Review article

Acute memory impairment following electroconvulsive therapy

1. Effects of electrical stimulus waveform and number of treatments

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ABSTRACT – The literature concerning acute changes in memory functions following electroconvulsive therapy (ECT) is reviewed. Most studies indicate that low-energy brief-pulse ECT is followed by less amnesia than high-energy sinusoidal ECT. Many studies show that amnestic deficits are exacerbated with increasing treatment number. However, it is unclear whether this exacerbation is related to increased electrical energy typically used to induce threshold seizures in the latter treatments or to more endogenous physiological alterations of the CNS across treatment number. Practical and theoretical implications of these issues are discussed.

Electroconvulsive therapy (ECT) produces both retrograde and anterograde memory impairment that may be minimized by various refinements in ECT technique; unilateral rather than bilateral stimulus electrode placement, threshold rather than suprathreshold electrical stimulation, utilization of a limited number of treatments spaced at least 2–3 days apart, administration of oxygen ensuring more than 90 % arterial saturation, adequate muscular relaxation, and administration of light anesthesia (1, 2, 3). This review and a subsequent one (4) examine results from studies in which memory was assessed following variations in electrical stimulus waveform, treatment number, and electrical stimulus electrode placement. Memory impairment in relation to these latter variables has been the subject of most of the quantitative memory investigations in the ECT literature. A review of ECT-induced *disorientation* in relation to these variables can be found elsewhere (5).

Amnesia and sine versus pulse wave stimulation

Based upon convenience rather than upon any scientific rationale, *Cerletti & Bini* (6) first used standard sinusoidal wall current to induce seizures in humans (7). Later on, *Liberson* (8), *Offner* (9) and others experimented with waveforms having a shorter

phase duration, and found that seizures could be induced with much less energy than was required with unmodified sinusoidal stimulation. Based on the hypothesis that a lowering of stimulus energy might decrease ECT-induced memory impairment (see 10, 11 for data supporting this hypothesis), *Liberson* (12, 13) and others (e.g. 14) advocated the use of low-energy pulse waveforms instead of sinusoidal waveforms for seizure induction.

Seven studies compared memory impairment in the acute period following sine versus pulse ECT (14–20). Three other studies contain information pertinent to the issue of memory impairment following sine versus pulse ECT.

Goldstein et al. (21) examined performance on the Halstead-Reitan Neuropsychological Battery following sine versus pulse stimulation, and found no inter-group difference either 1 day or 3 months post-ECT. In studies of retention in *rats, Docter* (22) found greater impairment following sine than pulse wave ECT, whereas *Spanis & Squire* (23) did not.

Of the seven *human* amnesia studies, five studies report more amnesia following sinusoidal than pulse ECT. However, four of these five studies contain methodological inadequacies that render the results equivocal. These include: failure to establish statistical significance for alleged inter-group amnestic differences (14, 15, 18); vague descriptions of time and method of memory testing (14, 15, 18); confounding of results by postictal confusion (14, 18); failure to specify electrical stimulus parameters (15, 16); confounding of results by administering pulse and sine ECT with unilateral versus bilateral electrode placements, respectively (14); and an inter-group difference in treatment spacing (16). Regarding the latter study, twice as many pulse as sine wave patients had inter-treatment breaks of 4 to 7 days (in-

stead of the usual 2–3 day interval), and the breaks tended to be longer with the pulse ECT group. Because there may be an increase in amnesia with closely spaced seizures (2, 18, 24), the inter-group amnestic difference found in this study may have been due to treatment spacing, not stimulus waveform.

In all four studies, either patients were not oxygenated or oxygenation is unspecified. *Marshall & Dobbs* (25) found greater postictal apnea following sine than pulse ECT. A greater amount of cerebral hypoxia may have occurred following sine than pulse ECT in any of these four studies, and it may have been this difference rather than stimulus waveform *per se* that produced the inter-group amnestic differences (2, 3, 26, 27, 28). In fact, *Epstein & Wender* (15), who reported more amnesia following sine than pulse ECT, noted that cyanosis frequently occurred in their sine wave patients, but was rarely seen in their pulse wave patients.

