

21. JANKIS, I. L., 1950. Psychotropic effects of electric convulsive treatments. *J. Nerv. Ment. Dis.* 3: 359-387.

22. JANKIS, I. L., & M. ASTRACHIAN, 1951. The effects of electroconvulsive treatments on memory efficiency. *J. Abnorm. Soc. Psychol.* 46: 501-511.

23. MCGAUGH, J. L., & M. M. HERZ, 1972. *Memory Consolidation*. Albin Publishing Co. San Francisco, Calif.

24. SQUIRE, L. R., & C. W. SPANIS, 1984. Long gradient of retrograde amnesia in mice: continuity with the findings in humans. *Behav. Neurosci.* 98: 345-348.

25. SQUIRE, L. R., C. D. WETZEL, & P. C. SLATER, 1979. Memory complaint after electroconvulsive therapy: assessment with a new self-rating instrument. *Biol. Psychiatry* 14: 791-801.

26. SQUIRE, L. R., & P. C. SLATER, 1983. Electroconvulsive therapy and complaints of memory dysfunction: a prospective three-year follow-up study. *Br. J. Psychiatry* 142: 1-8.

27. FREEMAN, C. P. L., & R. E. KENDELL, 1980. ECT. I. Patients' experiences and attitudes. *Br. J. Psychiatry* 137: 8-16.

28. ZORNETZER, S., & J. L. MCGAUGH, 1971. Retrograde amnesia and brain seizures in mice. *Physiol. Behav.* 7: 401-408.

29. SPANIS, C., & L. R. SQUIRE, 1981. Memory and convulsive stimulation: effects of stimulus waveform. *Am. J. Psychiatry* 138: 1177-1181.

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Effects of Stimulus Parameters on Cognitive Side Effects

(17)

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INTRODUCTION

This symposium has already focused on recent experimental data directed toward an understanding of the differential effects of electrode placement upon both therapeutic response and adverse cognitive effects with electroconvulsive therapy (ECT). In addition, we have yet to hear a number of further expositions on this subject. The available data, presented both here and elsewhere, suggest that unilateral nondominant (UL) ECT is roughly as effective as bilateral (BL) ECT in producing a remission in severely depressed patients.¹⁻³ At the same time, it must be pointed out that technical factors such as sufficient interelectrode distance and the assurance of suprathreshold stimuli also appear to play a role in the efficacy of UL treatments. In addition, there is also a possibility that some patients might respond better to the combination of more intense seizures and denser organic interictal changes produced by bilateral stimulation.

The situation with regard to adverse effects, however, is considerably clearer: unilateral nondominant ECT offers a distinct advantage to bilateral treatments with regard to the presence and extent of cognitive disruption, at least with respect to those functions that depend on the dominant hemisphere.⁴ Still, the extent of data indicating that such amnesic differences exist longer than a few weeks has been largely limited to reports of self-ratings.^{5,6}

Another form of ECT modification, discussed both within this volume as well as elsewhere in the literature, though to a lesser degree than electrode placement, is the

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stimulus waveform. Over the years, a number of attempts have been made to alter the electrical characteristics of the signal used to generate seizure activity with ECT.⁷ Such attempts, while suffering from a variety of methodological inadequacies, have suggested that as long as the duration of the basic stimulus waveform unit does not become too abbreviated, a reasonable degree of therapeutic equivalence appears to exist among the various waveform morphologies.

The role of stimulus waveform in adverse central nervous system (CNS) effects has been a particularly problematic area of investigation. Early studies, which favored low-energy stimuli,⁸ were confounded by concomitant differences in electrode placement. Later investigators reported mixed findings, with some claiming less impairment over a course of ECT given by lower energy stimuli,⁸⁻¹² and others finding no difference.^{13,14} In no case, however, has evidence been presented for persistent deficits on the basis of waveform type.

