ELECTROCONVULSIVE THERAPY

Consensus Development Conference June 10-12, 1985

Masur Auditorium Warren Grant Magnuson Clinical Center National Institutes of Health

Sponsored by the National Institute of Mental Health and the NIH Office of Medical Applications of Research

Contents

Introduction to Electroconvulsive Therapy	
Consensus Development Conference	1
Agenda	5
Consensus Panel	9
Speakers	1
Planning Committee	13
Abstracts	15
The Use of Somatic Treatments for Psychiatric Illnesses 1 Gerald L. Klerman, M.D.	17
Convulsive Therapy: How It Evolved	21
ECT: The Historical, Social, and Professional Sources of the Controversy	25
Utilization of ECT in U.S. Psychiatric Facilities, 1970 to 1980	32
Efficacy in Depression: Controlled Trials	37
Efficacy in Depression: ECT Versus Antidepressants	43
Efficacy of ECT in Schizophrenia, Mania, and Other Disorders	16

ECT: Possible Mechanisms of Action	50
Systemic Effects of ECT	55
Neuropathology and Cognitive Dysfunction From ECT	59
Quantitative Neuropathology in Electrically Induced Generalized Convulsions	65
Acute Cognitive Side Effects of ECT	69
The Question of Long-Term Effects	73
Patients' Attitudes Toward ECT	78
Legal Parameters of Informed Consent for ECT Administered to Mentally Disabled Persons	84
Data on Informed Consent for ECT	91
Clinical Strategies in Choice and Timing of ECT	94
General Technique of ECT	97
Electrical Dosage, Stimulus Parameters, and Electrode Placement	100

ECT: Possible Mechanisms of Action
Systemic Effects of ECT
Neuropathology and Cognitive Dysfunction From ECT
Quantitative Neuropathology in Electrically Induced Generalized Convulsions
Acute Cognitive Side Effects of ECT
The Question of Long-Term Effects
Patients' Attitudes Toward ECT
Legal Parameters of Informed Consent for ECT Administered to Mentally Disabled Persons
Data on Informed Consent for ECT
Clinical Strategies in Choice and Timing of ECT
General Technique of ECT
Electrical Dosage, Stimulus Parameters, and Electrode Placement

Neuropathology and Cognitive Dysfunction From ECT

Peter R. Breggin, M.D.

ECT always produces some degree of immediate brain damage and mental dysfunction, and frequently the patient never fully recovers. Permanent brain damage from ECT is demonstrated through clinical evaluations, psychological tests, EEG studies, CAT scans, human autopsy studies, and research on the effect of electrical current on the brain as well as through a variety of animal studies.

In every routine course of ECT, its devastating impact is displayed in the production of an organic brain syndrome, with severe symptoms of trauma to the brain. In its most mild form, the organic brain syndrome takes the form of an amnestic syndrome with loss of both recent and more remote memories. Typically, "apathy, lack of initiative, and emotional blandness are common," and the emotions are "shallow." More commonly, the organic brain syndrome becomes much more severe and takes the form of delirium with global disruption of all mental function, including intellect, judgment, emotional stability, memory, and orientation to time, place, and person. Severe delirium is not uncommon in routine ECT.

The brain-damaged patient tends to confabulate--to deny any mental impairment, even when it is grossly apparent to the observer. This denial of impairment by many ECT patients in the face of obvious mental dysfunction unhappily lends credence to false claims that the treatment is harmless.

Because ECT always produces an organic brain syndrome, the question is not "Does ECT cause brain damage and dysfunction?" ECT always produces brain damage and dysfunction. The proper question is "How complete is recovery from this trauma?" To assume it is routinely complete after electrically induced delirium defies common sense and general medical knowledge. Among body organs, the brain is especially ill-equipped to recover from damage.

During the phase of the acute organic brain syndrome, the impaired condition of the brain is routinely reflected in a disturbed EEG pattern similar to severe chronic epilepsy, toxic states, and other serious brain diseases.

Often this brain wave impairment becomes long lasting and even permanent.^{6,7} Shrinkage of the brain may be apparent on CAT scans.^{3,8,9}

Neuropathologically, the permanent damage can be visualized in human autopsies after modified ECT. $^{10-13}$ Reports show diffuse small hemorrhages throughout the brain, glial proliferation (scarring), and cell death. Extremely careful animal studies have shown similar findings. 3 , 8 , $^14-17$

The worst damage results from the passage of current through the brain and has been directly visualized in animals receiving modified ECT and demonstrated by angiography. Even a very weak current of electricity passing down the blood vessels severely constricts them, cutting off the supply of nutrients and oxygen to the surrounding brain cells, eventually causing vessel wall deterioration, hemorrhage, and cell death. Advocates of ECT, such as Meldrum (1985), 18 must claim that ECT-induced convulsions are theoretically less harmful than spontaneous seizures in epilepsy; these conclusions overlook the damaging effects of the electrical current. ECT combines the brain damage caused by epilepsy with the brain damage caused by electrical trauma.