Three studies (17, 19, 20) contain none of the previous methodological deficiencies. *Weiner et al.* (20) found that performance on a "personal memory" inventory was *not* significantly more impaired following bidirectional sinusoidal than bidirectional brief-pulse ECT (0.75–1.5 msec pulse width), although there was a trend towards significance ($P = 0.08$). *Daniel et al.* (19) found that verbal memory and memory for the episode in which the verbal learning initially occurred (*autobiographical* memory) were *not* more impaired following bidirectional sinusoidal than bidirectional brief-pulse ECT (0.75–1.5 msec pulse width), even though significantly greater electrical energy was delivered with sine wave ECT. However, *Cronholm & Ottosson* (17) found less retrograde amnesia in patients receiving ultrabrief unidirectional square wave stimulation (0.1 msec pulse width) than in pa-

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tients receiving unidirectional quarter sine wave stimulation (5.0 msec wave duration). The sine wave group received, on the average, three times more electrical energy than did the pulse wave group.

In summary, it has not been definitively demonstrated that more amnesia follows sinusoidal than pulse ECT, although existing studies suggest that such an effect may be demonstrated in future investigations. More studies need to be conducted keeping inter-group (sine versus pulse) differences in hypoxia, electrode placement, treatment spacing, and postictal confusion to a minimum. If an amnestic difference does occur after minimizing inter-group differences in these latter variables, it may be concluded that stimulus waveform alters ECT-induced amnesia.

However, clinical observations from several studies suggest that such an amnestic difference may be related to differences in degree of seizure generalization rather than, or in addition, to differences in total electrical energy. *Cronholm & Ottosson* (17) noted that, following ultrabrief pulse stimulation, some patients started breathing before seizure termination, and some patients regained consciousness soon after seizure termination. These incidents did not occur with quarter sine wave ECT. Other investigators report that both breathing and regainment of consciousness (with some preservation of memory for the experience) *during* seizures have occurred with pulse ECT (12, 13, 29, 30), particularly when very low-energy pulse stimuli have been used (pulse-widths 0.3 msec or less). Furthermore, *Marshall & Dobbs* (25) noted that the intensity of tonic-clonic movements seemed to be less with pulse (0.3–0.7 msec pulse-width) than with bidirectional sine wave ECT.

Taken collectively, these observations suggest that sinusoidal ECT may produce more highly generalized seizures than

pulse ECT (2). *Weiner et al.* (20) recently reported that more EEG slowing (2–3 days post-ECT) followed sinusoidal than brief-pulse ECT, a result which is consistent with this hypothesis, since one would expect more EEG slowing following more completely generalized seizures.

Inter-group differences in seizure generalization may be as important or more important than total electrical energy *per se* in explaining amnestic differences between sinusoidal and pulse ECT (see 17). To determine which is the more important amnesia-inducing variable, a study is needed that measures differences in seizure generalization (using multi-channel EEG techniques) and electrical energy between sinusoidal and pulse ECT. An appropriate statistical analysis (e.g. analysis of covariance) could then be performed to determine the relative contributions of these two variables to amnestic differences between sine and pulse ECT.

Amnesia in relation to number of treatments

It has also been suggested that the total number of treatments has some bearing on the degree of ECT-induced amnesia (e.g. 31, 32). Of 16 reviewed studies, 13 provide evidence that amnesia *increases* across treatment number (16, 20, 33–43). Two studies provide evidence of *decreased* amnesia across treatment number (44, 45). One study provides evidence of *unchanged* amnesia across treatment number (46).

Regarding the latter three studies, *Cronin* (45) administered the *same form* of two tests (the Modified Word-Learning Test and part of the Wechsler Memory Scale) 1 h after treatments one, six and eight. Their finding of decreased amnesia across treatment number may simply be the result of confounding practice effects. Another explanation is related to the find-

ing that seizure duration decreases across treatment number, with the first seizure in a series being longer than subsequent ones (26, 47–50). Amnesia (and other CNS disturbance) may *increase* with increasing seizure duration (2, 10), and a decrease in seizure duration across treatment number may be followed by *decreased* amnesia. *Cronholm & Lagergren* (44), who also found *decreased* amnesia across treatments (comparing retrograde amnesia for a number presented within a minute before treatments one and four), discuss this hypothesis as a possible explanation of their finding.

In another study (46), patients were shown pictures of common objects (e.g. a shoe) after treatments two or three (randomly determined) and after treatment six. Patients were tested on the original items in a recognition format with other unfamiliar objects after either a 0 min (immediate reproduction), 5 min, 20 min, or 60 min delay. *Zinkin & Birtchnell* found no significant increase in amnesia across treatment number, and suggest that their recognition test may have been insensitive to cumulative ECT-induced amnesia.

