Electroconvulsive Therapy and Memory Dysfunction: Is There Evidence for Prolonged Defects?

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The authors reviewed 39 papers which concern the long-term effects of electroconvulsive therapy (ECT) on human memory. Although the authors caution that methodological considerations preclude a decisive assessment, the majority of the studies suggest that ECT does not normally produce prolonged memory defects. Some recent studies do document subtle but persistent defects several months after ECT, especially in personal autobiographical material. These defects appear to be more annoying than seriously incapacitating. Variables considered important in an ideal design of studies on ECT and memory are discussed.

INTRODUCTION

Since its introduction in 1938, electroconvulsive therapy (ECT, EST) has proved to be an effective treatment for some psychiatric disorders (Huston and Lochar, 1948a, 1948b; Davis, 1965; Medical Research Council, 1965; Wechsler et al., 1965; Kalinowsky, 1967; Pitts, 1972; Hurwitz, 1974; Royal College of Psychiatrists, 1977; Greenblatt, 1977; Avery and Winokur, 1977; Barton, 1977; Turek and Hanlon, 1977; Fink, 1978, 1979), and is considered by many psychiatrists to be the treatment of choice for severe or psychotic depression (Pitts, 1972; Bordern, 1965; Dornbush, 1972; Glassman et al., 1975). Not only is ECT ef-

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effective but it is also quite safe, with mortality rates consistently reported to be less than 0.1% (Pitts, 1972; Alexander, 1956; Matthew and Constable, 1964; Davis and Baker, 1959; Arneson and Butler, 1960), and thus much less than the mortality from depression itself (Guze and Robins, 1970). However, it is not without important side effects and variable degrees of memory dysfunction frequently follow its administration. Memory disturbances were thought to be largely confined to the treatment period and to disappear by 1 month after treatment (Kalinowsky, 1967; Cronholm and Molander, 1964; Korin et al., 1956). However, since the early 1940s, shortly after its introduction, there were reports of prolonged memory defects after ECT, sometimes lasting for months or even years (Levy et al., 1942; Brody, 1944; Medlicott, 1948; Norman and Shea, 1946). Some authors have suggested that bilateral ECT may cause permanent brain damage, especially in those receiving relatively large numbers of treatments (Norman and Shea, 1946). Unfortunately, most of these early reports were anecdotal, and so the contributions which age, diagnosis, and severity of illness made to these complaints of memory problems could not be assessed.

Because memory dysfunction after ECT was evident so consistently, some early investigators felt that it was the major reason for the therapeutic efficacy of the treatments (Tyler and Lowenback, 1947). Since then, however, others have shown that memory dysfunction does not correlate with the alleviation of depressive symptoms (Ottoson, 1968; Fink, 1974; Korin et al., 1956; APA, 1978). Unilateral ECT, introduced in 1957 to help minimize the memory impairment found so commonly with bilateral treatments, has been shown to be an effective treatment modality (Lancaster et al., 1958; Halliday et al., 1968; Strain et al., 1968; Costello et al., 1970; Bidder et al., 1970; d’Elia and Raotma, 1975; Inglis, 1969; Davis, 1978) that does not produce the degree of memory loss found with the standard treatments (Halliday et al., 1968; Strain et al., 1968; Costello et al., 1970; Bidder et al., 1970; d’Elia and Raotma, 1975). Unilateral ECT applied to the nondominant hemisphere does however affect nondominant hemispheric functions, such as memories for visual and nonverbal material (Halliday et al., 1968; Inglis, 1969, 1970).

Ever since its introduction, ECT has been controversial. Terms such as barbaric and archaic are used to describe its continued use. Unfortunately, this is an area where well-reasoned and critical thinking have not always been evident. One author (Friedberg, 1975, 1976, 1977) suggests that the use of ECT may violate the physician’s Hippocratic oath not to employ methods which might harm or wrong any patient. Most psychiatrists, however, are not so choleric about this issue: only 2% of a representative sample of American psychiatrists describe themselves as “totally opposed” to the use of ECT (APA, 1978). Two authors, in fact, have suggested that withholding ECT from patients for whom it might be lifesaving constitutes negligence (Barton, 1977; Beresford, 1971). Editorial comments both defending (Practising Psychiatrist, 1965; Furlong, 1972; Barton and
Snaith, 1974; d'Agostino, 1975; Fink, 1976, 1977; Andren, 1976; Editorial, 1977) and condemning (Friedberg, 1975, 1976, 1977; Regenstein et al., 1975; Jones, 1974; Pribram, 1974; Roueche, 1974) the use of ECT have appeared in both the lay press and the medical literature. Quite clearly the issue of permanent brain damage as a possible sequela of ECT is an extremely important one. However, diatribe, polemic, and anecdote must not substitute for careful scientific investigation and critical thinking in the assessment of either the efficacy or the dangers of any particular medical treatment.

Therefore, a review of studies spanning the more than 40 years since the introduction of ECT seems appropriate to determine what is, in fact, the evidence for prolonged memory defects after the administration of ECT. The deficiencies in the methodology of most of the early studies, as well as improvements in the administration of ECT over the years, make comparison of the earlier reports with later ones hazardous. A number of variables are important in a critical analysis of each of these papers. Specifically, the variables of matched controls, blindness, the presentation of “hard” data vs. anecdotal reporting, the length of follow-up, sample size, diagnosis, and test sensitivity, are all important in the consideration of post-ECT memory dysfunction. No study was excluded because of sample size; even single case reports are included in the review. Nor were studies excluded because of varied diagnoses, even though some authors (Luborsky, 1948; Heaton et al., 1978) have demonstrated the need to separate patients of various diagnoses because of their quite different performances on psychological tests. One fact which was important was that the length of follow-up be long enough so as not to confuse the immediate posttreatment organic symptoms with more prolonged defects.

A brief reiteration of important methodological variables in an ideal study of this question seems in order. A matched control group, not treated with ECT, is essential in the assessment of long-term memory defects. This control group should be matched at least for age, sex distribution, diagnosis, and severity of illness. The variable of diagnosis itself may contribute either to complaints of memory defects (Kahn et al., 1975; Friedman, 1964) or to specific impairments of performance on psychological tests (Luborsky, 1948; Heaton et al., 1978; Sternberg and Jarvik, 1976). The variable of age is clearly important in any consideration of memory problems. The need to control for severity of illness seems reasonable since the most severely ill patients may not be able to cooperate with testing at all. The Rosenthal effect (Rosenthal, 1966), or “experimenter bias,” has been known for years to directly affect the outcome of certain specific variables under investigation and therefore to confound the interpretation of certain data. Hence, the need for a double-blind experimental procedure in an ideal study. Ethical considerations, however, make the feasibility of double-blind procedures questionable. The problem of anecdotal reporting or of isolated case reports without specific data is obvious: though these reports may be interesting, pro-
vocative, or even heuristic, they should not be the basis for generalization about
the effects of ECT on memory when other more important variables are not
detailed. The length of follow-up is obviously an important concern. Differences
found at different follow-up intervals have a good deal of hearing on one's inter-
pretation of the results. The obvious organicity present just after the administra-
tion of the treatment should not be confused with the evidence for defects in
memory long after the termination of treatment. Therefore, the term "prolonged"
should not be used to refer to defects of relatively short duration. Test sensitivity
is an extremely important issue. Many of these studies have been criticized for
using tests of only new learning, of only verbal memories, of material meaningless
to patients, of impersonal rather than personal memories, or for failing to incor-
porate delays between learning and reproduction, a measure known to maximize
the chance of finding memory defects (Cronholm and Ottoson, 1961; Squire
and Miller, 1974; Dornbush, 1972; Korin et al., 1956).

Even though very few of the studies employed a design which might be
described as ideal, it seems important to look at all the studies no matter what
their methodological deficiencies. If the evidence regarding memory changes after
ECT is based on isolated case reports or on studies whose methodologies are
unsound, it is important to recognize that fact. Alternately, if the evidence for
prolonged memory defects induced by ECT comes from careful well-designed
studies, that evidence would be more compelling. It is hoped that this is a reason-
ably exhaustive review. However, in a literature review spanning more than 40
years certain pertinent papers may have been omitted. None was omitted by
design.

REVIEW

The papers in the review are summarized in Table 1. This chronological
division is arbitrary and reflects more improvements in methodology than it does
the historical modification of ECT. Certain papers yielded results which are not
easily summarized in the Table; for these particular papers a single asterisk (*)
refers the reader to the pertinent explanation in the text. Studies were considered
"not blind" unless there were specific information otherwise. Prolonged defects
were recorded "yes" if there was any evidence of prolonged memory problems,
even though many or most of the subjects in the study might not have shown
these defects.

Eleven studies from the 1940s are considered (Levy et al., 1942; Brody,
1944; Norman and Shea, 1946; Medlicott, 1948; Tyler and Lowenback, 1947;
Luborsky, 1948; Huston and Strother, 1948; Smith et al., 1943; Perlson, 1945;
Stone, 1947; Rabin, 1948). None of the studies is blind. Seven of the studies
are entirely anecdotal or present minimal data (Medlicott, 1948; Brody, 1944;
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<thead>
<tr>
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<th>Test measures</th>
<th>Blindness</th>
<th>Follow-up interval</th>
<th>Mean no. (range) of treatments</th>
<th>Prolonged defects</th>
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<tbody>
<tr>
<td>Levy et al. (1942)</td>
<td>12</td>
<td>Varied (mostly depressives)</td>
<td>Metrazol-treated patients</td>
<td>EEGs, Mental status exams</td>
<td>Not blind</td>
<td>6 months</td>
<td>6.5 (2-11)</td>
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<td>Smith et al. (1943)</td>
<td>279</td>
<td>Varied</td>
<td>None</td>
<td>No data</td>
<td>Not blind</td>
<td>Up to 12 months</td>
<td>(9-10)</td>
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<td>Brody (1944)</td>
<td>5</td>
<td>Varied</td>
<td>None</td>
<td>No data (patients' subjective complaints)</td>
<td>Not blind</td>
<td>1-2 years</td>
<td>(4-15)</td>
<td>Yes</td>
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<td>Perlson (1945)</td>
<td>1</td>
<td>Dementia praecox paranoid type</td>
<td>None</td>
<td>Various psychological tests</td>
<td>Not blind</td>
<td>3 months</td>
<td>154-ECT 94-Metrazol</td>
<td>No</td>
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<td>Norman and Shea (1946)</td>
<td>266</td>
<td>Varied (mostly schizophrenes)</td>
<td>None</td>
<td>No data</td>
<td>Not blind</td>
<td>(&gt;).3 months?</td>
<td>(8-50)</td>
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<td>Tyler and Lovenbach (1947)</td>
<td>32</td>
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<td>Not blind</td>
<td>1-5 years</td>
<td>11.1 (5-22)</td>
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<td>Stone (1947)</td>
<td>29</td>
<td>Varied (mostly schizophrenes)</td>
<td>None</td>
<td>Wechsler Memory Scale</td>
<td>Not blind</td>
<td>2-3 weeks</td>
<td>17.0 (4-20)</td>
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<td>Huston and Strother (1948)</td>
<td>75</td>
<td>Varied (all with depressive symptoms)</td>
<td>Normals</td>
<td>Babcock, Shipley-Hartford</td>
<td>Not blind</td>
<td>6 months</td>
<td>7.8 (1-18)</td>
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<td>Medlicott (1948)</td>
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<td>None</td>
<td>No data (patients' subjective complaints on a questionnaire)</td>
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<td>&gt; 6 months</td>
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Table I. Continued