While a larger number of studies have considered the possible beneficial and/or adverse effects of either electrode placement or stimulus waveform, few have evaluated these effects simultaneously. Valentine *et al.* observed an apparent additive effect of these two modifications on cognitive function during the postictal period.¹² To some degree, Daniel *et al.* found similar additive effects after an individual electrically induced seizure.¹⁵⁻¹⁸ This latter group has also contributed relevant data concerning postictal orientation effects to the present volume.¹⁹

Still, there has been a notable absence of studies focusing upon differences in effects lasting beyond the postictal period. This is of particular interest, given recent claims that the theoretically most benign ECT combination, consisting of unilateral nondominant electrode placement and brief-pulse stimuli, may be at least relatively ineffective from a therapeutic standpoint.^{20,21} In order to investigate more fully the acute and long-term effects of both electrode placement and stimulus waveform on cognitive function, we undertook a prospective study, some of whose results will be presented here. Electrophysiologic findings, suggesting an additive effect for bilateral electrode placement and high-energy sine-wave stimuli in the development of acute adverse cerebral changes with ECT, are presented elsewhere in this volume.²²

METHODS

Subjects were severely ill psychiatric inpatients referred for ECT independently of the research protocol. All met Research Diagnostic Criteria for major depressive disorder,²³ had no ECT within the past year, and had no present or prior evidence of significant CNS disease. A reference group, consisting of similarly diagnosed inpatients not referred for ECT, was also included in the study design. Experimental subjects were randomly assigned to either bilateral or unilateral nondominant electrode placement and to either sine-wave (S) or brief-pulse (P) stimuli. A widely separated centroparietal to frontotemporal configuration,²⁴ applied using careful attention to electrode/scalp coupling, was chosen for the unilateral placement, in order to maximize efficiency of seizure induction.² MECTA (Mectra Corp.) and Medcraft B-24 Mark III (Medcraft Corp.) ECT devices, representing the most widespread pulse and sine-wave equipment available in the United States during the study period, were used to deliver the electrical stimulus.

Specific initial stimulus parameters for each device were chosen to be relatively equivalent with respect to seizure threshold. Single-channel EEG monitoring allowed iterative adjustment of intensity settings to produce seizures lasting longer than 25 seconds. Digital monitoring of stimulus energy was carried out to facilitate calculation

of a variety of electrical parameters. The number of ECT treatments was determined on clinical grounds by the subject's attending psychiatrist. Experimental subjects were tested before ECT, two to three days after the final ECT treatment, and six months following completion of the ECT course. Control subjects were tested at analogous time intervals. A variety of test measures, including those directed toward the assessment of therapeutic outcome, memory function, and EEG, were utilized. Analysis of variance and covariance, along with Pearson-product-moment correlations, formed the basis of statistical determinations.

RESULTS

A total of 53 experimental and 21 control subjects received both baseline and acute post-ECT course testing of clinical and memory parameters (TABLE 1). Thirty-nine of the experimental and 13 of the control subjects also completed the six-month follow-up testing. No differences on the basis of electrode placement or stimulus waveform were found for age ($m = 52.5$), years of education ($m = 11.0$), or socioeconomic status²⁵ ($m = 4.9$). UL subjects had somewhat lower IQs²⁶ than did BL subjects (86 vs. 96, $p < 0.01$). No BL vs. UL or S vs. P differences were found on the basis of history of previous ECT (30%), history of drug nonresponse during the present episode (50%), or evidence of psychosis during the present episode (50%).

TABLE 1. Number of Subjects Receiving Clinical and Memory Testing

	C	PUL	SUL	PBL	SBL
Acute effects	21	10	14	14	15
Long-term follow-up	13	8	10	9	12

As noted in a companion paper,²² no intergroup differences were found on the basis of number of ECT treatments ($m = 9.5$), fraction of treatment sessions resulting in inadequate seizures (less than 25 seconds by single-channel EEG) ($m = 0.08$), or either mean or cumulative adequate seizure duration (57.2 seconds, 509 seconds). Seizure morphology, rated blind to type of ECT, revealed intergroup differences only with regard to postictal suppression.²² This was particularly prominent for SBL ECT, supporting the hypothesis that this particular ECT combination is characterized by intense seizures.

Measures of stimulus intensity showed highly significant intergroup differences with respect to stimulus waveform ($p < 0.0001$), with sine-wave stimuli associated with 2.6 times the stimulus energy (Joules), 3.1 times the applied charge (coulombs), and 6.9 times the mean current (coulombs per second) as that associated with pulse stimuli. This difference in stimulus energy is similar to that reported elsewhere.² Subjects receiving UL ECT tended to receive lower intensity stimuli than those receiving BL treatments (e.g., for energy 36 vs. 44 Joules), though differences just missed statistical significance. In any regard, this case in producing seizures with the UL technique helps to validate the relatively optimal mode of delivery of UL ECT used in the present investigation.

