., Barbero, G. J., and Bolander, F. (1981a). Depression in hospitalized pediatric
ts. *J. Am. Acad. Child Psychiat.* 20: 123-134.
L., Husain, A., Shekim, W. O., Hodges, K. K., Cytryn, L., and McKnew, D. H.
b). Current perspectives on childhood depression: An overview. *Am. J. Psychiat.*
43-153.
., and Stewart, M. A. (1979). The diagnosis of depression in children. *J. Af-*
Disorders 1: 213-217.
, and Foster, F. G. (1978). EEG sleep and depression, in *Sleep Disorders: Diag-*
nd Treatment, Williams, R. L., and Karacan, I. (eds.), Wiley and Sons, New
pp. 163-204.
, Coble, P., Kane, J., Petti, T., and Conners, C. K. (1979). Imipramine and EEG
n children with depressive symptoms. *Psychopharmacology* 60: 117-123.
nd Dement, W. C. (1980). Sleep and aging. *Sleep* 3: 119-220.
I. (1980). Affective disorders in childhood: A review and perspective. *Psychiat.*
'orth Am. 3: 403-424.
J., Chambers, W., Halpern, F., Hanlon, C., and Sachar, E. J. (1979). Cortisol
ecretion in prepubertal depressive illness: A preliminary report. *Psychoneuro-*
inology 4: 191-197.
J., Tabrizi, M. A., Davies, M., Goetz, R., Chambers, W. J., Halpern, F., and
, E. J. (1981). Prepubertal endogenous major depressives hyposecrete growth
ne in response to insulin-induced hypoglycemia. *Biol. Psychiat.* 16: 801-818.
t. A., and Kales, A. (eds.) (1968). *A Manual of Standardized Terminology,*
ques and Scoring System for Sleep Stages of Human Subjects, NIH Publication
4, U.S. Government Printing Office, Washington, D.C.
Shaw, D. H., and Kupfer, D. J. (1980). Effects of aging on EEG sleep in depres-
'eep 3: 31-40.
Vogel, F., McAbee, R. S., and Thurmond, A. J. (1980). Improvement of depres-
' REM sleep deprivation: New findings and a theory. *Arch. Gen. Psychiat.* 37:
3.
A., Rutman, J., Sullivan, L., Penick, E. C., and Dietz, S. G. (1973). Depression
lren referred to an educational diagnostic center. *J. Pediat.* 83: 1065-1072.
.., Karacan, I., and Hursch, C. J. (1974). *EEG of Human Sleep*, Wiley and Sons,
ork.

Biological Psychiatry, Vol. 17, No. 10, 1982

ECT - Memory Loss

Review

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

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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; Dornbush, 1972; Glassman *et al.*, 1975). Not only is ECT ef-

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0006-3223/82/1000-1169$03.00/1

fective but it is also quite safe, with mortality rates consistently reported as much less than 0.1% (Pitts, 1972; Alexander, 1956; Matthew and Constan, 1964; Barker 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 extemely 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-

ıcative, or even heuristic, they should not be the basis for generalizations about ıe effects of ECT on memory when other more important variables are not ·tailed. The length of follow-up is obviously an important concern. Differences ınd at different follow-up intervals have a good deal of bearing on one's inter-retation of the results. The obvious organicity present just after the administra-on of the treatment should not be confused with the evidence for defects in ıemory long after the termination of treatment. Therefore, the term "prolonged" ıould not be used to refer to defects of relatively short duration. Test sensitivity an extremely important issue. Many of these studies have been criticized for sing tests of only new learning, of only verbal memories, of material meaningless patients, of impersonal rather than personal memories, or for failing to incor-ɔrate delays between learning and reproduction, a measure known to maximize ıe chance of finding memory defects (Cronholm and Ottoson, 1961; Squire ıd Miller, 1974; Dornbush, 1972; Korin *et al.*, 1956).