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<tr>
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<th>Non-ECT controls</th>
<th>Test measures</th>
<th>Blindness</th>
<th>Follow-up interval</th>
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<th>Prolonged defects</th>
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<td>Varied</td>
<td>None</td>
<td>Wechsler-Bellevue</td>
<td>Not blind</td>
<td>6 months</td>
<td>12</td>
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<td>Goodenough Man-Drawing</td>
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<td>Rabin (1948)</td>
<td>6</td>
<td>Schizophrenics</td>
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<td>Rorschach</td>
<td>Not blind</td>
<td>No data</td>
<td>(110-234)</td>
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<td>Janis (1950)</td>
<td>19</td>
<td>Varied (mostly schizophrenics)</td>
<td>Other psychiatric patients</td>
<td>Personal data inventory$^b$</td>
<td>Not blind</td>
<td>4 weeks</td>
<td>(8-27)</td>
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<td>Worschel and Narcisio (1950)</td>
<td>2</td>
<td>Psychopathic personality</td>
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<td>Nonsense syllables</td>
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<td>9 days</td>
<td>7</td>
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<tr>
<td>Stone (1950)</td>
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<td>Manic</td>
<td>None</td>
<td>Wechsler Memory Scale</td>
<td>Not blind</td>
<td>60 days</td>
<td>20</td>
<td>No</td>
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<tr>
<td></td>
<td></td>
<td>Schizophrenic</td>
<td></td>
<td>Army alpha</td>
<td></td>
<td>3 years</td>
<td>14</td>
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<td>Stepler et al. (1951)</td>
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<td>Paranoid</td>
<td>Normals</td>
<td>Wechsler Memory Scale</td>
<td>Not blind</td>
<td>3 weeks</td>
<td>15</td>
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<td></td>
<td></td>
<td>schizophrenics</td>
<td></td>
<td>CVS abbrev. intelligence scale</td>
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<td>(5-25)</td>
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<td></td>
<td>Personal memory inventory$^b$</td>
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<td>Pascal and Zeaman (1951)</td>
<td>1</td>
<td>Depression</td>
<td>None</td>
<td>Wechsler-Bellevue</td>
<td>Not blind</td>
<td>2 weeks</td>
<td>10</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Rorschach</td>
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<td>7 months</td>
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<td>Janis and Astrachan (1951)</td>
<td>9</td>
<td>Varied (mostly schizophrenics)</td>
<td>Other psychiatric patients</td>
<td>Personal data inventory$^b$</td>
<td>Not blind</td>
<td>4 weeks</td>
<td>(10-30)</td>
<td>Yes</td>
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$^b$ Abbreviations: CVS = abbreviated intelligence scale; Wechsler Memory Scale; Rorschach.
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<th>Study</th>
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<th>Diagnosis</th>
<th>Control Group</th>
<th>Test</th>
<th>Blindness</th>
<th>Duration</th>
<th>Memory Dysfunction</th>
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<td>Michael (1954)</td>
<td>30</td>
<td>Varied</td>
<td>Other psychiatric patients</td>
<td>Noun enumeration test</td>
<td>Not blind</td>
<td>1 week 6 weeks</td>
<td>7.06 (5-13) No</td>
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<td>Hetherington (1956)</td>
<td>30</td>
<td>Depression</td>
<td>Normals</td>
<td>Varied (verbal, nonverbal motor tests)</td>
<td>Not blind</td>
<td>2-3 days 10 days</td>
<td>5 (5-10) No</td>
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<tr>
<td>Korin et al. (1956)</td>
<td>40</td>
<td>Varied (depression-29, schizophrenia-11)</td>
<td>Other psychiatric patients (possible EST candidates)</td>
<td>Common words Nonsense syllables</td>
<td>Not blind</td>
<td>1, 2, and 3 weeks after ECT</td>
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<td>Miura et al. (1960)</td>
<td>1</td>
<td>Reactive confusional state</td>
<td>None</td>
<td>No data (patient’s subjective complaints)</td>
<td>Not blind</td>
<td>No data</td>
<td>11 Yes</td>
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<td>Cronholm and Molander (1964)</td>
<td>28</td>
<td>Varied (mostly depressives)</td>
<td>None</td>
<td>Word pairs Figures Personal data sheet</td>
<td>Not blind</td>
<td>27-52 days</td>
<td>5.3 (2-12) No</td>
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<td>Varied (many schizophrenic or borderline)</td>
<td>Other psychiatric patients</td>
<td>Wechsler Memory Scale</td>
<td>Not blind</td>
<td>No data</td>
<td>No No</td>
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<td>Halliday et al. (1968)</td>
<td>52</td>
<td>Endogenous depression</td>
<td>None (bilateral vs. unilateral)</td>
<td>Verbal &amp; nonverbal immediate &amp; delayed (see Williams, 1968) Digit span Remote memory</td>
<td>Double-blind</td>
<td>3 months</td>
<td>6.2 left unilateral 5.9 right unilateral 5.5 bilateral Yes</td>
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<tr>
<td>Strain et al. (1968)</td>
<td>96</td>
<td>Depression</td>
<td>None (bilateral vs. unilateral)</td>
<td>Paired associates Revised Benton visual retention test Personal data sheet</td>
<td>Double-blind</td>
<td>10 days</td>
<td>8.4 unilateral 7.5 bilateral No</td>
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<tr>
<td>Authors</td>
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<td>Diagnoses</td>
<td>Non-ECT controls</td>
<td>Test measures</td>
<td>Blindness</td>
<td>Follow-up interval</td>
<td>Mean no. (range) of treatments</td>
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<td>Bidder et al. (1970)</td>
<td>96</td>
<td>Depression</td>
<td>None (bilateral vs. unilateral)</td>
<td>Paired associates Revised Benton Personal data sheet&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Double-blind</td>
<td>30 days 1 year</td>
<td>7.5 bilateral (4-12)</td>
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<td>Cronin et al. (1970)</td>
<td>45</td>
<td>Depression (endogenous and reactive)</td>
<td>None (bilateral vs. unilateral)</td>
<td>Graham-Kendall Benton visual retention Modified word learning Digit span Wechsler Memory Scale</td>
<td>Blind</td>
<td>4-6 weeks</td>
<td>8.0</td>
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<tr>
<td>Miller (1970)</td>
<td>20</td>
<td>No data</td>
<td>Other psychiatric patients</td>
<td>Paired associates</td>
<td>Not blind</td>
<td>3-6 days 7-14 days</td>
<td>6.0</td>
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<tr>
<td>Brunswig et al. (1971)</td>
<td>96</td>
<td>Depression</td>
<td>None</td>
<td>Paired associates Revised Benton Personal data sheet&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Double-blind</td>
<td>10 days 1 year</td>
<td>7.8 bilateral (4-12)</td>
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<td>Goldman et al. (1972)</td>
<td>20</td>
<td>Chronic schizophrenia</td>
<td>Chronic schizophrenia</td>
<td>Bender-Gestalt Benton Revised Benton WAIS</td>
<td>Not blind</td>
<td>10-15 years</td>
<td>69.5 (50-219)</td>
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<td>Templer et al. (1973)</td>
<td>22</td>
<td>Chronic schizophrenia</td>
<td>Chronic schizophrenia</td>
<td>Bender-Gestalt Revised Benton WAIS MMPI</td>
<td>Double-blind</td>
<td>&gt; 7 years</td>
<td>58.5 (40-263)</td>
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<td>Small (1974)</td>
<td>50</td>
<td>Varied (mostly schizophrenics)</td>
<td>50 Fluorohally treated patients</td>
<td>Wechsler Memory Scale Shinsky-Hartford MMPI Other psychological tests</td>
<td>Double-blind</td>
<td>After 5th ECT After end of ECT 60-90 days</td>
<td>(&lt;10-&gt;30)</td>
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</tbody>
</table>

<sup>a</sup> Some data were not obtained until the last review date.
<sup>b</sup> Personal data sheet.
<table>
<thead>
<tr>
<th>Study</th>
<th>Sample Description</th>
<th>Test/Procedure Details</th>
<th>Control/Blinding</th>
<th>Duration</th>
<th>Mean(s.d.)</th>
<th>Results</th>
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<td>Squire et al. (1975)</td>
<td>Depression</td>
<td>Television program recognition(^b)</td>
<td>Not blind</td>
<td>1-2 weeks</td>
<td>7.6 (5-13)</td>
<td>No</td>
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<td>Squire and Chace (1975)</td>
<td>Varied (all with depressive symptoms)</td>
<td>Paired associates Television program recognition(^b) 32-item recognition Paragraph recall Complex figure drawing WAIS</td>
<td>Not blind</td>
<td>6-9 months</td>
<td>10.1 (5-17)</td>
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<td>Jackson (1978)</td>
<td>No data</td>
<td>Wechsler Memory Scale Verbal &amp; nonverbal memory</td>
<td>Double-blind</td>
<td>10 days</td>
<td>6.0</td>
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<td>Freeman et al. (1980)</td>
<td>Varied</td>
<td>Personal remote memory Logical memory Famous personalities Verbal memory Face-name connection Decision time Movement time</td>
<td>Not blind</td>
<td>9 months-30 years</td>
<td>No data</td>
<td>Yes(^a)</td>
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<tr>
<td>Weeks et al. (1980)</td>
<td>Depression</td>
<td>Famous personalities Delayed recall recognition Verbal memory Decision time Personal remote memory Movement time Fluid movement Visual memory Visual design Anomalous sentence repetition</td>
<td>Blind</td>
<td>4 months</td>
<td>7.2 (2-20)</td>
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</thead>
<tbody>
<tr>
<td>Squire et al.</td>
<td>43</td>
<td>Varied (42 with depression)</td>
<td>Other inpatients (n = 7)</td>
<td>Public events: recognition/recall, television program recall, personal events recall</td>
<td>Not blind</td>
<td>1 week, 7 months</td>
<td>10.2 (5-21)</td>
<td>Yesb</td>
</tr>
</tbody>
</table>

