Brit. J. Psychiat. (1973), 123, 441–3

ECT: Brain Damage

(8)

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

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

Problem

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

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

Method

Subjects were 14 male and 30 female schizophrenics in Western State Hospital, Hopkinsville, Kentucky. Of these patients 22 had a history of from 40 to 263 ECT with a median number of 58·5. All ECT was administered earlier than seven years ago. The 22 control patients were matched for age, sex, race, and level of education. Table I indicates the extent of the between-groups matching.

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

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

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

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

psychologists were given the following instructions:

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

Results

As indicated in Table I, the mean error score on the Bender-Gestalt was 124·27 for the ECT group and 56·82 for the control group ($t = 3{\cdot}20$, $p < {\cdot}01$). The mean Benton error score was 18·48 for the ECT group and 14·82 for the control group ($t = 2{\cdot}20$, $p < {\cdot}05$), and the mean Benton number correct score was 1·29 for the ECT group and 2·18 for the control group ($t = 1{\cdot}67$, $p < {\cdot}05$). On the WAIS, the ECT and control group respective means were 68·50 and 79·72 for Verbal IQ ($t = 2{\cdot}46$, $p < {\cdot}01$), 65·68 and 75·59 for Performance IQ ($t = 2{\cdot}02$, $p < {\cdot}05$), and 65·73 and 76·77 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 ·07 (n.s.); between number of ECT and Benton error score, ·34 ($p < {\cdot}10$); between number of ECT and Benton number correct score, ·37 ($p < {\cdot}05$); between number of ECT and Verbal IQ, ·10 (n.s.); between number of ECT and Performance IQ, ·34 ($p < {\cdot}10$); between number of ECT and Full Scale IQ, ·26 (n.s.).

The mean MMPI Sc Scale score was 40·90 for the 10 ECT patients who completed the MMPI and 36·50 for the 18 control patients who completed the MMPI ($t = {\cdot}93$, 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 41·78; the mean of the corresponding nine control patients was 35·89 ($t = 1{\cdot}07$, 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 38·00 and the 18 control patients obtained a mean score of 42·11 ($t = 1{\cdot}51$, 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 38·22 and 45·44 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 ·05 level in three of these comparisons. Furthermore, for the 10 ECT patients who completed the MMPI, the correlation coefficient between number of ECT received and Sc Scale score is ·77 ($p < {\cdot}01$).

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

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 intelligence. However, with the 22 ECT patients and their 22 control patients, the greatest level of significance was obtained with the Bender-Gestalt. Such a finding was also reported in the Goldman *et al.* study. The ECT patients' inferior Bender-Gestalt performance does suggest that ECT causes permanent brain damage.

ACKNOWLEDGEMENT

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

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.

Donald I. Templer, PH.D.,* *Chief Psychologist,*
Carol F. Ruff, M.A., *Chief Psychologist,*
Gloria Armstrong, M.A., *Psychologist,*
Western State Hospital, Hopkinsville, Kentucky 42240, U.S.A.

(*Received 29 August 1972*)

*Present address: Pleasant Grove Hospital, Louisville, Kentucky 40223

ECT INFO PACKET

1. Disabling the Brain with Electroshock Peter Roger Breggin
2. Neuropathology and Cognitive Dysfunction From ECT Peter R. Breggin
3. Peter Breggin's Testimony
4. Shock Treatment, Brain Damage, and Memory Loss: A Neurological Perspective John Friedberg
5. ECT: Is It Unsafe and Ineffective? Edward M. Opton, Jr.
6. Concussing the Brain with Electroconvulsive Shock Terapy (ECT): An Appropriate Treatment for Depression and Suicidal Ideation Gregory J. Boyle
7. Can ECT Permanently Harm the Brain? Donald Templer and David Veleber
8. Cognitive Functioning and Degree of Psychosis in Schizophrenics given many Electroconvulsive Treatments Donald Templer, Carol Ruff and Gloria Armstrong
9. Long-Term Effects of Electroconvulsive Terapy Upon Memory and Perceptual-Motor Performance Herbert Goldman, Frank Gomer and Donald Templer
10. Electroconvulsive Terapy and Complaints of Memory Dysfunction: A Prospective Three-Year Follu-up Study Larry Squire and Pamela Slater
11. Cognitive Functioning in Depressed Geriatric Patients with a History of ECT Helen Pettinati and Kathryn Bonner
12. The Effects of ECT Modifications on Autobiographical and Verbal Memoray Walter Daniel, Herbert Crovitz, Richard Weiner and Helen Rogers