Cognitively, ECT treatment always produces some degree of permanent memory loss for events surrounding the treatment and frequently produces permanent memory loss reaching back months and years into the past. 3,19 Many cases involve losses that prohibit a return to normal activities in the home or at work. Indeed, there are repeated warnings in the literature against giving ECT to individuals who earn their living through mentally taxing work. 20 ECT can also produce ongoing problems with learning and memorizing new material, with the tragic result that the patient feels permanently defective and disabled. I have described several such desperate cases, 3,8 and many similar reports continue to flow into the Center for the Study of Psychiatry each week.

Tests that examine the most relevant function—the patient's actual memory for past events—always show serious and lasting losses following ECT. 19,21-24 Similarly, when patients are questioned years after ECT, more than 50 percent typically respond with reports of chronic memory difficulties, which they attribute to ECT (Squire, 1982, reports 58 percent; Freeman and Kendall, 1980, report 64 percent). 25,26

Patient self-reports of permanent loss are so frequent that promoters of ECT have tried to argue that the patients have "subjective" memory losses without real or objective losses. ²⁷ But as we have seen, patients with memory defects from brain damage of any kind tend to confabulate and deny--that is, to minimize rather than to exaggerate their defects. ⁴

Squire's personally originated tests using recall for TV shows failed to show large memory losses. 28,29 But these tests are wholly of his own invention and have never been proved useful in detecting brain

damage. In recent years, Squire has placed more emphasis on patient self-reports and on tests that measure the actual loss of personal memories, 24,25 both of which indicate permanent memory loss following ECT.

The modern defense of electroshock often rests on the assertion that "recent" modifications of the treatment have ameliorated its damaging effects. But the most important modification of ECT--the use of anesthetics, muscle paralyzing agents, and artificial respiration with oxygen--is not new at all. As early as 1957 there were multiple reports in the literature of brain death from modified ECT. ¹⁰ I myself administered modified ECT more than 20 years ago! The bad reputation that ECT has among many professionals and many patients, and much of the scientific data indicting ECT as a dangerous therapy, stems from more than 30 years of experience with modified ECT.

Modified ECT of necessity tends to be more damaging than the older methods. The anesthesia used in modified ECT is a sedative that suppresses the ability of the brain to have a seizure. Therefore, higher doses of offending electricity must be used in modified ECT to force a seizure from the patient's brain. 3

Nondominant or unilateral ECT offers us no hope for a safer ECT. The fact that nondominant ECT does not so heavily affect the verbal centers on the left side of the brain makes it more difficult to measure its damaging effects; but this is merely because most of our tests are aimed at verbal memory loss. The nondominant side of the brain deals more with visual memory, musical memory, intuition, integration of knowledge, and creativity. Tests of visual memory find damage following nondominant ECT.²⁴

To assume that any innovations have ameliorated the hazards of ECT remains irresponsible speculation until backed by multiple animal autopsy studies. It is in keeping with traditional medical ethics to ask the profession to ban ECT until animal studies have been conducted to test the unproven and unlikely hypothesis that the newer methods of ECT are relatively harmless.

ECT can never be made harmless. First, enough damage must be done to elicit the convulsion. Second, the damage itself produces the emotional changes—apathy and indifference, and sometimes euphoria—that are labeled an "improvement." Therefore, a relatively inoffensive ECT would be a relatively ineffective ECT. 3,8,30-32 This is consistent with Weiner et al.'s observation that the most "benign" methods of ECT may be "relatively ineffective from a therapeutic standpoint." Thus, the innovations remain unpopular.

The idea that electroshock works by damaging the brain is not unprecedented in psychiatry. Before psychiatry became public-image conscious,

it was commonly claimed that ECT works by damaging the brain and mind and even by killing brain cells. $^{34-36}$

Electroshock victims can best describe the damaging effects of the treatment, and two cases will be described in the patients' own words to illustrate their anguished outcomes.

Informed consent is at the heart of the matter; the potential patient has a right to know about the controversial and dangerous nature of ECT. Kaplan and Sadock, authors of the widely read textbook of psychiatry, recently observed, "ECT remains one of the most controversial methods of treatment in psychiatry." The patient has a right to be informed of this!

REFERENCES

- Daniel WF, Crovitz HF. Disorientation during electroconvulsive therapy: technical, theoretical, and neuropsychological issues. Presented at the International Conference on ECT: Clinical and Basic Research Issues, New York City, January 16-18, 1985
- 2. Brengelman JC. The effect of repeated electroshock on learning in depressives. Berlin: Springer, 1959
- Breggin PR. Electroshock: its brain-disabling effects. New York: Springer, 1979
- 4. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 3rd ed. Washington, DC: American Psychiatric Association, 1980
- 5. Weiner RD, Rogers HJ, Davidson JRT, Kahn EM. Effects of ECT on brain electrical activity. Presented at the International Conference on ECT: Clinical and Basic Research Issues, New York City, January 16-18, 1985
- 6. Mosovitch A, Katzenelbogen S. Electroshock therapy: clinical and electroencephalographic studies. J Nerv Ment Dis 1948;107:517-30
- 7. Volavka J, Feldstein S, Abrams R, et al. EEG and clinical changes after bilateral and unilateral electroconvulsive therapy. Electroencephalogr Clin Neurophysiol 1972;32:631-69
- 8. Breggin PR. Disabling the brain with electroshock. In: Dongier M, Wittkower E, eds. Divergent views in psychiatry. Hagerstown: Harper and Row, 1981
- 9. Calloway SP, Dolan R, Jacoby R, Levy R. ECT and cerebral atrophy: a computed tomography study. Acta Psychiatr Scand 1981;64:442-5