*See text for explanation.
*bConstructed by the authors.
Anj Mcmory Dysfunction. Two of the papers are case reports. Three studies employed specific treatments specifically tested before and after ECT (Levy et al., 1942; Luborsky, 1948; Huston and Strother, 1948). Luborsky's (1948) follow-up is only 5 or 6 years after the completion of a series of 12 treatments; in the other two, the follow-up is at 6 months. One study used ECT techniques which were specifically designed to produce confusion (Tyler and Lowenback, 1947). Four of the studies suggest prolonged defects (Levy et al., 1942; Brody, 1944; Norman and Shea, 1946; Medlicott, 1948); six do not (Tyler and Lowenback, 1947; Luborsky, 1948; Huston and Strother, 1948; Smith et al., 1943; Perlson, 1945; Stone, 1947). Rabin's (1948) study is ambiguous. He investigated patients who had received more than 100 ESTs. He suggested that "more than 100 ESTs do not produce a characteristic organic personality pattern in all patients to whom the therapy is administered." He cautions the reader to consider "the individual reaction of the organism to treatment as opposed to the blanket statements, frequently improperly founded, regarding the inevitably and universally damaging effects of EST." And yet three of the six patients studied showed evidence of prolonged defects, while the other three apparently did not.

With the muddle of methodological inconsistencies and apparently contradictory results, one might be tempted to dismiss most of the studies from the 1940s out of hand. However, three of the studies deserve further comment.

Levy et al.'s (1942) study reports on 23 patients, 11 treated with Metrazol and 12 treated with ECT. Posttreatment analyses revealed disordered EEG findings in 55% of the total group and impaired intellectual functioning in 45%. Most of the EEG abnormalities and impairments in intellectual functioning returned to pretreatment levels within a few weeks. There was evidence, however, of impaired intellectual functioning in some of the patients at a 6-month follow-up. Unfortunately, the exact number is not specified. The authors noted that the EEG changes were "largely reversible." They state that "disturbances in cerebral functions, however, which manifest themselves as impaired intellectual functioning are unquestionably indicative of disordered cortical activity." Because the ECT-treated patients had more abnormalities the authors conclude "the electric shock produces more severe damage to the brain than Metrazol shock..." To suggest that "disturbances in cerebral function" are indicative of a "disordered cortical activity" is certainly true. But this is not necessarily evidence of cerebral "damage." The only decisive conclusion that can be drawn from this study is that there is a functional change in the central nervous system. EEG abnormalities or impaired intellectual functioning may come from a variety of sources including toxic, metabolic, infectious, vascular, or psychiatric, and may, in fact, represent truly reversible abnormalities. The authors state that the majority of their patients return to pretreatment levels in the follow-up period, a fact which suggests a reversible defect, and therefore argues against a structural change.
Abnormal findings at 6 months may well be regarded as prolonged however. The discussion at the end of that article indicates clearly why blindness is an important variable in scientific research. One of the authors argues against the mechanistic approach in psychiatry and says that “careful studies” revealed a “definite ‘organic’ change in memory which does not entirely clear up.” Unfortunately he does not cite these studies and gives the reader no evidence on which to base this conclusion.

Brody (1944) reports on five patients followed approximately 1 to 2 years after a course of ECT. This is a series of case reports with no indication of pretreatment status, no control group, and no specific data. These examples are convincing enough that some patients do, for a long period of time, complain of defects in their memory after a course of ECT. A careful look at the case reports however confounds the issue somewhat. One case is described as an “hysterical type,” and attempts to accurately assess memory defects in patients, one of whose major symptoms may be amnesia, is clearly difficult at best. Another case is a patient with “mild” arteriosclerosis and hypochondriasis. Since her arteriosclerosis is “mild,” he suggests that it may be ignored as an etiological factor in her memory dysfunction. The problems with case reports are clear enough. But in an area where the assessment of long-term effects is confounded by so many variables such as patients’ attitude toward and expectations of the treatment, the subjective nature of the complaints, the lack of controls, and the lack of blindness on the part of the examiner, accurate assessment of these complaints is impossible. When the etiology of these patients’ memory complaints is this difficult to answer, it seems risky to suggest, as the author does, that ECT causes “permanent or semi-permanent” damage to the brain when it is based on five case reports.

Huston and Strother’s (1948) paper reports on 75 patients of mixed diagnoses, all with affective symptoms. Older patients (median age 47) were age-matched with normal controls because of the effect of age on one of the testing instruments. On the first posttreatment follow-up, 11 days after the termination of ECT, there was clear evidence of impairment in mental efficiency measured by the Babcock Revision and the Shipley-Hartford Test. The authors maintain that with the Babcock “mental efficiency as measured by this test primarily involves memory and secondarily, attention and speed.” For those tested at the 6-month follow-up, mental efficiency scores were significantly improved (p < 0.01) over posttreatment scores. Age-matched subjects were not significantly different from controls who had not undergone ECT.

The Levy et al. (1942) and Brody (1944) papers are frequently quoted as definitive evidence for prolonged memory defects. Huston and Strother’s (1945) data, however, constitute the only well-controlled evidence from this era with a reasonably long-term follow-up and the data suggest return to pretreatment levels of memory function.
The 1950s brought some more rigorously designed studies and some interesting developments. Nine studies are reviewed from that decade (Janis, 1950; Worchel and Narcisco, 1950; Stone, 1950; Stieper et al., 1951; Pascal and Zeaman, 1951; Janis and Astrachan, 1951; Michael, 1954; Hetherington, 1956; Korin et al., 1956). None of the studies is blind. Three employ no control group (Worchel and Narcisco, 1950; Stone, 1950; Pascal and Zeaman, 1951), and three report on either one (Pascal and Zeaman, 1951) or only two patients (Worchel and Narcisco, 1950; Stone, 1950). Four of the six studies that are not case reports include patients with a variety of diagnoses. Three indicate prolonged defects (Janis, 1950; Stieper et al., 1951; Janis and Astrachan, 1951). Six show a return to pretreatment levels of mental efficiency (Worchel and Narcisco, 1950; Stone, 1950; Pascal and Zeaman, 1951; Michael, 1954; Hetherington, 1956; Korin et al., 1956).

Janis' study (1950) is frequently quoted in the literature on prolonged defects after ECT. Because it employed a control group and specifically tested personal rather than impersonal memories, the study has become a prominent one in the ECT literature. Some critics suggest that tests which measure impersonal memories, such as paired associates or learning of nonsense syllables, may not be sensitive indicators of post-ECT amnesias. The specific purpose of Janis' study was to investigate whether the short-term memory defects after ECT, so clearly evident on clinical grounds, persisted beyond the few weeks after treatment. Janis points out that "isolated case reports provide extremely inadequate evidence for the hypothesis that ECT produces sustained memory defect." He studied 19 patients who received between 8 and 27 standard bilateral ECTs with a mean of 17. Both patients and comparably ill controls who did not receive ECT were given an intensive pretreatment interview which included school, job, and psychiatric history, sexual, marital, and family relationships, childhood experiences, and other major life events. Approximately 4 weeks after the ECT there was evidence of retrograde amnesia in all 19 of the patients studied. These defects were described as "negligible" in the control patients who were tested after a similar time interval. In an attempt to determine the duration of these defects, Janis followed five patients for approximately 3 months more. There were minor improvements but a substantial defect was still present at the second follow-up. Because of the problems of motivation and the fact that some patients' recall was improved when extra time was given to "work on" recovering these memories, Janis concluded the following: (i) "it seems to be fairly probable that the post-ECT recall failures are especially likely to involve experiences which tend to arouse anxiety, guilt and a lowering of self-esteem"; (ii) "If it is true that the memory gaps found after ECT are largely determined by motivational factors, it would follow that the amnesias do not represent irreversible organic defect"; and (iii) "The view which emerges is that ECT amnesias involve a reversible retention loss." One may well argue about the verifiability of psychodynamic formulations of motivation and its relationship to memory defect.
Studies to document the effects of motivation on memory would be helpful in order to correctly interpret Janis' data. Later authors do not support Janis' conclusion that memory loss is important to the therapeutic process of ECT. This study is an interesting one and it is widely quoted. It involves controls, testing before and at two intervals after ECT, personal rather than impersonal memories, and follow-up intervals where the obvious clinical posttreatment organic symptoms do not confound the issue of whether the memory defects are prolonged. This study, though widely quoted, has several weaknesses. The patients studied had a variety of diagnoses, but most were schizophrenic. The effects of this variable in the sample population are uncertain. Janis qualifies his data as documentary rather than quantitative in character. The presentation of more standardized objective data with estimates of interrater reliability would have been more compelling. He followed only five patients of the original group for the second follow-up, and no selection criteria or clinical characteristics of these five are reported. The possibility exists that these five continued to be more symptomatic than the rest of the group and were therefore more available for interview. He suggests that one piece of evidence that ECT specifically caused these defects was that there was a fair degree of heterogeneity with respect to the additional forms of psychiatric treatment received by the ECT group, and yet all demonstrated memory loss. However, three of the patients received insulin-shock treatments during the test-retest interval which introduces another important factor. One patient received as many as 50 insulin coma treatments. Whether these three patients were included in the five studied at the second follow-up is uncertain. Janis does not comment on this. Until such confounding factors are removed it seems somewhat risky to regard ECT as causative. Despite the variables cited above, this study is widely quoted in the ECT literature as definitive evidence of prolonged memory defects. A second study by Janis (Janis and Astrachan, 1951) demonstrates the prolonged memory defects in personal, but this time "routine," memory items presumably minimizing the anxiety-provoking and motivational aspects of the earlier study. Again these defects were demonstrable approximately 4 weeks after treatment. Janis suggests that these "artificially induced repressions" helped to contribute to the reduction of affective disturbances and therefore, presumably, are important in the therapeutic aspects of the treatment. Again, later authors do not support this interpretation.