Squire & Miller (42) showed patients eight items after treatments one and four, and tested recognition memory for these items 30 min and 24 h later. On the 30 min delayed retention interval, they found no difference across treatment number. This finding is consistent with *Zinkin & Birtchnell's* results. Cumulative memory deficits across treatment number (on recognition tests) may not be demonstrable with delayed retention intervals of 5 to 60 min. With *Squire & Miller's* 24-h delayed retention interval, however, there was a statistically significant increase in forgetting across treatments one and four. One explanation of this latter finding is that it may take a longer retention interval to demonstrate forgetting (51) with increasing treatment number.

Theoretical note

The majority of the 16 reviewed studies support the notion that amnesia increases across treatment number. However, it is not clear that this increase is primarily related to endogenous physiological alterations of the CNS across treatment number.

The threshold amount of electrical energy needed to induce a tonic-clonic seizure usually increases across treatment number (26, 47, 52–56). It may therefore be necessary to increase the electrical stimulus energy across treatments to elicit a fully generalized seizure. However, *increased* amnesia across treatment number may now be caused by the increased electrical energy (10, 11), rather than by more endogenous physiological alterations of the CNS across treatment number.

If the increase in amnesia is the result of endogenous physiological alterations, important questions are raised about the relationship of *postictal* amnesia to *interictal* (the period between seizures, after the postictal period has cleared) and *post-treatment* (the period after the course of ECT, after the last postictal period has cleared) amnesia. Amnesia existing in the interictal or post-treatment periods may simply be the cumulative prolongation of postictal symptomatology (32). Conversely, persisted amnesia may be qualitatively and/or quantitatively different, on a neuropsychological or neurophysiological level, from postictal amnesia.

Unfortunately, the experiments needed to test either hypothesis have not been performed to date. Serial studies of amnesia conducted along with other neurological and neuropsychological tests that may be sensitive to postictal impairment

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(e.g. The Trailmaking Test, vigilance tests, EEG) need to be performed beginning soon after seizure termination. If memory recovery curves follow or parallel the recovery curves of these or other tests operationally defined as measuring postictal symptomatology, one may conclude that persistent ECT-induced amnesia is simply prolonged postictal impairment. *Lipowski* (57) noted that specific neurological and cognitive functions may be differentially impaired (in degree and uniformity) in acute confusional states, such as the postictal state following ECT. Following ECT, some cognitive and neurological functions (e.g. memory, EEG) probably recover slower than others (e.g. orientation), but may still be part of postictal symptomatology. The exact time-course of recovery of these various impairments is an empirical question that is open to further investigation.

Practical implications

Methodological deficiencies aside, the majority of the reviewed studies suggest that low-energy brief-pulse ECT is followed by less amnesia than high-energy sinusoidal ECT. The continued utilization of brief-pulse stimuli, which can induce a seizure with one-half or less the amount of electrical energy required with sine wave stimuli (19, 58, 59), therefore appears warranted in clinical settings, especially since markedly suprathreshold electrical stimulation has been shown to increase memory dysfunction (10, 11).

This utilization is further warranted because more EEG slowing (20) and disorientation (18) have been reported following sine than pulse ECT, and because the two treatment modalities had similar anti-depressive efficacy in recent studies (58, 60, 61). However, pulse stimuli with pulse-widths below 0.6 milliseconds may

produce "submaximal" or incompletely generalized seizures, which may have low anti-depressive efficacy (62). There may therefore be a lower limit to which electrical stimulus parameters may be decreased to reduce amnestic side-effects (3).

Regarding treatment number, retrograde and anterograde amnesia following each treatment in a series tends to increase, so that more or less continuous memory impairment may be noted following the latter treatments in the series (3). Furthermore, EEG slowing confusion, and other cognitive impairment tend to increase with increasing treatment number (36, 63–67). These deficits may be greater in severity or duration if treatments are spaced more frequently than 2–3 times per week (2). Because even a transient build-up of these deficits is undesirable, some investigators (e.g. 3) have suggested that no more treatments than are necessary for a remission of depressive symptomatology be given (6–10), and that intervals of at least 2–3 days between treatments be utilized.