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

REVIEW

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

Eleven studies from the 1940s are considered (Levy *et al.*, 1942; Brody, 44; Norman and Shea, 1946; Medlicott, 1948; Tyler and Lowenback, 1947; ɔorsky, 1948; Huston and Strother, 1948; Smith *et al.*, 1943; Perlson, 1945; ne, 1947; Rabin, 1948). None of the studies is blind. Seven of the studies entirely anecdotal or present minimal data (Medlicott, 1948; Brody, 1944;

Table I. Prolonged Memory Defects After Electroconvulsive Therapy

Authors	*n*	Diagnoses	Non-ECT controls	Test measures	Blindness	Follow-up interval	Mean no. (range) of treatments	Prolonged defects
Levy *et al.* (1942)	12	Varied (mostly depressives)	Metrazol-treated patients	EEGs Mental status exams	Not blind	6 months	6.5 (2-11)	Yes
Smith *et al.* (1943)	279	Varied	None	No data	Not blind	Up to 12 months	(9-10)	No
Brody (1944)	5	Varied	None	No data (patients' subjective complaints)	Not blind	1-2 years	(4-15)	Yes
Perlson (1945)	1	Dementia praecox paranoid type	None	Various psychological tests	Not blind	3 months	154-ECT 94-Metrazol	No
Norman and Shea (1946)	266	Varied (mostly schizophrenics)	None	No data	Not blind	No data (> 3 months?)	(8-50)	Yes
Tyler and Lowenbach (1947)	32	Varied (mostly schizophrenics)	None	No data	Not blind	1-5 years	11.1 (5-22)	No
Stone (1947)	29	Varied (mostly schizophrenics)	None	Wechsler Memory Scale	Not blind	2-3 weeks	17.0 (4-20)	No
Huston and Strother (1948)	75	Varied (all with depressive symptoms)	Normals	Babcock Shipley-Hartford	Not blind	6 months	7.8 (1-18)	No
Medlicott (1948)	100	Varied	None	No data (patients' subjective complaints on a questionnaire)	Not blind	> 6 months	No data	Yes

Table I. Continued

Authors	*n*	Diagnoses	Non-ECT controls	Test measures	Blindness	Follow-up interval	Mean no. (range) of treatments	Prolonged defects
Luborsky (1948)	12	Varied	None	Wechsler-Bellevue Stanford-Binet Goodenough Man-Drawing	Not blind	6 months	12	No
Rabin (1948)	6	Schizophrenics	None	Rorschach	Not blind	No data	(110-234)	[illegible]
Janis (1950)	19	Varied (mostly schizophrenics)	Other psychiatric patients	Personal data inventory[b]	Not blind	4 weeks 2½-3½ mos.	17 (8-27)	Yes
Worschel and Narcisco (1950)	2	Psychopathic personality Manic-depressive manic	None	Nonsense syllables	Not blind	9 days	7	No
Stone (1950)	2	Manic Schizophrenic	None	Wechsler Memory Scale Army alpha	Not blind	60 days 3 years	20 14	No
Stieper *et al.* (1951)	12	Paranoid schizophrenics	Normals	Wechsler Memory Scale CVS abbrev. intelligence scale Personal memory inventory[b]	Not blind	3 weeks	15 (5-25)	Yes
Pascal and Zeaman (1951)	1	Depression	None	Wechsler-Bellevue Rorschach	Not blind	2 weeks 7 months	10	No
Janis and Astrachan (1951)	9	Varied (mostly schizophrenics)	Other psychiatric patients	Personal data inventory[b]	Not blind	4 weeks	18.7 (10-30)	Yes
Michael (1954)	30	Varied	Other psychiatric patients	Noun enumeration test	Not blind	1 week 6 weeks	7.06 (5-13)	No
Hetherington (1956)	30	Depression	Normals	Varied (verbal, nonverbal motor tests)	Not blind	2-3 days 10 days	(5-10)	No
Korin *et al.* (1956)	40	Varied (depression-29, schizophrenia-11)	Other psychiatric patients (possible EST candidates)	Common words Nonsense syllables	Not blind	1, 2, and 3 weeks after ECT	No data	No
Miura *et al.* (1960)	1	Reactive confusional state	None	No data (patient's subjective complaints)	Not blind	No data	11	Yes
Cronholm and Molander (1964)	28	Varied (mostly depressives)	None (patients as own controls)	Word pairs Figures Personal data sheet[b]	Not blind	27-52 days	5.3 (2-12)	No
Schwartzman and Termansen (1967)	28	Varied (many schizophrenic or borderline)	Other psychiatric patients	Wechsler Memory Scale	Not blind	No data	No data	No[a]
Halliday *et al.* (1968)	52	Endogenous depression	None (bilateral *vs.* unilateral)	Verbal & nonverbal immediate & delayed (see Williams, 1968) Digit span Remote memory	Double-blind	3 months	6.2 left unilateral 5.9 right unilateral 5.5 bilateral	Yes
Strain *et al.* (1968)	96	Depression	None (bilateral *vs.* unilateral)	Paired associates Revised Benton visual retention test Personal data sheet[b]	Double-blind	10 days	8.4 unilateral 7.5 bilateral	No