Stieper et al.'s (1951) paper deserves separate comment. It involved pre-and post-treatment testing, a control group, both personal and impersonal memory items, and a 3-week follow-up. He found that general mental efficiency as measured by the CVS abbreviated intelligence scale was improved beyond the pretreatment level 3 weeks after ECT. He corroborated Janis' data that personal memories were more affected by ECT than were impersonal memories. Unlike Janis, though, he found that patients whose clinical status at follow-up was better, suffered less from amnesia, even for personal events. In addition his study
patients were chronic schizophrenics, a factor which is known to influence performance on psychological tests (Luborsky, 1948; Heaton et al., 1978).

Fewer studies were found in the 1960s which detailed prolonged memory defects after ECT. One might speculate that research interest in ECT declined somewhat after the introduction of the antidepressant medications. Five studies from the 1960s are reviewed (Miura et al., 1960; Cronholm and Molander, 1964; Schwartzman and Termansen, 1967; Halliday et al., 1968; Strain et al., 1968). One is a single case report (Miura et al., 1960). Two studies are not blind (Cronholm and Molander, 1964; Schwartzman and Termansen, 1967); the other two are double-blind (Halliday et al., 1968; Strain et al., 1968). Only one has a control group of patients not treated with ECT (Schwartzman and Termansen, 1967). The other three either compare bilateral with unilateral electrode placement (Halliday et al., 1968; Strain et al., 1968), or use the patients as their own controls (Cronholm and Molander, 1964). Three of these studies found no objective evidence for prolonged defects (Cronholm and Molander, 1964; Strain et al., 1968; Schwartzman and Termansen, 1967). The other two studies (Miura et al., 1960; Halliday et al., 1968) suggest such defects. Despite the lack of evidence for prolonged defects in Schwartzman and Termansen’s (1967) paper 27 of the original 79 patients in this sample answered a questionnaire and complained of persistent memory defects. Patients’ complaints of memory defects which are at variance with their performance on memory tests, especially when patients received bilateral ECT, will be seen to be a characteristic also found by later authors.

The study by Halliday et al. (1968) demonstrated memory defects 3 months after ECT. There was random assignment to three treatment groups: bilateral, right, and left unilateral ECT. There were specified criteria for the diagnosis of endogenous depression and other inclusion criteria included age under 65 years, no evidence of organic brain damage, no prolonged duration of depressive illness, and no ECT in the previous 3 months. The authors measured both verbal and nonverbal memories using a special test battery devised by Williams (1968). They also employed a delay of approximately ½ hr in the interval between learning and recall. Such a delay is known to maximize the chances of finding impairment in memory (Cronholm and Ottoson, 1961; Cronholm and Molander, 1964; Dornbush, 1972; Squire and Miller, 1974). They studied 52 patients before and after a series of four ECTs and again at the end of the series. For those who received more than four ECTs the mean number of extra ECTs was 3.6, 3.9, and 4.8, for the bilateral, right unilateral, and left unilateral treatment groups, respectively. Of the 52 original patients 44 were retested 3 months after the termination of ECT; of the 8 who were not retested, 3 had been switched to a different treatment modality, 1 had committed suicide, and 4 were lost to follow-up. They demonstrated the evidence for the differential effect of right vs. left hemispheric stimulation on nonverbal and verbal memories, respectively. After 3 months there was definite improvement in all of the test scores, but some residual defects per-
sisted. Nonverbal learning, originally worst in the right unilateral group, was now worst in the bilaterally treated group. The authors state that the study was conducted double-blind. The main point of the article was to determine the differential effects of dominant vs. nondominant hemispheric stimulation, rather than the persistence of memory defects per se. Though one cannot be sure that this necessarily protects against experimenter bias, it does seem more compelling. A longer follow-up is essential to determine how long these posttreatment anamnesis persist.

Cronholm and Molander's study (1964) is one in a series of studies which investigated the acute and long-term effects of ECT on memory (Cronholm and Molander, 1957, 1964; Cronholm and Blomquist, 1959; Ottoson, 1959; Cronholm and Ottonson, 1961, 1963). Their patients were studied before and at varying intervals after a course of ECT. Three different memory tests including words, figures, and personal data were employed. In summary, these studies showed that depression itself affects learning, while ECT affects retention of newly learned material. They found a decline in memory production just after the second ECT (Cronholm and Molander, 1975), and a pronounced decline when there was a delay of 3 hr between learning and reproduction. However, the increase in “forgetting scores,” the differences between immediate and delayed reproduction scores, was not found after 1 month (Cronholm and Molander, 1964).

The report by Strain et al. (1968) is one in a series of three papers (Strain et al., 1968; Bidder et al., 1970; Brunschwig et al., 1971), which report on a sample of 96 patients studied at various intervals before and after ECT. The Strain et al. data suggest that 10 days after the last ECT there are no significant differences between patients treated with bilateral and nondominant unilateral ECT on any of the tests they used. The worst defect was with recent memories on their Personal Data Sheet, corroborating Janis' findings.

Eleven studies from the 1970s are reviewed (Bidder et al., 1970; Cronin et al., 1970; Miller, 1970; Brunschwig et al., 1971; Goldman et al., 1972; Templer et al., 1973; Small, 1974; Regenstein et al., 1975; Squire et al., 1975; Squire and Chase, 1975; Jackson, 1978). Five studies are not blind (Miller, 1970; Goldman et al., 1972; Regenstein et al., 1975; Squire et al., 1975; Squire and Chase, 1975). the remainder are reportedly blind (Bidder et al., 1970; Brunschwig et al., 1971; Templer et al., 1973; Small, 1974; Jackson, 1978). Only one is a single case report (Regenstein et al., 1975). Seven do not suggest prolonged memory defects after ECT (Bidder et al., 1979; Miller, 1970; Brunschwig et al., 1971; Small, 1974; Squire et al., 1975; Squire and Chase, 1975; Jackson, 1978), though Small's study deserves separate comment. Four of the studies do report these defects (Cronin et al., 1970; Goldman et al., 1972; Templer et al., 1973; Regenstein et al., 1975), although Templer's data are puzzling and are discussed in more detail below.
Review

Electroconvulsive Therapy and Memory Dysfunction: Is There Evidence for Prolonged Defects?

John R. Taylor,1,4 Rachel Tompkins,2 Renee Demers,3 and Dale Anderson1

Received April 1, 1982

The authors reviewed 39 papers which concern the long-term effects of electroconvulsive therapy (ECT) on human memory. Although the authors caution that methodological considerations preclude a decisive assessment, the majority of the studies suggest that ECT does not normally produce prolonged memory defects. Some recent studies do document subtle but persistent defects several months after ECT, especially in personal autobiographical material. These defects appear to be more annoying than seriously incapacitating. Variables considered important in an ideal design of studies on ECT and memory are discussed.

INTRODUCTION

Since its introduction in 1938, electroconvulsive therapy (ECT, EST) has proved to be an effective treatment for some psychiatric disorders (Huston and Locher, 1948a, 1948b; Davis, 1965; Medical Research Council, 1965; Wechsler et al., 1965; Kalinowsky, 1967; Pitts, 1972; Hurwitz, 1974; Royal College of Psychiatrists, 1977; Greenblatt, 1977; Avery and Winokur, 1977; Barton, 1977; Turek and Hanlon, 1977; Fink, 1978, 1979), and is considered by many psychiatrists to be the treatment of choice for severe or psychotic depression (Pitts, 1972; Hordern, 1965; Dombush, 1972; Glassman et al., 1975). Not only is ECT ef-

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ffective but it is also quite safe, with mortality rates consistently reported as less than 0.1% (Pitts, 1972; Alexander, 1956; Matthew and Constan, 1964; Peck and Baker, 1959; Arneson and Butler, 1960), and thus much less than the mortality from depression itself (Guze and Robins, 1970). However, it is not without important side effects and variable degrees of memory dysfunction frequently follow its administration. Memory disturbances were thought to be largely confined to the treatment period and to disappear by 1 month after treatment (Kalinowsky, 1967; Cronholm and Molander, 1964; Korin et al., 1956). However, since the early 1940s, shortly after its introduction, there were reports of prolonged memory defects after ECT, sometimes lasting for months or even years (Levy et al., 1942; Brody, 1944; Medlicott, 1948; Norman and Shea, 1946). Some authors have suggested that bilateral ECT may cause permanent brain damage, especially in those receiving relatively large numbers of treatments (Norman and Shea, 1946). Unfortunately, most of these early reports were anecdotal, and so the contributions which age, diagnosis, and severity of illness made to these complaints of memory problems could not be assessed.

Because memory dysfunction after ECT was evident so consistently, some early investigators felt that it was the major reason for the therapeutic efficacy of the treatments (Tyler and Lowenback, 1947). Since then, however, others have shown that memory dysfunction does not correlate with the alleviation of depressive symptoms (Ottoson, 1968; Fink, 1974; Korin et al., 1956; APA, 1978). Unilateral ECT, introduced in 1957 to help minimize the memory impairment found so commonly with bilateral treatments, has been shown to be an effective treatment modality (Lancaster et al., 1958; Halliday et al., 1968; Strain et al., 1968; Costello et al., 1970; Bidder et al., 1970; d'Elia and Raotma, 1975; Inglis, 1969; Davis, 1978) that does not produce the degree of memory loss found with the standard treatments (Halliday et al., 1968; Strain et al., 1968; Costello et al., 1970; Bidder et al., 1970; d'Elia and Raotma, 1975). Unilateral ECT applied to the nondominant hemisphere does however affect nondominant hemispheric functions, such as memories for visual and nonverbal material (Halliday et al., 1968; Inglis, 1969, 1970).

Ever since its introduction, ECT has been controversial. Terms such as barbaric and archaic are used to describe its continued use. Unfortunately, this is an area where well-reasoned and critical thinking have not always been evident. One author (Friedberg, 1975, 1976, 1977) suggests that the use of ECT may violate the physician's Hippocratic oath not to employ methods which might harm or wrong any patient. Most psychiatrists, however, are not so choleric about this issue: only 2% of a representative sample of American psychiatrists describe themselves as "totally opposed" to the use of ECT (APA, 1978). Two authors, in fact, have suggested that withholding ECT from patients for whom it might be lifesaving constitutes negligence (Barton, 1977; Beresford, 1971). Editorial comments both, defending (Practising Psychiatrist, 1965; Furlong, 1972; Barton and...
Snaith, 1974; d'Agostino, 1975; Fink, 1976, 1977; Andren, 1976; Editorial, 1977 and condemning (Friedberg, 1975, 1976, 1977; Regenstein et al., 1975; Jones, 1974; Pribram, 1974; Roueche, 1974) the use of ECT have appeared in both the lay press and the medical literature. Quite clearly the issue of permanent brain damage as a possible sequela of ECT is an extremely important one. However, diatribe, polemic, and anecdote must not substitute for careful scientific investigation and critical thinking in the assessment of either the efficacy or the dangers of any particular medical treatment.