References

1. d'Elia G. Unilateral electroconvulsive therapy. In: Fink M, Kety S, McGaugh J, Williams T, eds. Psychobiology of convulsive therapy. Washington: V. H. Winston & Sons, 1974:21–34.
2. Fink M. Convulsive therapy: theory and practice. New York: Raven Press, 1979:1–308.
3. Ottosson J O. Convulsive therapy. In: Kisker K P, Meyer J E, Müller C, Strömgren E, eds. Psychiatrie der Gegenwart (vol. I/2, 2nd ed.). Berlin: Springer-Verlag, 1980:315–349.
4. Daniel W F, Crovitz H F. Acute memory impairment following electroconvulsive therapy. 2. Effects of electrode placement. Acta Psychiatr Scand 1983:67:In press.
5. Daniel W F, Crovitz H F. Recovery of orienta-

tion after electroconvulsive therapy. Acta Psychiatr Scand 1982;66:421-428.

6. Cerletti U, Bini L. Un nuevo metodo di shockterapie "L'elettro-shock". Boll Acad Med Roma 1938;64:136-138.

7. Weiner R D. The psychiatric use of electrically induced seizures. Am J Psychiatry 1979;136:1507-1517.

8. Liberson W T. New possibilities in electric convulsive therapy: "Brief stimuli" technique. Preliminary report. Dig Neurol Psychiatry 1944;12:368-369.

9. Offner F. Stimulation with minimum power. J Neurophysiol 1946;9:387-390.

10. Ottosson J O. Experimental studies of memory impairment after electroconvulsive therapy. The role of the electrical stimulation and of the seizure studied by variation of stimulus intensity and modification by lidocaine of seizure discharge. Acta Psychiatr Neurol Scand 1960:Suppl. 145:103-133.

11. Cronholm B, Ottosson J O. "Countershock" in electroconvulsive therapy: Influence on retrograde amnesia. Arch Gen Psychiatry 1961;4:254-258.

12. Liberson W T. Brief stimulus therapy: physiological and clinical observations. Am J Psychiatry 1948;105:28-39.

13. Liberson W T. Current evaluation of electric convulsive therapy: correlation of the parameters of electric current with physiological and psychologic changes. Res Publ Assoc Nerv Ment Dis 1953;31:199-231.

14. Medlicott R W. Brief stimuli electroconvulsive therapy. N Z Med J 1948;47:29-37.

15. Epstein J, Wender L. Alternating current vs. unidirectional current for electroconvulsive therapy – comparative studies. Confin Neurol 1956;16:137-146.

16. Kendall B S, Mills W B, Thale T. Comparison of two methods of electroshock in their effect on cognitive functions. J Consult Psychol 1956;20:423-429.

17. Cronholm B, Ottosson J O. Ultrabrief stimulus technique in electroconvulsive therapy: 1. Influence on retrograde amnesia of treatments with the Elther ES electroshock apparatus, Siemens Konvulsator III and of lidocaine-modified treatment. J Nerv Ment Dis 1963;137:117-123.

18. Valentine M, Keddie M G, Dunne D. A comparison of techniques in electro-convulsive therapy. Br J Psychiatry 1968;114:989-996.

19. Daniel W F, Crovitz H F, Weiner R D, Rogers H J. The effects of ECT modifications on autobiographical and verbal memory. Biol Psychiatry 1982;17:919-924.

20. Weiner R D, Rogers H J, Davidson J, Miller R D. Evaluation of the central nervous system risks of ECT. Psychopharmacol Bull 1982;18:29-31.

21. Goldstein S G, Filskov S B, Weawer L A, Ives J O. Neuropsychological effects of electroconvulsive therapy. J Clin Psychol 1977;33:798-806.

22. Docter R F. The effect of electroconvulsive shock (ECS) vs. "brief stimulus therapy" (BST) on

memory and nest building in albino rats. J Comp Physiol Psychol 1957;50:100-104.

23. Spanis C W, Squire L R. Memory and convulsive stimulation: effects of stimulus waveform. Am J Psychiatry 1981;138:1177-1181.

24. Glueck B C, Reiss H, Bernard L E. Regressive electric shock therapy. Psychiatr Q 1957;31:117-136.

25. Marshall T J, Dobbs D. Treatment technique and apnea in electroshock. Dis Nerv Syst 1959;20:582-583.

26. Holmberg G. The effect of certain factors on the convulsions in electric shock treatment. Acta Psychiatr Neurol Scand 1955:Suppl. 8:1-19.

27. Blachly P, Gowing D. Multiple monitored electroconvulsive treatment. Comprehens Psychiatry 1966;7:100-109.

28. Abrams R. Multiple ECT: What have we learned? In: Fink M, Kety S, McGaugh J, Williams T, eds. Psychobiology of convulsive therapy. Washington: V. H. Winston & Sons, 1974:79-84.