13A. Multiple-Monitored Electroconvulsive Therapy Barry Maletzky

13B. Electroconvulsive Therapy NIMH/NIH Consensus Conference

14. Differential Memory Complaints After Bilateral and Unilateral ECT Joanne Rosenberg and Helen Pettinati
15. In Favor of Wider ECT Ban Sidney Sament
16. Electroconvulsive Therapy Report of the Task Force on ECT of the APA
17. Electroshock: key element in personality-changing therapy
18. The Structure of Psychiatry in the Soviet Union Edward Babayan
19. Neurologic fragments. II. Remarks on anosognosia, confabulation, memory, and other topics; and an appendix on self-observation C. Miller Fisher
20. ECT-Ban Controversy Peter Breggin
21. Electroshock terapy and brain damage: The acute organic brain syndrome as treatment Peter Breggin
22. The Shame of My Life Peter Breggin
23. Memory Complaint After Electroconvulsive Therapy: Assessment with a New Self-Rating Instrument Larry Squire, Wetzel, and Slater
24. ECT III: Enduring Cognitive Defecits (sic)? D.Weeks, C.P.L. Freeman and R.E. Kendell

ECT: Brain Damage

(9)

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

HERBERT GOLDMAN, FRANK E. GOMER, AND DONALD I. TEMPLER

VA Hospital, Jefferson Barracks, Mo.

PROBLEM

This study investigated whether there are memory and perceptual-motor deficits in patients who have had in excess of 50 electroconvulsive treatments (ECT). A number of investigators have explored the effects of ECT upon psychological tests sensitive to organicity. These researchers usually found decreased performance during and shortly after a course of ECT[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

LO

The Bender-
tests that reflec
systems. The Pa
Gestalt designs
(1) the number o
scores" that cons
card[1]. The ir
($p < .005$) for
error scores, and

As indicated
for the ECT grou
Benton error sco
($t = 1.90$, $p <$
ECT group and 3
For the ECT
and Bender-Gest
Benton error scor
correct score $-.4$
The groups w
investigators mai
important in this
years of hospitali
were .28 for Ben
Benton number c
were .04, .27, and

The significa
Bender-Gestalt ar
course of treatme
more, it seems pla
damaging effect of
between number o
ECT-produced str
observation of pr
treated[4].

Nevertheless,
permanent brain p
to receive ECT ar
it has been report
orders tend to do
lutely positive tha
ECT psychopathol

1. BENTON, A. L. *
tions. New York: P
2. ERWIN, E. F. and
treatment. *Percept*
3. KENDALL, B. S.,
their effect on cog
4. NIELSON, J. M.
5. PASCAL, G. R. a
6. PASCAL, G. R.
the individual pat

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*

W. J. CRAIG

Queen's University and Kingston (Ontario) Psychiatric Hospital

PROBLEM

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

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

METHOD

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

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

Intracerebral hemorrhage following electroconvulsive therapy

Leon A. Weisberg, MD; Debra Elliott, MD; and David Mielke, MD

Muscle relaxants and anesthetic agents are given with electroconvulsive therapy (ECT) to prevent traumatic injuries during convulsion. Cardiovascular or cerebrovascular complications may develop in high-risk patients who are hypertensive or have other cardiac disorders and undergo ECT.[1] We now report a patient who developed an intracerebral hemorrhage (ICH) during ECT.

Case report. A 69-year-old woman was admitted for ECT. She had several recent episodes of depression that had responded to antidepressant medication; however, recent depression had lasted 4 months without improvement despite treatment with amitriptyline, imipramine, lithium, fluoxetine, and alprazolam. She was nonfunctional and required hospitalization.

The patient had no family history of dementia or psychiatric illness, had no recent head trauma, was not hypertensive, and had no prior cerebrovascular symptoms. She did not drink alcohol. When initially admitted to the hospital, her mental state was consistent with severe depression. She had no memory impairment or clinical dementia. Chest radiograph and ECG were normal. Laboratory studies, including complete blood count, platelet count, prothrombin time, partial thromboplastin time, and erythrocyte sedimentation rate, were normal.

ECT was performed on four occasions. The patient was induced with etomidate, and succinylcholine was used for muscle relaxation. Following the first three treatments, the patient had confusion for 15 minutes. After the fourth ECT, confusion lasted 4 hours. She also reported that her vision became blurred and she bumped into objects on her right side. She reported no headache or nausea. Neurologic examination showed right homonymous hemianopia, right pronator drift, right-sided Babinski's sign, and recent memory impairment. During ECT, her blood pressure was recorded at 120 to 135 mm Hg systolic and 60 to 80 mm Hg diastolic. She had no signs of head injury. Laboratory studies, including complete blood count, coagulation profile, collagen vascular studies, ECG, and echocardiogram, were normal. CT showed a left parieto-occipital hemorrhage with no abnormal enhancement (figure, A).

Four days later, she awakened from sleep feeling "strange," and she had bitten her tongue. EEG showed left posterior hemisphere slow-wave activity without spike discharges. Anticonvulsant therapy was initiated with phenytoin for a presumed seizure, and no further seizures occurred. One week later, CT showed that the hemorrhage was resolving. MRI showed the hemorrhage with surrounding edema, but there was no evidence of neoplasm or arteriovenous malformation (figure, B). Four months later, she had no more seizures and visual field examination showed improvement. Depression was markedly improved, and she had no cognitive or memory impairment. No further ECT had been performed. She now lives independently.