Table I. Continued

Authors	*n*	Diagnoses	Non-ECT controls	Test measures	Blindness	Follow-up interval	Mean no. (range) of treatments	Prolonged defects
Bidder *et al.* (1970)	96	Depression	None (bilateral *vs.* unilateral)	Paired associates Revised Benton Personal data sheet[b]	Double-blind	30 days 1 year	7.5 bilateral 8.4 unilateral (4-12)	No[a]
Cronin *et al.* (1970)	45	Depression (endogenous and reactive)	None (bilateral *vs.* unilateral)	Graham-Kendall Benton visual retention Modified word learning Digit span Wechsler Memory Scale	Blind (to type of ECT)	4-6 weeks	8.0	Yes
Miller (1970)	20	No data	Other psychiatric patients	Paired associates	Not blind	3-6 days 7-14 days	6.0	No
Brunschwig *et al.* (1971)	96	Depression	None	Paired associates Revised Benton Personal data sheet[b]	Double-blind	10 days 1 year	7.8 bilateral (4-12)	No
Goldman *et al.* (1972)	20	Chronic schizophrenics	Chronic schizophrenics	Bender-Gestalt Revised Benton WAIS	Not blind	10-15 years	69.5 (50-219)	Yes
Templer *et al.* (1973)	22	Chronic schizophrenics	Chronic schizophrenics	Bender-Gestalt Revised Benton WAIS MMPI	Double-blind	> 7 years	58.5 (40-263)	Yes
Small (1974)	50	Varied (mostly schizophrenics)	50 Flurothyl-treated patients	Wechsler Memory Scale Shipley-Hartford MMPI Other psychological tests	Double-blind	After 5th ECT After end of ECT 60-90 days 2-5 years	(< 10-> 30)	No[a]
Regenstein *et al.* (1975)	1	Probably involutional melancholia	None	WAIS Mental status exam	Not blind	11 months	Approx. 145	Yes
Squire *et al.* (1975)	16	Depression	Normals	Television program recognition[b] WAIS	Not blind	1-2 weeks	7.6 (5-13)	No
Squire and Chace (1975)	16	Varied (all with depressive symptoms)	Other psychiatric patients	Paired associates Television program recognition[b] 32-item recognition Paragraph recall Complex figure drawing WAIS	Not blind	6-9 months	10.1 (5-17)	No
Jackson (1978)	34	No data	Other psychiatric patients	Wechsler Memory Scale Verbal & nonverbal memory	Double-blind	10 days	6.0	No
Freeman *et al.* (1980)	26	Varied	Normal volunteers	Personal remote memory Logical memory Famous personalities Verbal memory Face–name connection Decision time Movement time	Not blind	9 months-30 years ($\bar{x}$ = 10 years)	No data	Yes[a]
Weeks *et al.* (1980)	51	Depression	Non-ECT depression Normal volunteers	Famous personalities Delayed recall recognition Verbal memory Decision time Personal remote memory Movement time Fluid movement Visual memory Visual design Anomalous sentence repetition	Blind	4 months 7 months	7.2 (2-20)	No

Table I. Continued

Authors	*n*	Diagnoses	Non-ECT controls	Test measures	Blindness	Follow-up interval	Mean no. (range) of treatments	Prolonged defects
Squire *et al.* (1981)	43	Varied (42 with depression)	Other inpatients (n = 7)	Public events: recognition/recall Television program recall Personal events recall	Not blind	1 week 7 months	10.2 (5-21)	Yes[a]

[a] See text for expalnation.
[b] Constructed by the authors.

Tyler and Lowenback, 1947; Norman and Shea, 1946; Smith *et al.*, 1943; Perlson, 1945; Stone, 1947). Two of the papers are case reports. Three studies employed patients specifically tested before and after ECT (Levy *et al.*, 1942; Lubarsky, 1948; Huston and Strother, 1948). Lubarsky's (1948) follow-up is only 5 or 6 days 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; Lubarsky, 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 schock...." 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 dificult 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' 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 postECT 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 populatiom 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," momory 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. Patents' 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 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 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 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 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 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), alhough 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 summary 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 sheet. 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 (Indoklon) 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 antrograde 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

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 monhts. 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 nondominant 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

Alexander, G. G. (1956). Deaths following electrotherapy. *J. Am. Med. Assoc.* 161: 577-581.