Therapeutic outcome measures included the Hamilton Depression Rating Scale (HDRS),²⁷ the Brief Psychiatric Rating Scale (BPRS),²⁸ the Zung Self-Rating Depression Scale (SDS),²⁹ and a retrospective four-point global rating based on the

discharge summary for the index hospitalization. All ratings were done blind to experimental subgroup. All groups, including controls, were equivalent with regard to baseline HDRS ($m = 23.1$, based on 17-item scale) and degree of acute improvement in HDRS, BPRS, and Zung SDS over the course of treatment ($m = 11.7, 22.5, 15.2$, respectively). A variety of responder criteria, based on combination of HDRS change and cutoff limits, were investigated, without the appearance of significant intergroup differences. Similarly, the retrospective four-point global rating also revealed no variation among subgroups. These findings together suggest an apparent acute therapeutic equivalence on the basis of both electrode placement and stimulus waveform. Finally, no intergroup differences were found in terms of HDRS, BPRS, and Zung SDS change scores between baseline and six-month post-ECT testing, all of which continued to show evidence of improvement ($m = 13.9, 23.9, 11.4$). As described elsewhere,²⁷ therapeutic response was not related to mean or cumulative seizure duration.

Measures of memory function were subdivided into those assessing newly learned information (anterograde memory performance), information learned prior to the study (retrograde memory performance), and self-perceived, or subjective, memory function. Tasks were specifically chosen to be sensitive to ECT-induced effects, based upon previously reported findings. Specific anterograde memory measures included verbal paired associates,³⁰ paragraph retention,³¹ and complex figure reproduction tasks, along with a newly designed instrument involving the learning and recognition of unfamiliar faces. All of these included 20-minute delayed recall testing. Retrograde memory function was evaluated using newly designed and periodically updated famous events and famous faces recall tasks, in addition to an autobiographical, or personal, memory questionnaire. Subjective memory function was tested using a modified version of Squire's Subjective Memory Questionnaire.³²

The personal memory questionnaire was developed to cover a number of items relevant to the subject's life experiences, especially the last several years prior to baseline testing. A careful focus upon this difficult area of memory function was chosen because of earlier findings by others,³³ and is consistent with the nature of memory complaints by ECT patients themselves. This questionnaire included material on the following topics: place of residence, neighbors, family members, close friends, last birthday, last New Year's Eve, last overnight trip out of town, favorite television show, last movie seen at a theater, current hospitalization, and recent outstanding experiences. Only questions responded to at baseline were used at post-ECT test sessions.

Acute effects of ECT upon memory function were evaluated by determining the difference between baseline and two to three day post-ECT course scores, except for the personal memory task, where the percentage of items not recalled after ECT was calculated. TABLE 2 presents a listing of significant acute differences in memory function on the basis of electrode placement and stimulus waveform. In terms of acute effects of ECT upon anterograde measures, the verbal paired associate task proved quite sensitive in separating UL from BL subjects and P from S subjects. As in all cases to be discussed here, both BL and S treatments were associated with greater deficits. In addition, S and BL subjects also tended to perform much worse than control subjects. In what proved an extremely common phenomenon throughout these data, the SBL group was clearly the most impaired. The PUL group, on the other hand, in a manner consistent throughout virtually the entire data set, did not differ from the control group. The complex figure reproduction task was quite sensitive in separating S from P subjects ($p < 0.0008$), but was relatively insensitive in separating UL from BL subjects. This is not surprising, given the fact that nonverbally encodable figural information relies heavily upon the nondominant cerebral hemisphere. Control

subjects, in this regard, performed much better than both S1UL and SBL subjects. The use of baseline HDRS scores as a covariate with regard to acute memory change scores produced no alterations in the above findings. Because of intergroup baseline differences, the IQ score was also used as a covariate, resulting in additional findings favoring UL ECT over BL ECT for both paragraph recall and complex figure reproduction ($p(\text{BL} > \text{UL}) < 0.03, 0.009$).