Therefore, a review of studies spanning the more than 40 years since the introduction of ECT seems appropriate to determine what is, in fact, the evidence for prolonged memory defects after the administration of ECT. The deficiencies in the methodology of most of the early studies, as well as improvements in the administration of ECT over the years, make comparison of the earlier reports with later ones hazardous. A number of variables are important in a critical analysis of each of these papers. Specifically, the variables of matched controls, blindness, the presentation of "hard" data vs. anecdotal reporting, the length of follow-up, sample size, diagnosis, and test sensitivity, are all important in the consideration of post-ECT memory dysfunction. No study was excluded because of sample size; even single case reports are included in the review. Nor were studies excluded because of varied diagnoses, even though some authors (Luborsky, 1948; Heaton et al., 1978) have demonstrated the need to separate patients of various diagnoses because of their quite different performances on psychological tests. One fact which was important was that the length of follow-up be long enough so as not to confuse the immediate posttreatment organic symptoms with more prolonged defects.

A brief reiteration of important methodological variables in an ideal study of this question seems in order. A matched control group, not treated with ECT, is essential in the assessment of long-term memory defects. This control group should be matched at least for age, sex distribution, diagnosis, and severity of illness. The variable of diagnosis itself may contribute either to complaints of memory defects (Kahn et al., 1975; Friedman, 1964) or to specific impairments of performance on psychological tests (Luborsky, 1948; Heaton et al., 1978; Sternberg and Jarvik, 1976). The variable of age is clearly important in any consideration of memory problems. The need to control for severity of illness seems reasonable since the most severely ill patients may not be able to cooperate with testing at all. The Rosenthal effect (Rosenthal, 1966), or "experimenter bias," has been known for years to directly affect the outcome of certain specific variables under investigation and therefore to confound the interpretation of certain data. Hence, the need for a double-blind experimental procedure in an ideal study. Ethical considerations, however, make the feasibility of double-blind procedures questionable. The problem of anecdotal reporting or of isolated case reports without specific data is obvious: though these reports may be interesting, pro-
vocative, or even heuristic, they should not be the basis for generalizations about
the effects of ECT on memory when other more important variables are not
detailed. The length of follow-up is obviously an important concern. Differences
found at different follow-up intervals have a good deal of bearing on one's inter-
pretation of the results. The obvious organicity present just after the administra-
tion of the treatment should not be confused with the evidence for defects in
memory long after the termination of treatment. Therefore, the term "prolonged"
should not be used to refer to defects of relatively short duration. Test sensitivity
is an extremely important issue. Many of these studies have been criticized for
using tests of only new learning, of only verbal memories, of material meaningless
to patients, of impersonal rather than personal memories, or for failing to incor-
porate delays between learning and reproduction, a measure known to maximize
the chance of finding memory defects (Cronholm and Ottoson, 1961; Squire
and Miller, 1974; Dornbush, 1972; Korin et al., 1956).

Even though very few of the studies employed a design which might be
described as ideal, it seems important to look at all the studies no matter what
their methodological deficiencies. If the evidence regarding memory changes after
ECT is based on isolated case reports or on studies whose methodologies are
unsound, it is important to recognize that fact. Alternateely, if the evidence for
prolonged memory defects induced by ECT comes from careful well-designed
studies, that evidence would be more compelling. It is hoped that this is a reason-
ably exhaustive review. However, in a literature review spanning more than 40
years certain pertinent papers may have been omitted. None was omitted by
design.

**REVIEW**

The papers in the review are summarized in Table 1. This chronological
division is arbitrary and reflects more improvements in methodology than it does
the historical modification of ECT. Certain papers yielded results which are not
easily summarized in the Table; for these particular papers a single asterisk (*)
refers the reader to the pertinent explanation in the text. Studies were considered
"not blind" unless there were specific information otherwise. Prolonged defects
were recorded "yes" if there was any evidence of prolonged memory problems,
even though many or most of the subjects in the study might not have shown
these defects.