American Psychiatric Association (1978). *Electroconvulsive Therapy*, Task Force Report No. 14, APA, Washington, D.C.

Andren, H. E. (1976). Treatment or mistreatment in psychiatry. (Observations on electroconvulsive therapy.) *Dis. Nerv. Syst.* 37: 605-609.

Arneson, G. A., and Butler, T. (1960). Cardiac arrest and electroschock therapy. *Am. J. Psychiat.* 117: 1020-1022.

Avery, D., and Winokur, G. (1977). The efficacy of electroconvulsive therapy and antidepressants in depression. *Biol. Psychiat.* 12: 507-523.

Barker, J. C., and Baker, A. A. (1959). Deaths associated with electroplexy. *J. Mental Sci.* 105: 339-348.

Barton, J. L. (1977). ECT in depression: The evidence of controlled studies. *Biol. Psychiat.* 12: 687-695.

Barton, J. L., and Snaith, R. P. (1974). In defense of ECT. *World Med.* 10:12.

Beresford, H. R. (1971). Legal issues relating to electroconvulsive therapy. *Arch. Gen. Psychiat.* 25: 100-102.

Bidder, T. G., Strain, J. J., and Brunschwig, L. (1970). Bilateral and unilateral ECT: Follow-up study and critique. *Am. J. Psychiat.* 127: 737-745.

Brody, M. B. (1944). Prolonged memory defects following electrotherapy. *J. Mental Sci.* 90: 777-779.

Brunschwig, L., Strain, J. J., and Bidder, T. G. (1971). Issues in the assessment of post-ECT memory changes. *Brit. J. Psychiat.* 119: 73-74.

Costello, C. G., Belton, G. P., Abra, J. C., and Dunn, B. E. (1970). The amnesic and therapeutic effects of bilateral and unilateral ECT. *Brit. J. Psychiat.* 116: 69-78.

Cronholm, B., and Blomquist, C. (1959). Memory disturbances after electroconvulsive therapy. 2. Conditions one week after a series of treatments. *Acta Psychiat. Scand.* 34: 18-25.

