law for all pathological amnesias: the new dies before the old. This, of course, is the opposite of normal forgetting. Squire, however, has shown that the loss may extend to items learned more than 30 years before (41).

The effect of ECT on memory was common knowledge within a few years of its introduction. There were reports of persons who forgot they had children (42, 43), although most amnesias involved humbler matters, such as the woman who forgot how to cook familiar dishes (44) and another who couldn't remember her own clothing and demanded to know who had put the unfamiliar dresses in her closet (45). Some doctors dismissed these sequelae as trivial or transient, although one psychiatrist remarked that psychotherapy was useless in patients undergoing ECT because they couldn't remember "either the analyst or the content of the analytic sessions from one day to the next" (46).

Numerous such case reports finally led to a definitive study of the effects of ECT on memory by Irving Janis in 1950 (47). He found that all 19 subjects in a controlled prospective investigation had significant memory loss 4 weeks after ECT, compared to negligible losses among control subjects. He also noted that these losses may involve events of early childhood dating back 20 to 40 years, with the more recently encoded memories being the most vulnerable. Patient E, for example, a 38-year-old woman, had told Janis in an interview prior to ECT that thyroid medication had caused heart palpitations and panic which led to her admission to the psychiatric hospital. When asked after a course of 10 shocks if she had ever taken thyroid she responded, "I don't think so."

In the late 1940s, when the enthusiasm for ECT seemed to have passed its peak (48), Lancaster and associates (49) advocated the use of unilateral nondominant ECT in treating patients who earn their livelihood with retained knowledge. In this variant the current path and most of the damage is confined to the nonverbal side of the brain, usually the right hemisphere. This exploits the well-known neurological phenomenon of anosognosia, or denial, that is associated with right-hemisphere lesions—victims can't verbalize their difficulties. They complain less. Cohen and associates (50), however, using design-completion tests, proved that shock to the right hemisphere produces its own kind of memory loss—visual and spatial. Inglis found in 1970 (51) that the effects of unilateral ECT were comparable to those of right and left temporal lobectomy, with identical impairment of memory and learning.

Recently there has been a good deal of human experimentation in a futile effort to find electrode placements that eliminate amnesia. As the use of ECT has shifted from state hospitals to private practice, the literature has focused more and more on memory loss.

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.[1]

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

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[1] Sham ECT, an essential control technique, has been employed in only two studies, which were tests of efficacy, not tests of memory loss. Neither study showed any superiority of ECT over the control treatment (52, 53).

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 outselves whether we, as doctors sworn to the Hippocratic Oath, should be offering it.