Eleven studies from the 1940s are considered (Levy et al., 1942; Brody,
1944; Norman and Shea, 1946; Medlicott, 1948; Tyler and Lowenback, 1947;
Luborsky, 1948; Huston and Strother, 1948; Smith et al., 1943; Perlson, 1945;
Stone, 1947; Rabin, 1948). None of the studies is blind. Seven of the studies
are entirely anecdotal or present minimal data (Medlicott, 1948; Brody, 1944;
<table>
<thead>
<tr>
<th>Authors</th>
<th>n</th>
<th>Diagnoses</th>
<th>Non-ECT controls</th>
<th>Test measures</th>
<th>Blindness</th>
<th>Follow-up interval</th>
<th>Mean no. (range) of treatments</th>
<th>Prolonged defects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Levy et al. (1942)</td>
<td>12</td>
<td>Varied (mostly depressives)</td>
<td>Metrazol-treated patients</td>
<td>LEGs Mental status exams</td>
<td>Not blind</td>
<td>6 months</td>
<td>6.5 (2-11)</td>
<td>Yes</td>
</tr>
<tr>
<td>Smith et al. (1943)</td>
<td>279</td>
<td>Varied</td>
<td>None</td>
<td>No data</td>
<td>Not blind</td>
<td>Up to 12 months</td>
<td>(9-10)</td>
<td>No</td>
</tr>
<tr>
<td>Brody (1944)</td>
<td>5</td>
<td>Varied</td>
<td>None</td>
<td>No data (patients' subjective complaints)</td>
<td>Not blind</td>
<td>1-2 years</td>
<td>(4-15)</td>
<td>Yes</td>
</tr>
<tr>
<td>Perlson (1945)</td>
<td>1</td>
<td>Dementia praecox paranoid type</td>
<td>None</td>
<td>Various psychological tests</td>
<td>Not blind</td>
<td>3 months</td>
<td>154-ECT 94-Metrazol</td>
<td>No</td>
</tr>
<tr>
<td>Norman and Shea (1946)</td>
<td>266</td>
<td>Varied (mostly schizophrenics)</td>
<td>None</td>
<td>No data</td>
<td>Not blind</td>
<td>(&gt; 3 months?)</td>
<td>(8-50)</td>
<td>Yes</td>
</tr>
<tr>
<td>Tyler and Lowenbach (1947)</td>
<td>32</td>
<td>Varied (mostly schizophrenics)</td>
<td>None</td>
<td>No data</td>
<td>Not blind</td>
<td>1-5 years</td>
<td>11.1 (5-22)</td>
<td>No</td>
</tr>
<tr>
<td>Stone (1947)</td>
<td>29</td>
<td>Varied (mostly schizophrenics)</td>
<td>None</td>
<td>Wechsler Memory Scale</td>
<td>Not blind</td>
<td>2-3 weeks</td>
<td>17.0 (4-20)</td>
<td>No</td>
</tr>
<tr>
<td>Huston and Strother (1948)</td>
<td>75</td>
<td>Varied (all with depressive symptoms)</td>
<td>Normals</td>
<td>Babcock Shipley-Hartford</td>
<td>Not blind</td>
<td>6 months</td>
<td>7.8 (1-18)</td>
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<tr>
<td>Medlicott (1948)</td>
<td>100</td>
<td>Varied</td>
<td>None</td>
<td>No data (patients' subjective complaints on a questionnaire)</td>
<td>Not blind</td>
<td>&gt; 6 months</td>
<td>No data</td>
<td>Yes</td>
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<tr>
<td>Authors</td>
<td>n</td>
<td>Diagnoses</td>
<td>Non-ECT controls</td>
<td>Test measures</td>
<td>Blindness</td>
<td>Follow-up interval</td>
<td>Mean no. (range) of treatments</td>
<td>Prolonged defects</td>
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<tr>
<td>Luborsky (1948)</td>
<td>12</td>
<td>Varied</td>
<td>None</td>
<td>Wechsler-Bellevue</td>
<td>Not blind</td>
<td>6 months</td>
<td>12</td>
<td>No</td>
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<td>Rabkin (1948)</td>
<td>6</td>
<td>Schizophrenics</td>
<td>None</td>
<td>Stanford-Binet</td>
<td>Not blind</td>
<td>No data</td>
<td>(110-234)</td>
<td>24</td>
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<tr>
<td>Janis (1950)</td>
<td>19</td>
<td>Varied (mostly schizophrenics)</td>
<td>Other psychiatric patients</td>
<td>Rorschach</td>
<td>Not blind</td>
<td>4 weeks</td>
<td>17</td>
<td>Yes</td>
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<tr>
<td>Worschel and Narcisco (1950)</td>
<td>2</td>
<td>Psychopathic personality Manic-depressive manic</td>
<td>None</td>
<td>Nonsense syllables</td>
<td>Not blind</td>
<td>9 days</td>
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<td>Stone (1950)</td>
<td>2</td>
<td>Manic Schizophrenic</td>
<td>None</td>
<td>Wechsler Memory Scale</td>
<td>Not blind</td>
<td>60 days</td>
<td>20</td>
<td>No</td>
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<td>Stieper et al. (1951)</td>
<td>12</td>
<td>Paranoid schizophrenics</td>
<td>Normals</td>
<td>Wechsler-Bellevue</td>
<td>Not blind</td>
<td>3 weeks</td>
<td>15</td>
<td>Yes</td>
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<td>Pascal and Zeaman (1951)</td>
<td>1</td>
<td>Depression</td>
<td>None</td>
<td>Wechsler-Bellevue</td>
<td>Not blind</td>
<td>2 weeks</td>
<td>10</td>
<td>No</td>
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<td>Janis and Astrachan (1951)</td>
<td>9</td>
<td>Varied (mostly schizophrenics)</td>
<td>Other psychiatric patients</td>
<td>Stanford-Binet</td>
<td>Not blind</td>
<td>4 weeks</td>
<td>18.7</td>
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<td>Study</td>
<td>Sample Size</td>
<td>Conditions</td>
<td>Tests</td>
<td>Double-blind</td>
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<td>Outcome</td>
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<tr>
<td>Michael (1954)</td>
<td>30</td>
<td>Varied</td>
<td>Other psychiatric patients</td>
<td>Not blind</td>
<td>1 week, 6 weeks</td>
<td>Yes</td>
<td>7.46 (5-13)</td>
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<td>Hetherington (1956)</td>
<td>30</td>
<td>Depression</td>
<td>Normals</td>
<td>Not blind</td>
<td>2-3 days, 10 days</td>
<td>No</td>
<td>(5-10)</td>
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<td>Korin et al. (1956)</td>
<td>40</td>
<td>Varied (depression-29, schizophrenia-11)</td>
<td>Other psychiatric patients (possible EST candidates)</td>
<td>Not blind</td>
<td>1, 2, and 3 weeks after ECT</td>
<td>No data</td>
<td>No</td>
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<tr>
<td>Miura et al. (1960)</td>
<td>1</td>
<td>Reactive confusional state</td>
<td>None</td>
<td>Not blind</td>
<td>No data (patient's subjective complaints)</td>
<td>No</td>
<td>11</td>
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<tr>
<td>Cronholm and Mølender (1964)</td>
<td>28</td>
<td>Varied (mostly depressives)</td>
<td>None (patients as own controls)</td>
<td>Not blind</td>
<td>27-52 days</td>
<td>No</td>
<td>5.3 (2-12)</td>
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<td>Schwartzman and Termansen (1967)</td>
<td>28</td>
<td>Varied (many schizophrenic or borderline)</td>
<td>Other psychiatric patients</td>
<td>Not blind</td>
<td>No data</td>
<td>No data</td>
<td>No</td>
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<td>Halliday et al. (1968)</td>
<td>52</td>
<td>Endogenous depression</td>
<td>None (bilateral vs. unilateral)</td>
<td>Double-blind</td>
<td>3 months</td>
<td>Yes</td>
<td>6.2 left unilateral, 5.9 right unilateral, 5.5 bilateral</td>
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<td>Strain et al. (1968)</td>
<td>96</td>
<td>Depression</td>
<td>None (bilateral vs. unilateral)</td>
<td>Double-blind</td>
<td>10 days</td>
<td>No</td>
<td>8.4 unilateral, 7.5 bilateral</td>
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<tr>
<td>Authors</td>
<td>n</td>
<td>Diagnoses</td>
<td>Non-ECT controls</td>
<td>Test measures</td>
<td>Blindness</td>
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<td>Bidder et al. (1970)</td>
<td>96</td>
<td>Depression</td>
<td>None (bilateral vs. unilateral)</td>
<td>Paired associates, Revised Benton, Personal data sheet</td>
<td>Double-blind</td>
<td>30 days</td>
<td>7.5 bilateral (4-12)</td>
<td>Yes</td>
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<tr>
<td>Cronin et al. (1970)</td>
<td>45</td>
<td>Depression (endogenous and reactive)</td>
<td>None (bilateral vs. unilateral)</td>
<td>Graham-Kendall, Benton visual retention, Modified word learning, Digit span, Wechsler Memory Scale</td>
<td>Blind (to type of ECT)</td>
<td>4-6 weeks</td>
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<td>Miller (1970)</td>
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<td>No data</td>
<td>Other psychiatric patients</td>
<td>Paired associates</td>
<td>Not blind</td>
<td>3-6 days</td>
<td>6.0</td>
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<tr>
<td>Brunschwig et al. (1971)</td>
<td>96</td>
<td>Depression</td>
<td>None</td>
<td>Paired associates, Revised Benton, Personal data sheet</td>
<td>Double-blind</td>
<td>10 days</td>
<td>7.8 bilateral (4-12)</td>
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<td>Goldman et al. (1972)</td>
<td>20</td>
<td>Chronic schizofrenics</td>
<td>Chronic schizophrenia</td>
<td>Bender-Gestalt, Revised Benton, WAIS</td>
<td>Not blind</td>
<td>10-15 years</td>
<td>69.5 (50-219)</td>
<td>Yes</td>
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<td>Templer et al. (1973)</td>
<td>22</td>
<td>Chronic schizofrenics</td>
<td>Chronic schizophrenia</td>
<td>Bender-Gestalt, Revised Benton, WAIS, MMPI</td>
<td>Double-blind</td>
<td>&gt; 7 years</td>
<td>58.5 (40-263)</td>
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<td>Small (1974)</td>
<td>50</td>
<td>Varied (mostly schizofrenics)</td>
<td>50 Fluorohydrocortisone-treated patients</td>
<td>Wechsler Memory Scale, Shirley-Hartford, MMPI</td>
<td>Double-blind</td>
<td>After 5th ECT</td>
<td>(&lt; 10-&gt; 30)</td>
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<tr>
<td></td>
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<td>After end of ECT</td>
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<td>Study</td>
<td>Methodological Details</td>
<td>Test Measures</td>
<td>Double-blind</td>
<td>Duration</td>
<td>Delayed Recall</td>
<td>ECT/DMF?</td>
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<td>Repenstein et al. 1975</td>
<td>Probably institutional melancholia</td>
<td>Memory tests (WAIS)</td>
<td>Not blind</td>
<td>11 months</td>
<td>Approx 14'</td>
<td>Yes</td>
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<tr>
<td>Squire et al. 1975</td>
<td>Depression, normals</td>
<td>Television program recognition, WAIS</td>
<td>Not blind</td>
<td>1-2 weeks</td>
<td>7.6 (5-13)</td>
<td>No</td>
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<td>Squire and Chace 1975</td>
<td>Varied (all with depressive symptoms)</td>
<td>Paired associates</td>
<td>Not blind</td>
<td>6-9 months</td>
<td>10.1 (5-17)</td>
<td>No</td>
<td></td>
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<tr>
<td>Jackson 1978</td>
<td>No data</td>
<td>Wechsler Memory Scale</td>
<td>Double-blind</td>
<td>10 days</td>
<td>6.0</td>
<td>No</td>
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<tr>
<td>Freeman et al. 1980</td>
<td>Varied, normals</td>
<td>Personal remote memory, Logical memory, Famous personalities, Verbal memory, Face-name connection, Decision time, Movement time</td>
<td>Not blind</td>
<td>9 months-30 years ( \bar{X}=10 ) years</td>
<td>No data</td>
<td>Yes</td>
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<tr>
<td>Weeks et al. 1980</td>
<td>Depression, non-ECT depression, normals</td>
<td>Famous personalities, Delayed recall recognition, Verbal memory, Decision time, Personal remote memory, Movement time, Fluid movement, Visual memory, Visual design, Anomalous sentence repetition</td>
<td>Blind</td>
<td>4 months</td>
<td>7.2 (2-20)</td>
<td>No</td>
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### Table I. Continued

<table>
<thead>
<tr>
<th>Authors</th>
<th>n</th>
<th>Diagnoses</th>
<th>Non-ECT controls</th>
<th>Test measures</th>
<th>Blindness</th>
<th>Follow-up interval</th>
<th>Mean no. (range) of treatments</th>
<th>Prolonged defects</th>
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<tbody>
<tr>
<td>Squire et al. (1981)</td>
<td>43</td>
<td>Varied (42 with depression)</td>
<td>Other inpatients (n = 7)</td>
<td>Public events: recognition/recall</td>
<td>Not blind</td>
<td>1 week 7 months</td>
<td>10.2 (5-21)</td>
<td>Yes(^a)</td>
</tr>
</tbody>
</table>

\(^a\) See text for explanation.  
\(^b\) Constructed by the authors.
T and Memory Dysfunction

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Abnormal findings at 6 months may well be regarded as prolonged however. The discussion at the end of that article indicates clearly why blindness is an important variable in scientific research. One of the authors argues against the mechanistic approach in psychiatry and says that "careful studies" revealed a "definite 'organic' change in memory which does not entirely clear up." Unfortunately he does not cite these studies and gives the reader no evidence on which to base this conclusion.

Brody (1944) reports on five patients followed approximately 1 to 2 years after a course of ECT. This is a series of case reports with no indication of pretreatment status, no control group, and no specific data. These examples are convincing enough that some patients do, for a long period of time, complain of defects in their memory after a course of ECT. A careful look at the case reports however confounds the issue somewhat. One case is described as an "hysterical type," and attempts to accurately assess memory defects in patients, one of whose major symptoms may be amnesia, is clearly difficult at best. Another case is a patient with "mild" arteriosclerosis and hypochondriasis. Since her arteriosclerosis is "mild," he suggests that it may be ignored as an etiological factor in her memory dysfunction. The problems with case reports are clear enough. But in an area where the assessment of long-term effects is confounded by so many variables such as patients' attitude toward and expectations of the treatment, the subjective nature of the complaints, the lack of controls, and the lack of blindness on the part of the examiner, accurate assessment of these complaints is impossible. When the etiology of these patients' memory complaints is difficult to answer, it seems risky to suggest, as the author does, that ECT causes "permanent or semi-permanent" damage to the brain when it is based on five case reports.

Huston and Strother's (1948) paper reports on 75 patients of mixed diagnoses, all with affective symptoms. Older patients (median age 47) were age-matched with normal controls because of the effect of age on one of the testing instruments. On the first posttreatment follow-up, 11 days after the termination of ECT, there was clear evidence of impairment in mental efficiency measured by the Babcock Revision and the Shipley-Hartford Test. The authors maintain that with the Babcock "mental efficiency as measured by this test primarily involves memory and secondarily, attention and speed." For those tested at the 6-month follow-up, mental efficiency scores were significantly improved \( p < 0.01 \) over posttreatment scores. Age-matched subjects were not significantly different from controls who had not undergone ECT.

The Levy et al. (1942) and Brody (1944) papers are frequently quoted as definitive evidence for prolonged memory defects. Huston and Strother's (1948) data, however, constitute the only well-controlled evidence from this era with a reasonably long-term follow-up and the data suggest return to pretreatment levels of memory function.
The 1950s brought some more rigorously designed studies and some interesting developments. Nine studies are reviewed from that decade (Janis, 1950; Worchel and Narcisco, 1950; Stone, 1950; Stieper et al., 1951; Pascal and Zeaman, 1951; Janis and Astrachan, 1951; Michael, 1954; Hetherington, 1956; Korin et al., 1956). None of the studies is blind. Three employ no control group (Worchel and Narcisco, 1950; Stone, 1950; Pascal and Zeaman, 1951), and three report on either one (Pascal and Zeaman, 1951) or only two patients (Worchel and Narcisco, 1950; Stone, 1950). Four of the six studies that are not case reports include patients with a variety of diagnoses. Three indicate prolonged defects (Janis, 1950; Stieper et al., 1951; Janis and Astrachan, 1951). Six show a return to pretreatment levels of mental efficiency (Worchel and Narcisco, 1950; Stone, 1950; Pascal and Zeaman, 1951; Michael, 1954; Hetherington, 1956; Korin et al., 1956).