Cronholm, B., and Molander, L. (1957). Memory disturbances after electroconvulsive therapy. I. Conditions 6 hours after electroshock treatments. *Acta Psychiat. Scand.* 32: 280-306.
Cronholm, B., and Molander, L. (1964). Memory disturbances after electroconvulsive therapy. 5. Conditions one month after a series of treatments. *Acta Psychiat. Scand.* 40: 212-216.
Cronholm, B., and Ottoson, J. O. (1961). Memory functions in endogenous depression. Before and after electroconvulsive therapy. *Arch. Gen. Psychiat.* 5: 193-199.
Cronholm, B., and Ottoson, J. O. (1963). The experience of memory function after electroconvulsive therapy. *Brit. J. Psychiat.* 109: 251-258.
Cronin, D., Bodley, P., Potts, L., Mather, M. D., Gardner, R. K., and Tobin, J. C. (1970). Unilateral and bilateral ECT: A study of memory disturbance and relief from depression. *J. Neurol. Neurosurg. Psychiat.* 33: 705-713.
d'Agostino, A. M. (1975). Depression: Schism in contemporary psychiatry. *Am. J. Psychiat.* 132: 629-632.
Davis, J. M. (1965). Efficacy of tranquilizing and antidepressant drugs. *Arch. Gen. Psychiat.* 13: 552-572.
Davis, J. (1978). Efficacy of unilateral ECT. Paper read at NIMH-sponsored conference: "ECT: Efficacy and Impact," New Orleans, Louisiana (proceedings in press).
d'Elia, G., and Raotma, H. (1975). Is unilateral ECT less effective than bilateral ECT? *Brit. J. Psychiatry* 126: 83-89.
Dornbush, R. L. (1972). Memory and induced convulsions. *Semin. Psychiat.* 4: 47-54.
Editorial (1977). Electroconvulsive therapy. *Lancet* 2: 593-594.
Fink, M. (1974). Induced seizures and human behavior, in *Psychobiology of Convulsive Therapy*, Fink, M., Kety, S., McGaugh, J., *et al.* (eds.), V. H. Winston and Sons, Washington, D.C., pp. 1-17.
Fink, M. (1976). Antipsychiatrists and ECT. *Brit. Med. J.* 1(6004): 280.
Fink, M. (1977). Myths of "shock therapy." *Am. J. Psychiat.* 134: 991-996.
Fink, M. (1978). Efficacy and safety of induced seizures (EST) in man. *Compr. Psychiat.* 19: 1-18.
Fink, M. (1979). *Convulsive Therapy: Theory and Practice*, Raven Press, New York.
Frankel, F. (1977). Current perspectives on ECT: A discussion. *Am. J. Psychiat.* 134: 1014-1019.
Freeman, D. P. L., Weeks, D., and Kendell, R. E. (1980). ECT: II. Patients who complain. *Brit. J. Psychiat.* 137: 17-25.
Friedberg, J. (1975). Electroconvulsive therapy: Let's stop blasting the brain. *Psychology Today* 9: 18-23.
Friedberg, J. (1976). *Shock Treatment Is Not Good for Your Brain*, Glide Publications, San Francisco.
Friedberg, J. (1976). Shock treatment, brain damage and memory loss: A neurological perspective. *Am J. Psychiat.* 134: 1010-1014.
Friedman, A. S. (1964). Minimal effects of severe depression on cognitive functioning. *J. Abnormal Soc. Psychol.* 69: 237-243.
Furlong, F. W. (1972). The mythology of electroconvulsive therapy. *Compr. Psychiat.* 13: 235-239.
Glassman, A. H., Kantor, S. J., and Shostak, M. (1975). Depression, delusions and drug response. *Am. J. Psychiat.* 132: 716-719.
Goldman, H., Gomer, F. E., and Templer, D. I. (1972). Long-term effects of electroconvulsive therapy upon memory and perceptual-motor performance. *J. Clin. Psychol.* 28: 32-34.
Greenblatt, M. (1977). Efficacy of ECT in affective and schizophrenic illness. *Am. J. Psychiat.* 134: 1001-1005.
Guze, S. B., and Robins, E. (1970). Suicide and primary affective disorder. *Brit. J. Psychiat.* 117: 437-438.
Halliday, A. M., Davison, K., Browne, M. W., and Kreeger, L. C. (1968). A comparison of the effects on depression and memory of bilateral E.C.T. and unilateral E.C.T. to the dominant and non-dominant hemisphere. *Brit. J. Psychiat.* 114: 997-1012.