In terms of acute effects on retrograde memory measures, the famous events recall task was sensitive in differentiating the acute effects of both UL from BL, and P from S ECT. The SBL group was again more impaired than all others ($p < 0.0001$). The famous faces recall task was somewhat less sensitive in discriminating acute effects of ECT on the basis of electrode placement and waveform. The personal memory recall questionnaire, however, proved to be a very sensitive memory measure (FIGURE 1). All groups except control and PUL showed significant levels of relative impairment in the percent of initial items not recalled at the two to three day post-ECT test session. Main

TABLE 2. Acute Memory Impairment (Two to Three Days Post-ECT vs. Baseline Scores)

	p-Values ($2 \times 2 + 1$ ANOVAS)					
	BL > UL	BL > C	UL > C	S > P	S > C	P > C
Anterograde Deficits (Based on Delayed Recall)						
Verbal paired associates	0.002	0.0001	NS	0.002	0.0001	NS
Paragraph recall	NS	0.01	NS	0.002	0.0008	NS
Unfamiliar faces recognition	NS	NS	NS	NS	NS	NS
Complex figure reproduction	NS	0.0008	0.002	0.0008	0.0001	NS
Retrograde Deficits						
Famous events recall	0.0001	0.0001	NS	0.0001	0.0001	NS
Famous faces recall	0.006	0.0001	NS	0.02	0.0001	NS
Personal memory recall	0.0001	0.0001	NS	0.0001	0.0001	NS
Global self-rating of memory function	NS	NS	NS	NS	NS	NS

effects for UL vs BL and P vs. S differences were present and, in addition, SUL, PBL, and SBL were more impaired than controls ($p < 0.03, p < 0.004, p < 0.0001$, respectively). In order to evaluate the role of guessing at the time of pre-ECT testing, the percent of baseline items not recalled at the second test session but later recalled at the third test session was determined. This measure showed similar relationships to that described above, suggesting that the results regarding acute effects are in fact valid. Use of baseline HDRS and IQ scores as covariates affected only the famous faces recall findings, for which the differences between UL and BL ECT groups disappeared. This may indicate that our famous faces recall test was not as sensitive as its famous events counterpart.

Overall, the objective data with respect to acute memory changes strongly implicate both bilateral electrode placement and sine-wave stimuli as potent risk factors, as do the EEG results reported elsewhere in this volume.²² In terms of subjective memory function, however, a rather uniform tendency for self-perceived memory function to improve acutely following ECT was noted. No differences in terms

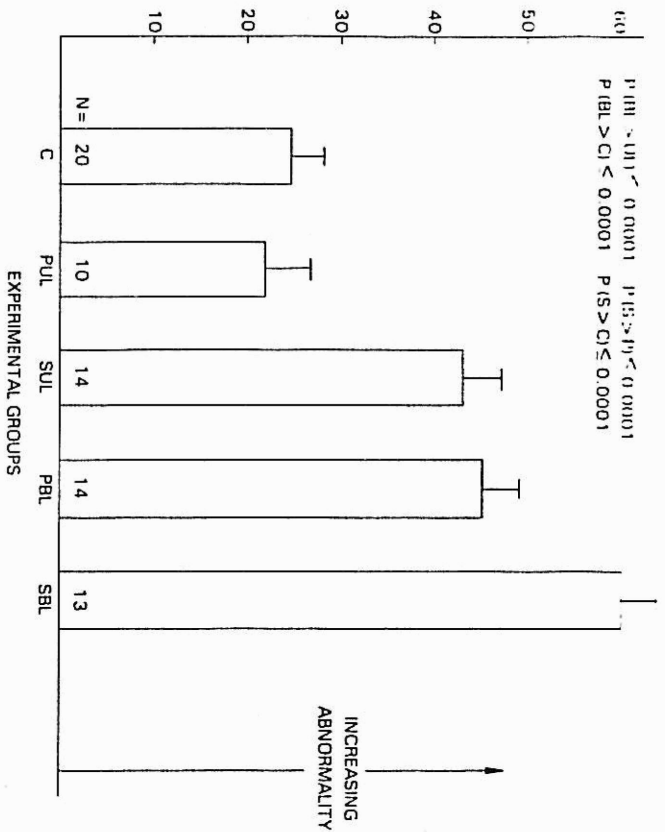


FIGURE 1. Acute personal memory impairment. Ordinate represents percent of baseline items not recalled two to three days post-ECT (+ standard error).