Janis's study (1950) is frequently quoted in the literature on prolonged defects after ECT. Because it employed a control group and specifically tested personal rather than impersonal memories, the study has become a prominent one in the ECT literature. Some critics suggest that tests which measure impersonal memories, such as paired associates or learning of nonsense syllables, may not be sensitive indicators of post-ECT amnesias. The specific purpose of Janis's study was to investigate whether the short-term memory defects after ECT, so clearly evident on clinical grounds, persisted beyond the few weeks after treatment. Janis points out that "isolate case reports provide extremely inadequate evidence for the hypothesis that ECT produces sustained memory defect." He studied 19 patients who received between 8 and 27 standard bilateral ECTs with a mean of 17. Both patients and comparably ill controls who did not receive ECT were given an intensive pretreatment interview which included school, job, and psychiatric history, sexual, marital, and family relationships, childhood experiences, and other major life events. Approximately 4 weeks after the ECT there was evidence of retrograde amnesia in all 19 of the patients studied. These defects were described as "negligible" in the control patients who were tested after a similar time interval. In an attempt to determine the duration of these defects, Janis followed five patients for approximately 3 months more. There were minor improvements but a substantial defect was still present at the second follow-up. Because of the problems of motivation and the fact that some patients' recall was improved when extra time was given to "work on" recovering these memories, Janis concluded the following: (i) "it seems to be fairly probable that the post-ECT recall failures are especially likely to involve experiences which tend to arouse anxiety, guilt and a lowering of self-esteem"; (ii) "If it is true that the memory gaps found after ECT are largely determined by motivational factors, it would follow that the amnesias do not represent irreversible organic defect"; and (iii) "The view which emerges is that ECT amnesias involve a reversible retention loss." One may well argue about the verifiability of psychodynamic formulations of motivation and its relationship to memory defect.
Studies to document the effects of motivation on memory would be helpful in order to correctly interpret Janis' data. Later authors do not support Janis' conclusion that memory loss is important to the therapeutic process of ECT. This study is an interesting one and it is widely quoted. It involves controls, testing before and at two intervals after ECT, personal rather than impersonal memories, and follow-up intervals where the obvious clinical posttreatment organic symptoms do not confound the issue of whether the memory defects are prolonged. This study, though widely quoted, has several weaknesses. The patients studied had a variety of diagnoses, but most were schizophrenic. The effects of this variable in the sample population are uncertain. Janis qualifies his data as documentary rather than quantitative in character. The presentation of more standardized objective data with estimates of interrater reliability would have been more compelling. He followed only five patients of the original group for the second follow-up, and no selection criteria or clinical characteristics of these five are reported. The possibility exists that these five continued to be more symptomatic than the rest of the group and were therefore more available for interview. He suggests that one piece of evidence that ECT specifically caused these defects was that there was a fair degree of heterogeneity with respect to the additional forms of psychiatric treatment received by the ECT group, and yet all demonstrated memory loss. However, three of the patients received insulin-shock treatments during the test-retest interval which introduces another important factor. One patient received as many as 50 insulin coma treatments. Whether these three patients were included in the five studied at the second follow-up is uncertain. Janis does not comment on this. Until such confounding factors are removed it seems somewhat risky to regard ECT as causative. Despite the variables cited above, this study is widely quoted in the ECT literature as definitive evidence of prolonged memory defects. A second study by Janis (Janis and Astrachan, 1951) demonstrates the prolonged memory defects in personal, but this time "routine," memory items presumably minimizing the anxiety-provoking and motivational aspects of the earlier study. Again these defects were demonstrable approximately 4 weeks after treatment. Janis suggests that these "artificially induced repressions" helped to contribute to the reduction of affective disturbances and therefore, presumably, are important in the therapeutic aspects of the treatment. Again, later authors do not support this interpretation.

Stieper et al.'s (1951) paper deserves separate comment. It involved pre- and post-treatment testing, a control group, both personal and impersonal memory items, and a 3-week follow-up. He found that general mental efficiency as measured by the CVS abbreviated intelligence scale was improved beyond the pre-treatment level 3 weeks after ECT. He corroborated Janis' data that personal memories were more affected by ECT than were impersonal memories. Unlike Janis, though, he found that patients whose clinical status at follow-up was better, suffered less from amnesia, even for personal events. In addition his study
patients were chronic schizophrenics, a factor which is known to influence performance on psychological tests (Luborsky, 1948; Heaton et al., 1978).

Fewer studies were found in the 1960s which detailed prolonged memory defects after ECT. One might speculate that research interest in ECT declined somewhat after the introduction of the antidepressant medications. Five studies from the 1960s are reviewed (Miura et al., 1960; Cronholm and Molander, 1964; Schwartzman and Ternansen, 1967; Halliday et al., 1968; Strain et al., 1968). One is a single case report (Miura et al., 1960). Two studies are not blind (Cronholm and Molander, 1964; Schwartzman and Ternansen, 1967); the other two are double-blind (Halliday et al., 1968; Strain et al., 1968). Only one has a control group of patients not treated with ECT (Schwartzman and Ternansen, 1967). The other three either compare bilateral with unilateral electrode placement (Halliday et al., 1968; Strain et al., 1968), or use the patients as their own controls (Cronholm and Molander, 1964). Three of these studies found no objective evidence for prolonged defects (Cronholm and Molander, 1964; Strain et al., 1968; Schwartzman and Ternansen, 1967). The other two studies (Miura et al., 1960; Halliday et al., 1968) suggest such defects. Despite the lack of evidence for prolonged defects in Schwartzman and Ternansen's (1967) paper 27 of the original 79 patients in this sample answered a questionnaire and complained of persistent memory defects. Patients' complaints of memory defects which are at variance with their performance on memory tests, especially when patients received bilateral ECT, will be seen to be a characteristic also found by later authors.

The study by Halliday et al. (1968) demonstrated memory defects 3 months after ECT. There was random assignment to three treatment groups: bilateral, right, and left unilateral ECT. There were specified criteria for the diagnosis of endogenous depression and other inclusion criteria included age under 65 years, no evidence of organic brain damage, no prolonged duration of depressive illness, and no ECT in the previous 3 months. The authors measured both verbal and nonverbal memories using a special test battery devised by Williams (1968). They also employed a delay of approximately ½ hr in the interval between learning and recall. Such a delay is known to maximize the chances of finding impairment in memory (Cronholm and Ottoson, 1961; Cronholm and Molander, 1964; Dornbush, 1972; Squire and Miller, 1974). They studied 52 patients before and after a series of four ECTs and again at the end of the series. For those who received more than four ECTs the mean number of extra ECTs was 3.6, 3.9, and 4.8, for the bilateral, right unilateral, and left unilateral treatment groups, respectively. Of the 52 original patients 44 were retested 3 months after the termination of ECT; of the 8 who were not retested, 3 had been switched to a different treatment modality, 1 had committed suicide, and 4 were lost to follow-up. They demonstrated the evidence for the differential effect of right vs. left hemispheric stimulation on nonverbal and verbal memories, respectively. After 3 months there was definite improvement in all of the test scores, but some residual defects per-
sisted. Nonverbal learning, originally worst in the right unilateral group, was not worst in the bilaterally treated group. The authors state that the study was conducted double-blind. The main point of the article was to determine the differential effects of dominant vs. nondominant hemispheric stimulation, rather than the persistence of memory defects per se. Though one cannot be sure that this necessarily protects against experimenter bias, it does seem more compelling. A longer follow-up is essential to determine how long these posttreatment amnesias persist.

Cronholm and Molander’s study (1964) is one in a series of studies which investigated the acute and long-term effects of ECT on memory (Cronholm and Molander, 1957, 1964; Cronholm and Blomquist, 1959; Ottoson, 1959; Cronholm and Ottoson, 1961, 1963). Their patients were studied before and at varying intervals after a course of ECT. Three different memory tests including words, figures, and personal data were employed. In summary, these studies showed that depression itself affects learning, while ECT affects retention of newly learned material. They found a decline in memory production just after the second ECT (Cronholm and Molander, 1975), and a pronounced decline when there was a delay of 3 hr between learning and reproduction. However, the increase in “forgetting scores,” the differences between immediate and delayed reproduction scores, was not found after 1 month (Cronholm and Molander, 1964).

The report by Strain et al. (1968) is one in a series of three papers (Strain et al., 1968; Bidder et al., 1970; Brunswig et al., 1971), which report on a sample of 96 patients studied at various intervals before and after ECT. The Strain et al. data suggest that 10 days after the last ECT there are no significant differences between patients treated with bilateral and nondominant unilateral ECT on any of the tests they used. The worst defect was with recent memories on their Personal Data Sheet, corroborating Janis’ findings.

Eleven studies from the 1970s are reviewed (Bidder et al., 1970; Cronin et al., 1970; Miller, 1970; Brunswig et al., 1971; Goldman et al., 1972; Temple et al., 1973; Small, 1974; Regenstein et al., 1975; Squire et al., 1975; Squire and Chace, 1975; Jackson, 1978). Five studies are not blind (Miller, 1970; Goldman et al., 1972; Regenstein et al., 1975; Squire et al., 1975; Squire and Chace, 1975). The remainder are reportedly blind (Bidder et al., 1970; Brunswig et al., 1971; Templer et al., 1973; Small, 1974; Jackson, 1978). Only one is a single case report (Regenstein et al., 1975). Seven do not suggest prolonged memory defects after ECT (Bidder et al., 1979; Miller, 1970; Brunswig et al., 1971; Small, 1974; Squire et al., 1975; Squire and Chace, 1975; Jackson, 1978), though Small’s study deserves separate comment. Four of the studies do report these defects (Cronin et al., 1970; Goldman et al., 1972; Templer et al., 1973; Regenstein et al., 1975), although Templer’s data are puzzling and are discussed in more detail below.
Brunschwig et al. (1971) report on 33 patients tested 1 year after ECT. In memory, the authors found the expected decline in memory 36 hr after the last ECT in verbal, nonverbal, and personal memory. They also corroborate earlier studies suggesting that the worst deficit is with personal memory. The verbal memory scores 30 days posttreatment were better than those before treatment, a fact which may be explained by improvement in depressive illness alone. Nonverbal test scores, based on a visual reproduction task, did not show any significant decline from pretesting to posttesting. The 1-year follow-up revealed an additional significant improvement in memory on the paired associate tasks, but unfortunately the authors apparently did not readminister the personal data set. This is unfortunate since data on this particular item may have been very illuminating.