Heaton, R. K., Baade, L. E., and Johnson, K. L. (1978). Neuropsychological test results associated with psychiatric disorders in adults. *Psychol. Bull.* 85: 141-162.
Hetherington, R. (1956). The effects of E.C.T. on the efficiency and retentivity of depressed patients. *Brit. J. Med. Psychol.* 29: 258-269.
Hordern, A. (1965). The antidepressant drugs. *New Engl. J. Med.* 272: 1159-1169.
Hurwitz, T. D. (1974). Electroconvulsive therapy: A review. *Compr. Psychiat.* 15: 303-314.
Huston, P. E., and Locher, L. M. (1948a). Involutional psychosis. Course when untreated and when treated with electric shock. *Arch. Neurol. Psychiat.* 59: 385-394.
Huston, P. E., and Locher, L. M. (1948b). Manic-depressive psychosis. Course when untreated and when treated with electric shock. *Arch. Neurol. Psychiat.* 60: 37-48.
Huston, P. E., and Strother, C. T. (1948). The effect of electric shock on mental efficiency. *Am J. Psychiat.* 104: 707-712.
Inglis, J. (1969). Electrode placement and the effect of E.C.T. on mood and memory in depression. *Can. Psychiat. Assoc. J.* 14: 463-471.
Inglis J. (1970). Shock, surgery and cerebral asymmetry. *Brit. J. Psychiat.* 117: 143-148.
Jackson, B. (1978). The effects of unilateral and bilateral ECT on verbal and visual spatial memory. *J. Clin. Psychol.* 34: 4-13.
Janis, I. L. (1950). Psychologic effects of electric convulsive treatments. (I. Post-treatment amnesias.) *J. Nervous Mental Disease* 111: 359-382.
Janis, I. L., and Astrachan, M. (1951). The effects of electroconvulsive treatment on memory efficiency. *J. Abnormal Soc. Psychol.* 46: 501-511.
Jones, J. E. (1974). Non-ECT. *World Med.* 10: 24.
Kahn, R. L., Sarit, S. H., Hilbert, N. M., and Niederehe, M. A. (1975). Memory complaint and impairment in the aged. The effect of depression and altered brain function. *Arch. Gen. Psychiat.* 32: 1569-1573.
Kalinowsky, L. (1967). The convulsive therapies, in *Comprehensive Textbook of Psychiatry*, Freeman, A. N., and Kaplan, H. I. (eds.), Williams and Wilkinson Co., Baltimore, pp. 1279-1285.
Korin, H., Fink, M., and Kwalwasser, S. (1956). Relation of changes in memory and learning to improvement in electroshock. *Confin. Neurol.* 16: 88-96.
Lancaster, N. P., Steinert, R. P., and Frost, I. (1958). Unilateral electroconvulsive therapy. *J. Mental Sci.* 104: 221-227.
Levy, N. A., Serota, H. M., and Grinker, R. E. (1942). Disturbances in brain function following convulsive shock therapy: Electroencephalographic and clinical studies. *Arch. Neurol. Psychiat.* 47: 1009-1029.
Luborsky, L. B. (1948). Psychometric changes during electric shock treatment. *J. Nervous Mental Disease* 107: 531-536.
Matthew, J. R., and Constan, E. (1964). Complications following ECT over a three-year period in a state institution. *Am. J. Psychiat.* 120: 1119-1120.
Medical Research Council (1965). Report by Clinical Psychiatry Committee. Clinical trial of the treatment of depressive illness. *Brit. Med. J.* 1: 881-886.
Medlicott, R. W. (1948). Convulsive therapy. Results and complications in four hundred cases. *New Zealand Med. J.* 47: 338-348.
Michael, S. T. (1954). Impairment of mental function during electric convulsive therapy. *Arch. Neurol. Psychiat.* 71: 362-366.
Miller, E. (1970). The effect of ECT on memory and learning. *Brit. J. Med. Psychol.* 43: 57-62.
Miura, E., Okada, M., and Masao, O. (1960). Retrogade amnesia provoked by electroconvulsive therapy. *Dis. Nerv. Syst.* 21: 649-650.
Norman, J., and Shea, J. T. (1946). Three years' experience with electric convulsive therapy. *New Engl. J. Med.* 234: 857-860.
Ottoson, J. O. (1959). Experimental studies of memory impairment after electroconvulsive therapy. *Acta Psychiat. Scand. Suppl.* 145: 103-131.
Ottoson, J. O. (1968). Psychological or physiological theories of ECT. *Int. J. Psychiat.* 5: 170-174.
Pascal, G. R., and Zeaman, J. B. (1951). Measurement of some effects of electroconvulsive therapy on the individual patient. *J. Abnormal Soc. Psychol.* 46: 104-115.