29. Gayle R F, Josephs D J. Brief stimulus therapy. South Med J 1948;41:245-251 (see discussion by Anderson J L, p. 250).

30. Goldman D. Brief stimulus electric shock therapy. J Nerv Ment Dis 1949;110:36-45.

31. Stone C P. Losses and gains in cognitive functions as related to electroconvulsive shocks. J Abnorm Soc Psychol 1947;42:206-214.

32. Hargreaves W A, Fischer A, Elashoff R M, Blacker K H. Delayed onset of impairment following electrically induced convulsions. Acta Psychiatr Scand 1972;48:69-77.

33. Glueck B C. Psychopathologic reactions and electric shock therapy. N.Y. State J Med 1942;42:1553-1557.

34. Wilcox K W. Intellectual functioning as related to electroconvulsive therapy. Univ. of Michigan: unpublished doctoral dissertation. 1954:1-196.

35. Brengelmann J C. The effect of repeated electrical shocks on learning (in depression). Monogr Gesamtgeb Neurol Psychiatr (Berlin) 1959;84:1-52.

36. Wilson I, Gottlieb G. Unilateral electro-convulsive shock therapy. Dis Nerv Syst 1967;28:541-545.

37. Costello C G, Belton G P, Abra J C, Dunn B E. The amnesic and therapeutic effects of bilateral and unilateral ECT. Br J Psychiatry 1970;116:69-78.

38. Miller E. The effect of ECT on memory and learning. Br J Med Psychol 1970;43:57-62.

39. Goldman H, Gomer F E, Templer D I. Long-term effects of electroconvulsive therapy upon memory and perceptual-motor performance. J Clin Psychol 1972;28:32-34.

40. Squire L R. Amnesia for remote events following electroconvulsive therapy. Behav Biol 1974;12:119-125.

41. Squire L R. A stable impairment in remote memory following electroconvulsive therapy. Neuropsychologia 1975;13:51-58.

42. Squire L R, Miller P L. Diminution of anterograde amnesia following electroconvulsive therapy. Br J Psychiatry 1974;125:490-495.

43. Reichert H, Benj risson G. Bilater ECT. Part II – Can Psychiatr As

44. Cronholm B, Lag after electroconvu study of retrogra sive shock. Acta

45. Cronin D, Bodley R, Tobin J. Unila of memory distu sion. J Neurol N 713.

46. Zinkin S, Birtch sive therapy: Its peutic efficacy. B

47. Finner R W. Di shock therapy. J

48. Green M A. Rel ration of seizures ing convulsive th 131:117-120.

49. Pollack M, Rose electroshock con seizures. Exp Ne

50. Small J G, Sma siology of EST. Killam K F, ed eration of prog 1978:759-769.

51. Cronholm B, Mc after electroconv after electroshoc Scand 1957;32:28(

52. Toman J E P, C fying convulsion: Nerv Ment Dis 1

53. Weil P J. "Reg phrenics. J Ment

54. Woodbury L A, of a new electr analysis of facto patterns. Arch I 97-107.

55. Brockman R J, Gleser G C, U threshold as rela Neurol 1956;16:9

56. Essig C F. Freq sions and the ac response. Exp Ne

57. Lipowski Z J. Or and classification

albino rats. J Comp
104.

mory and convulsive
his waveform. Am J

ard L E. Regressive
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tment technique and
erv Syst 1959:20:582–

ertain factors on the
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Suppl. 8:1–19.

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augh J, Williams T,
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:206–214.

A, Elashoff R M,
t of impairment fol-
onvulsions. Acta Psy-

logic reactions and
. State J Med 1942:

inctioning as related
Univ. of Michigan:
ition. 1954:1–196.

: of repeated electri-
depression). Monogr
(Berlin) 1959:84:1–52.

iteral electro-convul-
Syst 1967:28:541–545.

bra J C, Dunn B E.
: effects of bilateral
ychiatry 1970:116:69–

CT on memory and
970:43:57–62.

Templer D I. Long-
ilsive therapy upon
tor performance. J

iote events following
iehav Biol 1974:12:

airment in remote
ilsive therapy. Neu-

minution of antero-
troconvulsive thera-
90–495.

43. Reichert H, Benjamin J, Neufeldt A H, Marjer-
risson G. Bilateral and non-dominant unilateral
ECT. Part II – development of prograde effects.
Can Psychiatr Assoc J 1976:21:79–86.

44. Cronholm B, Lagergren A. Memory disturbances
after electroconvulsive therapy: an experimental
study of retrograde amnesia after electroconvul-
sive shock. Acta Psychiatr Scand 1959:34:283–310.