Small (1974), in a prospective double-blind study, investigated memory function in patients before and at varying intervals after ECT and flurothyl (neloklon) therapy. Subjects were randomly assigned to either treatment modality. Fifty patients treated with flurothyl and 50 patients treated with one form of ECT (bilateral, right, or left unilateral) were compared using the Wechsler Memory Scale, other psychological tests, and clinical ratings. Forty-four patients were available for follow-up 2 to 5 years after the termination of the treatment. There were no significant differences among the four groups on the Wechsler Memory Scale, though patients treated with bilateral ECT had the lowest mean scores. Patients' evaluations of their memory did, however, correlate with the method of seizure induction. Half of the patients who had had bilateral ECT complained of persistent memory defects. Very few of those treated with one of the other modalities complained of such defects, though some did. Small concluded that "severe impairment of memory persisting long after convulsive therapy was complexly related to the severity of the index illness, as well as the number of convulsive treatments received and clinical status at the time of follow-up." There was no correlation between the subjective complaints of memory defects and performance on the Wechsler Memory Scale.

Throughout the 1970s and early 1980s Squire and associates have published a series of well-designed studies on the amnesic effects of ECT (Squire and Miller, 1974; Squire, 1974, 1975, 1977; Squire et al., 1975, 1976a, 1976b, 1981; Squire and Slater, 1975, 1978; Squire and Chace, 1975). In summary, they replicated earlier studies documenting both the retrograde and the anterograde amnesias; they also found that even remote memories acquired years before the treatment could be affected by ECT (Squire et al., 1975).

Squire and Chace (1975) studied patients 6 to 9 months after at least five bilateral or right unilateral ECTs. There were two control groups. One consisted of comparably ill patients hospitalized 6 months previously who did not receive ECT. The other was an inpatient group currently receiving ECT who were tested after the fifth treatment. This inpatient group gave an estimate of the marked
memory impairment shortly after ECT so well documented in other studies. Six tests were administered, evaluating recent and remote memories as well as new learning. Their results suggest that memory function in all areas had returned to normal within 6 to 9 months after treatment when study and control patients were compared. It is unfortunate that the sample population was not more uniform. The sample population consisted of various diagnoses even though all patients had depressive symptoms. One of the most interesting aspects of this study is a statistically significant observation in patients who had received bilateral ECT: these patients complained more often of memory dysfunction than did patients who had received unilateral treatments, and yet their complaints were discrepant with objective memory tests, replicating Small's (1974) findings.

In the discussion Squire and Chace (1975) suggest an interesting possibility: "having experienced pronounced memory difficulty shortly after ECT an individual may subsequently be alert to each failure of memory... recall failures or forgetfulness may be noticed more readily than before, even though they occur at a normal frequency. In this way, bilateral ECT, which initially causes this marked memory problem, conceivably could produce in healthy persons, as well as in ill persons, a persistent but erroneous belief that memory remains impaired."

It is an interesting hypothesis. The authors caution, however, that patients likely to receive ECT may be more predisposed initially to develop memory problems, although why this would be true is uncertain. They also caution that available psychological tests, even though they may be designed to maximize the chances of finding memory difficulties, may be insensitive to failures of recall. They suggest a prospective research design in an ideal study.

Templer et al.'s study (1973) is an amplification of a previous study done by Goldman et al. (1972), which found evidence of decreased visual retention and signs of cerebral dysfunction 10 years after the administration of 50 or more ECTs in psychiatric patients. Templer et al. tested 22 chronic schizophrenic patients who had received more than 50 ECTs, and 22 control schizophrenic patients who had not received ECT. The purposes of their study were to replicate their earlier findings, to compare ECT and control patients on the Wechsler Adult Intelligence Scale (WAIS), and to compare the degree of psychosis between groups. This latter factor was thought to be an important variable in the poorer performance demonstrated in the ECT patients in the earlier study. When this degree of psychosis was controlled for in the two groups, no differences were found between the ECT and the control patients on either the Benton or the WAIS. However, the performances on the Bender-Gestalt remained significantly inferior in the ECT-treated group. Testing was done at least 7 years after ECT: the number of prior ECTs was between 50 and 263 treatments. Why such defects were obtained on a test of perceptual motor functioning rather than on tests of memory or general intelligence is uncertain. However, the authors conclude that these inferior Bender-Gestalt performances suggest that ECT does in fact cause brain damage. There are a number of problems with both this and with the
Goldman et al. study. The difficulty distinguishing chronic schizophrenic patients from organic patients on psychological tests has already been discussed (Heaton et al., 1978). In both studies, experimental and control patients were matched for age, sex, race, and years of education. The length of hospitalization is not mentioned in the Templer et al. paper; it is 2½ years less for the control group in Goldman et al.'s subjects, but this difference is minimized by the authors since there were no significant correlations between test scores and years of hospitalization. No information is provided in either paper about the type or duration of medication given to both groups, a factor which may have some bearing. Initial differences between schizophrenic patients who receive ECT and those who do not are not discussed.

Perhaps the most convincing evidence of prolonged memory defects after ECT come from two more recent studies. Squire et al. (1981) prospectively studied 43 patients before and after ECT. They found that 7 months after ECT the defects in memory, clearly evident 1 week after treatments, were no longer present and in some cases the memory function was better than that before ECT. These data obtained for recall and recognition of public events, as well as for recall of television programs. However, for the recall of personal autobiographical material the authors found that ECT “markedly” affected these memories and that some deficit was still present 7 months later. This was especially true for events temporally close to the treatment, especially for the day of admission to hospital. Even prompting of previously recalled material did not elicit recognition of that material in half the patients. This particular aspect of the study was undertaken to replicate Janis' earlier findings, and did indeed replicate them. However, the defects were thought to be confined primarily to the period of treatment and were described as “relatively subtle.”

Similar findings are reported in a retrospective study by Freeman et al. (1980). Patients who “complained” of unwanted side effects of ECT were elicited from a newspaper advertisement (n = 14) and referral from local psychiatrists (n = 12). By far the most common complaint was some form of memory dysfunction. Normal volunteers served as controls. The complainers did rate themselves as being more depressed and anxious and were taking more medications than controls. ECT patients, including 13 patients who had had ECT but did not complain of unwanted side effects, were significantly impaired on 8 cognitive tests and unimpaired on 11, when compared with normal controls. An analysis of variance was done to control for the effects of medication use, severity of depression, number of other symptoms, age, and social class. When these variables were controlled for the differences between the two groups were less significant, but there were still significant differences in logical memory, verbal learning, and a face-name connection task. Whether the defects found in the ECT-treated patients were actually attributable to ECT is certainly debatable, especially considering the method of sample selection and the retrospective nature of the study. What is important however is that the subjects themselves
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clearly associated the memory impairment with having had ECT. Most of the subjects found the memory impairment "irritating rather than incapacitating," and the authors feel that the data "indicate fairly convincingly that ECT does not normally produce such enduring defects on memory, though they do not prove that it never does so." They estimate that enduring defects may affect 1 in 100 patients.

In a companion paper (Weeks et al., 1980) the same authors described a prospective study of cognitive function in ECT and non-ECT treated depressed patients. Subjects were studied before ECT, 1 week after the treatment, and again at 4 and 7 months. Before ECT, patients were significantly more impaired in 9 of 19 cognitive tests than the non-ECT controls, presumably documenting the effects of more severe depression in the ECT-treated patients. At 4-month follow-up, however, the ECT patients did better on one task and worse on another than the controls. At 7 months there was only one significant difference between the two groups and this was in favor of the ECT-treated patients. However, both groups of depressed patients performed less well than nondepressed normal controls.

CONCLUSIONS

Although the improvement in the design of more recent studies over those from the 1940s and the 1950s is obvious, a number of methodological problems remain with many of them. The authors have reviewed 39 papers which concern the relatively long-term effects of ECT on human memory. If one were simply to count the studies which indicate prolonged defects vs. those that do not, the results appear to indicate that ECT does not produce prolonged memory defects. A tally of this kind, however, would be a major oversimplification of the results of the studies which have been reviewed. Frankel (1977) makes a convincing point that only by "painstakingly sifting the evidence" can one draw reasonable conclusions about this complicated issue. A major problem in interpreting the validity of these studies is the lack of uniformity and sensitivity of the memory tests used. The other deficiencies in methodology have already been referred to and should provoke caution in the reader.

Some general conclusions do seem possible, however: (i) bilateral ECT frequently induces memory changes, even with the standard number of treatments, e.g., eight or nine; (ii) the effects of ECT on memory appear to be cumulative, with greater effects seen with successive treatments; (iii) the majority of cognitive and memory defects appear to be entirely reversible with a return to pretreatment levels of function or better usually within 6 to 7 months; (iv) some subtle but persistent defects may be found in some patients some months after ECT.
especially in personal or autobiographical material; and (v) the persistent defects tend to be of an irritating rather than a seriously incapacitating nature. In light of these conclusions, a number of authors have documented convincingly that predominant unilateral ECT has a clear advantage over bilateral ECT in minimizing the amount of apparent memory defect. Given that cognitive dysfunction, primarily in memory, is virtually the only significant side effect of ECT, one that is irrelevant to its therapeutic efficacy, one might argue, as some authors do, that unilateral ECT should be used more frequently that it is.

In summary, then, there is good evidence that ECT does not routinely produce serious long-term memory defects among the more well-designed prospective studies. Some of the more recent well-designed studies, however, do document persisting, though usually not serious, memory problems. These studies need to be replicated. There are too few well-designed studies which demonstrate conclusively that ECT either does or does not produce prolonged memory defects. The need for well-designed, adequately controlled, and carefully executed studies with long-term follow-up is very clear. In this controversial area there can be no substitute.

REFERENCES


Dysfunction


Memory Dysfunction