- 10. Impastato D. Prevention of fatalities in electroshock therapy. Dis Nerv Syst 1957; 18(sec 2):34-75
- 11. Grabner HK, McHugh RB. Regressive electroshock therapy in schizophrenia, a controlled study (preliminary report). Lancet 1960; 80:24-7
- 12. McKegney FP, Panzetta AF. An unusual fatal outcome of electroconvulsive therapy. Am J Psychiatry 1963;120:398-400
- 13. Erwin CW, Thompson EM. ECT in schizophrenia: a study in nosological imprecision. In: Brady J, Brodie H, eds. Controversy in psychiatry. Philadelphia: W.B. Saunders, 1978
- 14. Hartelius H. Cerebral changes following electrically induced convulsions. Acta Psychiatr Neurol Scand 1952;77(suppl):1-128
- 15. Ferraro A, Roizen L. Cerebral morphologic changes in monkeys subjected to a large number of electrically induced convulsions. Am J Psychiatry 1949;106:278-84
- 16. Ferraro A, Roizen L, Helford M. Morphological changes in the brain of monkeys following electrically induced convulsions. J Neuropathol Exp Neurol 1946;5:285-308
- 17. Friedberg J. Shock treatment, brain damage, and memory loss: a neurological perspective. Am J Psychiatry 1977;134:1010-4
- 18. Meldrum BS. Neuropathological consequences of chemically and electrically induced seizures. Presented at the International Conference on ECT: Clinical and Basic Research Issues, New York City, January 16-18, 1985
- 19. Janis IL, Astrachan M. The effect of convulsive treatments on memory efficiency. J Abnorm Psychol 1951;46:501-11
- 20. Valentine M, Keddie HMG, Dunne D. A comparison of techniques in electroconvulsive therapy. Br J Psychiatry 1968;114:988-96
- 21. Brunschwig L, Strain J, Bidder TG. Issues in the assessment of post-ECT memory changes. Br J Psychiatry 1971;119:73-4
- 22. Daniel WF, Crovitz HF, Weiner RD, Rogers HF. The effects of ECT modifications on autobiographical and verbal memory. Biol Psychiatry 1982;17:919-24
- 23. Taylor JR, Tomplins R, Demers R, Anderson D. Electroconvulsive therapy and memory dysfunction: is there evidence for prolonged defects? Biol Psychiatry 1982;17:1169-83

- 24. Squire LR. Memory functions as affected by ECT. Presented at the International Conference on ECT: Clinical and Basic Research Issues, New York City, January 16-18, 1985
- 25. Squire LR. Memory and electroconvulsive therapy (letter). Am J Psychiatry 1982;139:1221
- 26. Freeman CPL, Kendell RE. ECT: I. Patients' experiences and attitudes. Br J Psychiatry 1980;137:8-16
- 27. American Psychiatric Association. Task force on electroconvulsive therapy. Washington, DC: American Psychiatric Association, 1978
- 28. Squire LR, Chace PM. Memory functions six to nine months after electroconvulsive therapy. Arch Gen Psychiatry 1975;32:1557-64
- 29. Squire LR. A stable impairment in remote memory following electroconvulsive therapy. Neuropsychologia 1975;13:51-8
- 30. Breggin PR. Psychiatric drugs: hazards to the brain. New York: Springer, 1983
- 31. Breggin PR. Iatrogenic helplessness in authoritarian psychiatry. In: Morgan R, ed. The iatrogenics handbook. Toronto: IPI Publishing, 1983. Reprinted in Morgan R (ed). Electric shock. Toronto: IPI Publishing, 1985
- 32. Breggin PR. Electroshock therapy and brain damage: the acute organic brain syndrome as treatment. Behavioral and Brain Sciences 1984;7:24-5
- 33. Weiner RD, Rogers HJ, Davidson JRT, Squire LR. Effects of stimulus parameters on cognitive side effects. Presented at the International Conference on ECT: Clinical and Basic Research Issues, New York City, January 16-18, 1985
- 34. Hoch P. Discussion and concluding remarks. J Pers 1948;17:48-51
- 35. Freeman W. Brain damaging therapeutics (editorial). Dis Nerv Syst 1941;2:83
- 36. Wilcox PH. Brain facilitation not destruction the aim of electroshock therapy. Dis Nerv Syst 1946;7:201-7
- 37. Kaplan HI, Sadock BJ. Modern synopsis of comprehensive textbook of psychiatry, III. 3rd ed. Baltimore: Williams & Wilkins, 1981