REFERENCES

1. Friedberg J: Shock Treatment Is Not Good For Your Brain. San Francisco, Glide Publications, 1976
2. Jaffe R: Electropathology: a review of the pathologic changes produced by electric current. Arch of Psychiatry S:868–864, 1928
3. Cerletti U: Electroshock therapy, in The Great Physiodynamic Therapies in Psychiatry. Edited by Sackler A, Sackler R, Sackler M, et al. New York, Hoeber-Harper, 1956, pp 91–120
4. Bini L: Experimental researches on epileptic attacks induced by the electric current. Am J Psychiatry 94:172–174, 1938
5. Heilbrunn G, Liebert E: Biopsies on the brain following artificially produced convulsions. Arch Neurol Psychiatry 46:548–552, 1941
6. Neubuerger KT, Whitehead RW, Rutledge RK, et al: Pathologic changes in the brains of dogs given repeated electric shocks. Am J Med Sci 204:381–387, 1942
7. Heilbrunn G, Weil A: Pathologic changes in the central nervous system in experimental electric shock. Arch Neurol Psychiatry 47:918, 1942
8. Alpers BJ, Hughes J: Changes in the brain after electrically induced convulsions in cats. Arch Neurol Psychiatry 47:385, 1942
9. Alexander L, Lowenbach H: Experimental studies on electroshock treatment: the intracerebral vascular reaction as an indicator of the path of the current and the threshold of early changes within the brain tissue. Neuropathol Exp Neurol 3:139, 1944
10. Ferraro A, Roizin L, Helfand M: Morphologic changes in the brain of monkeys following convulsions electrically induced. J Neuropathol Exp Neurol 5:285, 1946
11. Ferraro A, Roizin L: Cerebral morphologic changes in monkeys subjected to a large number of electrically induced convulsions (32-100). Am J Psychiatry 106:278, 1949
12. Hartelius H: Cerebral changes following electrically induced convulsions. Acta Psychiat et Neurol Scand Supplement 77, 1952
13. Meldrum B, Roger V, Brierley J: Systemic factors and epileptic brain damage. Arch Neurol 29:82–87, 1973
14. Meldrum B, Horton R, Brierley J: Epileptic brain damage in adolescent baboons following seizures induced by allylglycine. Brain 97:407–418, 1974
15. Alpers BJ, Hughes J: The brain changes in electrically induced convulsions in the human. J Neuropathol Exp Neurol 1:173, 1942
16. Ebaugh FG, Barnacle CH, Neubuerger KT: Fatalities following electric convulsive therapy: report of two cases, with autopsy. Arch Neurol Psychiatry 49:107, 1943
17. Riese W: Report of two new cases of sudden death after electric shock treatment with histopathological findings in the central nervous system. J Neuropathol Exp Neurol 7:98–100, 1948
18. Larsen EG, Vraa-Jansen G: Ischaemic changes in the brain following electroshock therapy. Acta Psychiat et Neurol Scand 28:75–80, 1953
19. Impastato D: Prevention of fatalities in electroshock therapy. Dis Nerv Syst 18:34–75, 1957
20. Allen I: Cerebral lesions from electric shock treatment. NZ Med J 58:369, 1959
21. McKegney FP, Panzetta AF: An unusual fatal outcome of electro-convulsive therapy. Am J Psychiatry 120:398–400, 1963
22. Ebaugh FG, Barnacle CH, Neubuerger KT: Fatalities following electric convulsive therapy. A report of 2 cases with autopsy findings. Trans Am Neurol Assoc, June 1942, p 36
23. Gralnick A: Fatalities associated with electric shock treatment of psychoses: report of two cases, with autopsy observations in one of them. Arch Neurol Psychiatry 51:397, 1944
24. Jetter WW: Fatal circulatory failure caused by electric shock therapy. Arch Neurol Psychiatry 51:557, 1944
25. Meyer A, Teare D: Cerebral fat embolism after electrical convulsion therapy. Br Med J 2:42, 1945
26. Sprague DW, Taylor RC: The complications of electric shock therapy with a case study. Ohio State Med J 44:51–54, 1948
27. Will OA Jr, Rehfeldt FC: A fatality in electroshock therapy: report of a case and review of certain previously described cases. J Nerv Ment Dis 107:105–126, 1948
28. Martin PA: Convulsive therapies: review of 511 cases at Pontiac State Hospital. J Nerv Ment Dis 109:142–157, 1949
29. Riese W, Fultz GS: Electric shock treatment succeeded by complete flaccid paralysis, hallucinations, and sudden death: case report with anatomical findings in the central nervous system. Am J Psychiatry 106:206–211, 1949
30. Liban E, Halpern L, Rozanski J: Vascular changes in the brain in a fatality following electroshock. J Neuropathol Exp Neurol 10:309–318, 1951
31. Corsellis J, Meyer A: Histological changes in the brain after uncomplicated electro-convulsant treatment. J Ment Sci 100:375–383, 1954
32. Madow L: Brain changes in electroshock therapy. Am J Psychiatry 113:337–347, 1956
33. Faurbye A: Death under electroshock treatment. Acta Psychiat et Neurologica 17:39, 1942   also #18
34. Maclay WS: Death due to treatment. Proc Soc Med 46:13–20, 1953
35. Matthew JR, Constan E: Complications following ECT over a three-year period in a state institution. Am J Psychiatry 120:1119–1120, 1964
36. Barker J, Baker A: Deaths associated with electroplexy. J Ment Sci 105:339–348, 1959
37. Blaurock M, Lorimer F, Segal M, et al.: Focal electroencephalographic changes in unilateral electric convulsion therapy. Arch Neurol Psychiatry 64:220–226, 1950
38. Sutherland E, Oliver J, Knight D: EEG, memory and confusion in dominant, non-dominant and bi-temporal ECT. Br J Psychiatry 115:1059–1064, 1969
39. Wada J, Osawa T: Spontaneous recurrent seizure state induced by daily electric amygdaloid stimulation in senegalese baboons (papio papio). Neurology 26:273–286, 1976
40. Parfitt D: Persisting epilepsy following shock therapy. Br Med J 2:514, 1942
41. Squire L: A thirty-year retrograde amnesia following electroconvulsive therapy in depressed patients. Presented at the 3rd annual meeting of the Society for Neuroscience, San Diego, Calif, 1973
42. Tyler B, Lowenbach H: Polydiurnal electric shock treatment in mental disorders. NC Med J 8:577–582, 1947
43. Medlicott R: Convulsive therapy. Results and complications in four hundred cases. NZ Med J 47:538, 1948
44. Brody M: Prolonged memory defects following electro-therapy. J Ment Sci 90:777–779, 1944
45. Zubin J: Objective studies of disordered persons in Methods of Psychology. Edited by Andrews T. New York, John Wiley & Sons, 1948, pp 595–623
46. Stainbrook E: Shock therapy: psychologic theory and research. Psychol Bull 43:21–60, 1956
47. Janis I: Psychologic effects of electric convulsive treatments. Part 1: post-treatment amnesias. J Nerv Ment Dis 3:359–382, 1950
48. Spiegel E (ed): Progress In Neurology and Psychiatry: An Annual Review. New York, Grune & Stratton, 1957
49. Lancaster N, Steinert R, Frost I: Unilateral electro-convulsive therapy. J Ment Sci 104:221–227, 1958