of post-ECT minus pre-ECT change scores, on either a total or an item-by-item basis, were observed as a function of ECT type or between controls and ECT subjects. Also interestingly, no appreciable relationship between subjective and objective memory measures was found, but, instead, acute measures of subjective memory function were significantly correlated with the respective differences in interviewer-rated and self-rated depression scales. While it is possible that the two to three day post-ECT rating time may not have allowed subjects sufficient opportunity to be aware of the extent of their deficits, this latter relationship suggests that self-rated memory changes with regard to a course of ECT may be more a function of the clinical response than of objectively demonstrable changes in memory function. Such a finding is compatible with available data reported by others,^{34,35} but represents the first time this has been demonstrated in a systematic fashion.

Persistent ECT-associated effects upon memory function were investigated in a fashion identical to acute effects, except that baseline scores were compared to those obtained at six-month follow-up testing. In general, a return to at least pre-ECT level of function was found, with no evidence of intergroup differences for any of the measures of anterograde memory function. The famous events and famous faces recall tasks were likewise not productive of any long-term intergroup differences, although there was the suggestive finding that the three subjects with the largest long-term losses on the famous events task were all in the SBL group. The personal memory recall task, however, revealed a highly significant persistent intergroup difference favoring UL over BL ECT (Figure 2). The percentage of initial items not recalled at both the

two to three day post-ECT and six-month post-ECT test sessions revealed a greater impairment for BL subjects than either UL subjects ($p < 0.005$) or control subjects ($p < 0.002$). These differences were significant even when partitioning on the basis of stimulus waveform, and were not affected by the use of baseline HDRS or IQ as covariates. There was also suggestive evidence for a long-term toxicity of S with respect to control subjects ($p < 0.01$) on autobiographic memory, though no P vs. S differences were observed in this regard, and the latter difference disappeared with use of baseline IQ as a covariate. Again, no intergroup differences in long-term subjective memory function, as determined by either total or item-by-item change scores, were found.

The above results represent provocative evidence for what amounts to objective personal memory losses lasting at least six months with BL but *not* with UL ECT, and represents the first time such a differential effect has been reported. While analysis of personal memory data with respect to recency effects has not been completed, a preliminary assessment indicates that items dealing with the year immediately preceding the ECT may have been most affected.³⁶ At the same time, however, it does appear that the described period of retrograde amnesia is greater than, say, a few weeks.

Unfortunately, the study of autobiographic memory function, as carried out in the present protocol, is confounded by the possibility that some personal memory information given at the time of baseline testing may have been incorrect. In an attempt to partially compensate for such a potential bias all subjects were asked, immediately

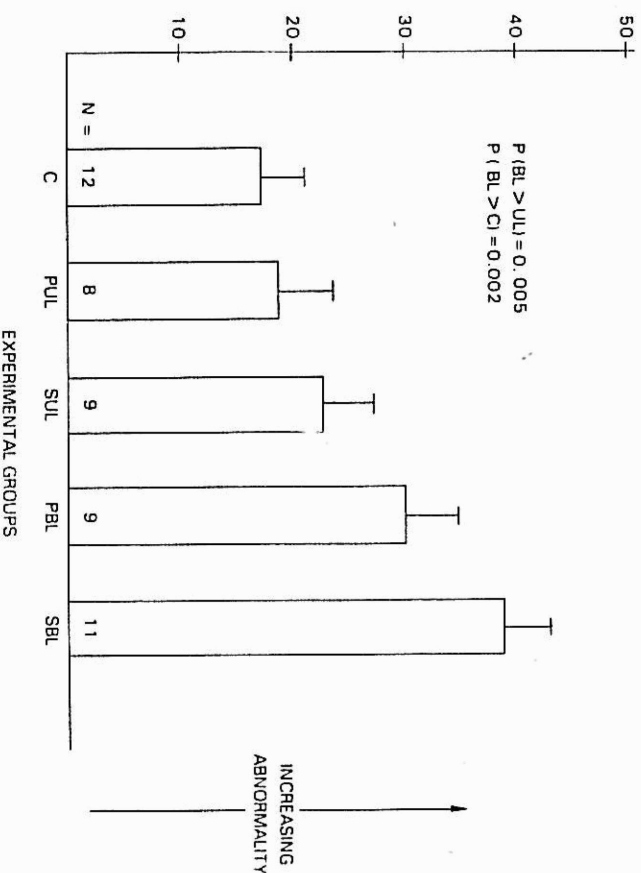


FIGURE 2. Long-term personal memory impairment. Ordinate represents percent of baseline items not recalled at both two to three day and six-month post-ECT test sessions (+ standard error).