45. Cronin D, Bodley P, Potts L, Mather M, Gardner
R, Tobin J. Unilateral and bilateral ECT: a study
of memory disturbance and relief from depres-
sion. J Neurol Neurosurg Psychiatry 1970:33:705–
713.

46. Zinkin S, Birtchnell J. Unilateral electroconvul-
sive therapy: Its effects on memory and its thera-
peutic efficacy. Br J Psychiatry 1968:114:973–988.

47. Finner R W. Duration of convulsion in electric
shock therapy. J Nerv Ment Dis 1954:119:530–537.

48. Green M A. Relation between threshold and du-
ration of seizures and electrographic change dur-
ing convulsive therapy. J Nerv Ment Dis 1960:
131:117–120.

49. Pollack M, Rosenthal F, Macey R. Changes in
electroshock convulsive response with repeated
seizures. Exp Neurol 1963:7:98–106.

50. Small J G, Small I F, Milstein V. Electrophy-
siology of EST. In: Lipton M A, DiMascio A,
Killam K F, eds. Psychopharmacology: a gen-
eration of progress. New York: Raven Press
1978:759–769.

51. Cronholm B, Molander L. Memory disturbances
after electroconvulsive therapy: conditions 6 hours
after electroshock treatment. Acta Psychiatr
Scand 1957:32:280–306.

52. Toman J E P, Goodman L S. Conditions modi-
fying convulsions in animals. Res Publ Assoc
Nerv Ment Dis 1947:26:141–162.

53. Weil P J. "Regressive" electroplexy in schizo-
phrenics. J Ment Sci 1950:96:514–520.

54. Woodbury L A, Davenport V D. Design and use
of a new electroshock seizure apparatus, and
analysis of factors altering seizure threshold and
patterns. Arch Int Pharmacodyn Ther 1952:92:
97–107.

55. Brockman R J, Brockman J C, Jacobsohn U,
Gleser G C, Ulett A. Changes in convulsive
threshold as related to type of treatment. Confin
Neurol 1956:16:97–104.

56. Essig C F. Frequency of repeated electroconvul-
sions and the acquisition-rate of a tolerance-like
response. Exp Neurol 1969:25:571–574.

57. Lipowski Z J. Organic brain syndromes: overview
and classification. In: Benson D F, Blumer D,

eds. Psychiatric aspects of neurologic disease.
New York: Grune & Stratton, 1975:11–35.

58. Weaver L A, Ives J O, Williams R, Nies A.
A comparison of standard alternating current and
low-energy brief-pulse electrotherapy. Biol Psy-
chiatry 1977:12:525–543.

59. Weiner R D. ECT and seizure threshold: effects
of stimulus wave form and electrode placement.
Biol Psychiatry 1980:15:225–241.

60. Carney M W P, Sheffield B F. The effects of
pulse ECT in neurotic and endogenous depres-
sion. Br J Psychiatry 1974:125:91–94.

61. Welch C A, Weiner R D, Weir D et al. Efficacy
of ECT in the treatment of depression: wave
form and electrode placement considerations. Psy-
chopharmacol Bull 1982:18:31–34.

62. Cronholm B, Ottosson J O. Ultrabrief stimulus
technique in electroconvulsive therapy. 2. Com-
parative studies of therapeutic effects and memo-
ry disturbances in treatment of endogenous de-
pression with the Elther ES electroshock appa-
ratus and Siemens Konvulsator III. J Nerv Ment
Dis 1963:137:268–276.

63. Pacella B L, Barrera S E, Kalinowsky L. Varia-
tions in EEG associated with EST of patients
with mental disorders. Arch Neurol Psychiatr
1942:47:367–384.

64. Mosovich A, Katzenelbogen S. Electroshock ther-
apy, clinical and EEG studies. J Nerv Ment Dis
1948:107:517–530.

65. Roth M. Changes in the EEG under barbiturate
anaesthesia produced by ECT and their signifi-
cance for the theory of ECT action. EEG Clin
Neurophysiol 1951:3:261–280.

66. Templer D I, Ruff C F, Armstrong G. Cognitive
functioning and degree of psychosis in schizo-
phrenics given many electroconvulsive treatments.
Br J Psychiatry 1973:123:441–443.

67. Regestein Q R, Murawski B J, Engle R P. A
case of prolonged reversible dementia associated
with abuse of ECT. J Nerv Ment Dis 1975:161:
200–203.

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