Discussion. The well-described adverse effects of ECT are acute confusion and memory impairment.[1] It is controversial whether ECT causes structural brain damage.[2-4] There have been reports of localized edema, gliosis, and petechial hemorrhages located directly underneath the electrode site.[4] The most common systemic complications of ECT are cardiovascular. When the electrical stimulus is applied without atropine pretreatment, there is vagal activation which causes bradycardia and hypotension. When the electrical seizure occurs, there is increased sympathomimetic activity, causing increased arterial blood pressure and tachycardia.[1]

This patient was not hypertensive and had no other cardiovascular disease; however, during the course of ECT she developed a lobar hemorrhage. The CT and MRI findings showed no characteristics to indicate traumatic hemorrhagic contusion or hemorrhagic infarction from a potential cardiac embolism. Without angiography, we cannot rule out an "occult" vascular malformation.[5] The blood coagulation profile showed no evidence of coagulopathy. The etiology of the ICH could not be determined. When an elderly normotensive patient develops a nontraumatic lobar ICH, the possibility of cerebral amyloid angiopathy (CAA) should be considered.[6,7] There was no documented evidence of acute hypertensive episodes during ECT; however, it is probable that CAA patients have fragile vessels that are particularly sensitive to even mild blood pressure elevations that may have occurred during ECT.

In summary, we describe an elderly normotensive woman who developed medically refractive depression and suffered nontraumatic parieto-occipital hemorrhage following ECT. Since CAA is an increasingly recognized cause of behavioral change in elderly patients, it is important to consider this condition as a cause of ICH in elderly patients, especially if ECT is being considered.

From the Department of Psychiatry and Neurology of the Tulane University Medical School, New Orleans, LA.

Received March 22, 1991. Accepted for publication in final form May 1, 1991.

Address correspondence and reprint requests to Dr. Leon A. Weisberg, Department of Psychiatry and Neurology, 1430 Tulane Avenue, New Orleans, LA 70112.

References

1. Sachs GS, Gelenberg AJ. Adverse effects of electroconvulsive therapy. In: Review of psychiatry, vol 7. Washington, DC: American Psychiatric Press, 1988:498-512.
2. Calloway SP, Dolan RJ, Jacoby RJ. ECT and cerebral atrophy: a computed tomographic study. Acta Psychiatr Scand 1981;64:442-445.
3. Kolbeinsson H, Arnaldsson OS, Petursson H. Computed tomographic scans in ECT patients. Acta Psychiatr Scand 1986;73:28-32.
4. Friedberg J. Shock treatment, brain damage and memory loss: a neurologic perspective. Am J Psychiatry 1977;134:1010-1014.
5. Weisberg LA, Stazio A, Shamsnia M, Elliott D. Nontraumatic parenchymal brain hemorrhages. Medicine 1990;69:277-295.
6. Vinters HV. Cerebral amyloid angiopathy. Stroke 1987;18:311-324.
7. Greene GM, Godersky JC, Biller J, Hart MN, Adams HP. Surgical experience with cerebral amyloid angiopathy. Stroke 1990;21:1545-1549.

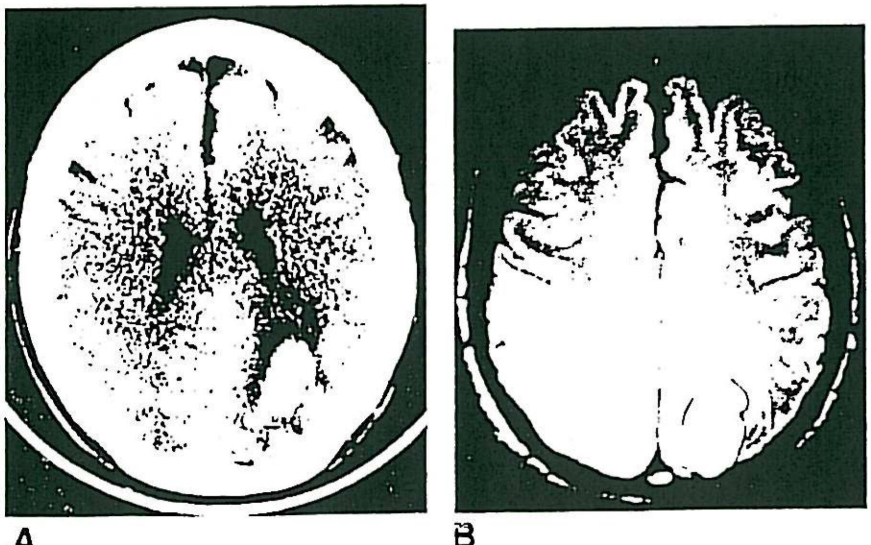

Figure. (A) Initial CT. There is a left (reader's right) posterior parietal hyperdense hemorrhage with surrounding hypodense edema in the white matter and no evidence of enhancement. (B) MRI (performed 2 weeks later). There is a subacute left posterior parieto-occipital hemorrhage. There is a centrally increased signal intensity with a peripheral hypointense rim. Surrounding the hemorrhage, there is high signal intensity in the white matter, representing edema.