following completion of the six-month follow-up personal memory questionnaire, to respond to a series of recognition trials. These consisted of questions based upon all items where both acute and follow-up responses differed from those given at baseline testing. In every case, subjects were given three choices: baseline response correct, six-month follow-up response correct, or unclear which of the two responses was correct. This procedure, in effect, approximated an attempt at "self-corroboration" of baseline items. Choices of the response that was given at baseline would suggest that responses given at the time of six-month follow-up testing may have been based upon incorrect recall, but that recognition of the correct response was still intact. Similarly, choices of the "unclear" alternative would suggest that both recall and recognition might be deficient.

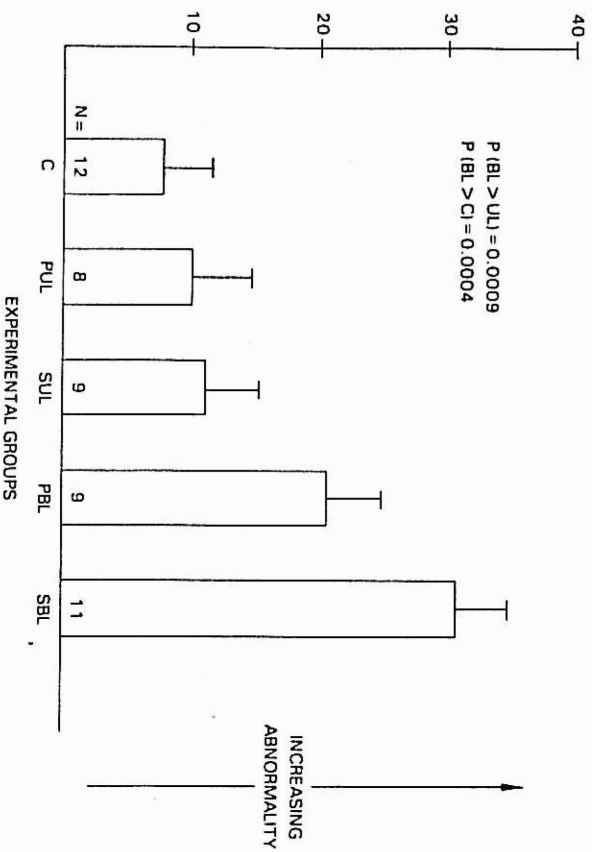


FIGURE 3. Long-term personal memory impairment adjusted using self-corroborative technique (see text for details). Ordinate represents percent of baseline items not recalled at both two to three day and six-month post-ECT test sessions (+ standard error.) Corrected to include only items with "session 1" and "uncertain" self-corroborative responses.

Upon applying this self-corroborative technique and rejecting all equivocal items (i.e., where subjects felt that their follow-up response, rather than their initial response, was correct), the level of difference between BL and both UL and C groups was indeed found to increase (FIGURE 3). This strengthens the likelihood that the observed findings represent a true persistent deficit with BL ECT. Still, it must be pointed out that any such self-corroboration would be even more useful when supplemented by external corroboration using significant others or other relevant sources. For this reason, attempts to provide this modification were begun in the latter portion of the study. This allowed a comparison of long-term effects of ECT between C (four subjects) and SBL (three subjects) groups, using externally corroborated items.

It was, in fact, found that corroborated items, which constituted around three-fourths of the entire data set for those subjects, showed at least as much persistent forgetting (35% of baseline items for SBL vs. 13% for C subjects) as was observed based upon analysis without the use of external corroboration.