Perlson, J. (1945). Psychologic studies on a patient who received two hundred and forty-eight shock treatments. *Arch. Neurol. Psychiat.* 54: 409-411.
Pitts, F. N. (1972). Medical aspects of ECT. *Sem. Psychiat.* 4: 27-32.
Practising Psychiatrist (1965). The experience of electro-convulsive therapy. *Brit. J. Psychiat.* 111: 365-367.
Pribram, K. (1974). Interview. *American Psychological Association Monitor* 9-10 (Sept.-Oct.).
Rabin, A. (1948). Patients who received more than one hundred electric shock treatments. *J. Pers.* 17: 42-48.
Regenstein, Q. R., Muroski, B. J., and Engle, R. P. (1975). A case of prolonged reversible dementia associated with abuse of electro-convulsive therapy. *J. Mental Dis.* 161: 200-203.
Rosenthal, R. (1966). *Experimenter Effects in Behavioral Research*, Appleton-Century-Crofts, New York.
Roueche, B. (1974). Annals of medicine: As empty as Eve. *New Yorker* 50(29): 84-100.
Royal College of Psychiatrists (1977). Memorandum on the use of electroconvulsive therapy. *Brit. J. Psychiat.* 131: 261-272.
Schwartzman, A. E., and Termansen, P. E. (1967). Intensive electro-convulsive therapy: A follow-up study. *Can. Psychiat. Assoc. J.* 12: 217-218.
Small, I. F. (1974). Inhalant convulsive therapy, in *Psychobiology of Convulsive Therapy*, Fink, M., Key, S., McGaugh, J., *et al.* (eds.), V. H. Winston, Washington, D.C., pp. 63-77.
Smith, L. H., Hastings, D. W., and Hughes, J. (1943). Immediate and follow-up results of electroshock therapy. *Am. J. Psychiat.* 100: 351-354.
Squire, L. R. (1974). Amnesia for remote events following electroconvulsive therapy. *Behav. Biol.* 12: 119-125.
Squire, L. R. (1975). A stable impairment in remote memory following electroconvulsive therapy. *Neuropsychologia* 12: 51-58.
Squire, L. R. (1977). ECT and memory loss. *Am. J. Psychiat.* 134: 997-1001.
Squire, L. R., and Chace, P. M. (1975). Memory functions six to nine months after electro-convulsive therapy. *Arch. Gen. Psychiat.* 32: 1557-1564.
Squire, L. R., and Miller, P. L. (1974). Diminution of anterograde amnesia following electro-convulsive therapy. *Brit. J. Psychiat.* 125: 490-495.
Squire, L. R., and Slater, P. C. (1975). Forgetting in very long-term memory as assessed by an improved questionnaire technique. *J. Exptl. Psychol.: Human Learning and Memory* 104: 50-54.
Squire, L. R., and Slater, P. C. (1978). Bilateral and unilateral ECT: Effects on verbal and non-verbal memory. *Am. J. Psychiat.* 134: 1316-1320.
Squire, L. R., Slater, P. D., and Chace, P. M. (1975). Retrograde amnesia: Temporal gradient in very long-term memory following electroconvulsive therapy. *Science* 187: 77-79.
Squire, L. R., Chace, P. M., and Slater, P. C. (1976a). Retrograde amnesia following electro-convulsive therapy. *Nature* 260: 775-777.
Squire, L. R., Slater, P. C., and Chace, P. M. (1976b). Anterograde amnesia following electro-convulsive therapy. No evidence for state-dependent learning. *Behav. Biol.* 17: 31-41.
Squire, L. R., Slater, P. C., and Miller, P. L. (1981). Retrograde amnesia and bilateral electro-convulsive therapy. *Arch. Gen. Psychiat.* 38: 95.
Sternberg, D. E., and Jarvik, M. E. (1976). Memory functions in depression. *Arch. Gen. Psychiat.* 33: 219-224.
Stieper, D. R., Williams, M., and Duncan, C. P. (1951). Changes in impersonal and personal memory following electro-convulsive therapy. *J. Clin. Psychol.* 7: 361-366.
Stone, C. P. (1947). Losses and gains in cognitive functions as related to electro-convulsive shocks. *J. Abnormal Soc. Psychol.* 42: 206-214.
Stone, C. P. (1950). Pre-illness test records compared with performance during and after electro-convulsive shocks. *J. Abnormal Soc. Psychol.* 45: 154-159.
Strain, J. J., Brunschwig, L., Duffy, J. P., Agle, D. P., Rosenbaum, D. L., and Bidder, T. G. (1968). Comparison of therapeutic effects and memory changes with bilateral and unilateral ECT. *Am. J. Psychiat.* 125: 294-304.

Templer, D. I., Ruff, C. T., and Armstrong, G. (1973). Cognitive functioning and degree of psychosis in schizophrenics given many electroconvulsive treatments. *Brit. J. Psychiat.* 123: 441-443.
Turek, I. S., and Hanlon, T. E. (1977). The effectiveness and safety of electroconvulsive therapy (ECT). *J. Nervous Mental Disease* 164: 419-431.
Tyler, E. A., and Lowenback, H. (1947). Polydiurnal electric shock treatment in mental disorders. *North Carolina Med. J.* 8: 577-582.
Wechsler, H., Grosser, G. H., and Greenblatt, M. (1965). Research evaluating antidepressant medications on hospitalized mental patients: A survey of published reports during a five-year period. *J. Nervous Mental Disease* 141: 231-239.
Weeks, D., Freeman, D. P. L., and Kendell, R. E. (1980). ECT III: Enduring cognitive deficits? *Brit. J. Psychiat.* 137: 26-37.
Williams, M. (1968). The measurement of memory in clinical practice. *Brit. J. Clin. Soc. Psychol.* 7: 19-34.
Worchel, P., and Narcisco, J. C. (1950). Electroshock convulsions and memory: The interval between learning and shock. *J. Abnormal Soc. Psychol.* 45: 85-98.