50. Cohen B, Noblin C, Silverman A: Functional asymmetry of the human brain. Science 162:475–477, 1968
51. Inglis J: Shock, surgery and cerebral asymmetry. Br J Psychiatry 117:143–148, 1970
52. Miller D, Clancy J, Cummings E: A comparison between unidirectional current nonconvulsive electrical stimulation given with Reiter's machine, standard alternating current electroshock (Cerletti method), and pentothal in chronic schizophrenia. Am J Psychiatry 109:617–620, 1953
53. Brill H, Crumpton E, Eiduson S, et al: Relative effectiveness of various components of electroconvulsive therapy. Arch Neurol Psychiatry 81:627–635, 1959
54. Templer D, Ruff C, Armstrong G: Cognitive functioning and degree of psychosis in schizophrenics given many electroconvulsive treatments. Br J Psychiatry 123:441–443, 1973
55. Penfield W, Perot P: The brain's record of auditory and visual experience. Brain 86, Part 4, 1963
56. Scoville W, Milner B: Loss of recent memory after bilateral hippocampal lesions. J Neurol Neurosurg Psychiatry 20:11–21, 1957
57. Friedberg J: Electroshock therapy: let's stop blasting the brain. Psychology Today 9(8):18–23, 1975
58. Kalinowsky LB: The convulsive therapies, in Comprehensive Textbook of Psychiatry, 2nd ed, vol II. Edited by Freedman AM, Kaplan HI, Sadock BJ. Baltimore, Williams & Wilkins Co, 1975, pp 1972–1973

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 Bini reference:

We have so far employed exclusively the method of Viale, which consists of passing the street current (120 volts) 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