Given that both acute and persistent memory deficits were present, a further series of points can be made with respect to their possible correlates. First, even though HDRS scores were highly correlated with subjective memory ratings, no such relationship was established between HDRS scores and results on objective memory testing for either acute or long-term effects. This suggests that the findings are in fact organic rather than functional, and is supported by a number of highly significant correlations between acute objective memory test changes and acute EEG abnormalities. Second, the presence and amount of ongoing psychotropic medications presumably could affect memory performance. Analyses of medication effects are pending, though preliminary consideration of these factors indicates that BL subjects, for example, were no more likely to be medicated at the final test session than UL subjects. Next, the possibility that memory changes might be related to number of ECT treatments or EEG seizure parameters was considered. No significant correlations were observed, though the range of available values could have precluded relationships from appearing.

Finally, the effects of stimulus intensity per se (energy, current, and charge) upon objectively assessed memory function were evaluated. Here it was determined that stimulus intensity, especially energy, was correlated significantly with a variety of measures of memory function, particularly both acute and long-term personal memory performance ($p < 0.0002$, $p < 0.0009$, respectively). While at first this was felt to represent perhaps a reflection of S vs. P differences, these relationships were found to be present only with S ECT and *not* with P ECT, despite the existence of wide parameter ranges in each case. This suggests that, as long as stimuli are only slightly suprathreshold, the relatively low-energy stimuli present with the pulse waveform may lie below a cutoff for intensity-related effects upon memory performance with ECT and, furthermore, that only the higher energy sine-wave stimulus is able to exceed this cutoff in an appreciable number of cases.

DISCUSSION AND CONCLUSIONS

This study has provided a number of findings that are both new and potentially pertinent to clinical practice. Evidence presented suggests that unilateral nondominant electrode placement and brief-pulse stimuli may each provide significantly fewer acute CNS adverse effects while remaining equally effective. Given the present widespread clinical reluctance to use such ECT modifications, the finding of long-term personal memory impairment with bilateral electrode placement is particularly important.

The bases for why electrode placement and stimulus waveform should each exert independent and additive differential effects upon memory systems are poorly understood.⁶ UL nondominant ECT appears to be associated with less intracerebral current flow, less generalization of the seizure discharge, and less postictal suppression in the dominant, contralateral hemisphere. It has even been proposed that seizures produced by UL and BL ECT differ in their onset, i.e., focal cortical initiation with the former and generalized diencephalic onset with the latter.³⁷ Certainly, such electrophysiologic differences could well account for the relative sparing of at least verbal memory function with UL nondominant ECT.

The basis of stimulus waveform effects on cognitive performance could be

secondary to the direct electrical effects of a higher mean current or charge distribution within the cerebral structures subserving memory function with S ECT. Alternatively, the apparently more intensely generalized seizures produced by the higher energy stimuli, as discussed in a companion paper in this volume,²² may be involved. The former possibility, when combined with the reported significant relationship between sine wave stimulus intensity and extent of both acute and long-term memory deficits, raises a concern about the use of grossly suprathreshold stimuli,²³ something that was not dealt with in the present study.

At present, we are involved in an attempt to replicate and extend the findings described above. It is felt that such work is crucial, not only to the understanding of how ECT produces both its beneficial and adverse effects, but also in the optimization of a beleaguered and maligned treatment modality which has time and time again proven too clinically valuable to consign to the halls of oblivion.

REFERENCES

1. D'ELIA, G. & H. RAOTMA. 1975. Is unilateral ECT less effective than bilateral? *Br. J. Psychiatry* **126**: 83-89.
2. WEINER, R. D. 1980. ECT and seizure threshold. *Biol. Psychiatry* **15**: 225-241.
3. WELCH, C. A. 1982. The relative efficacy of unilateral nondominant and bilateral stimulation. *Psychopharmacol. Bull.* **18**: 68-70.
4. SQUIRE, L. R. 1982. Neuropsychological effects of ECT. *In Electroconvulsive Therapy: Biological Foundations and Clinical Applications*. R. Abrams & W. B. Essman, Eds.: 169-186. SP Medical and Scientific Books, New York, N.Y.
5. SQUIRE, L. R. & P. CHACE. 1975. Memory functions six to nine months after ECT. *Arch. Gen. Psychiatry* **32**: 1557-1564.
6. WEINER, R. D. 1984. Does ECT cause brain damage? *Behav. Brain Sci.* **7**: 1-53.
7. WEAVER, L. A., JR. & R. W. WILLIAMS. 1982. The electroconvulsive therapy stimulus. *In Electroconvulsive Therapy: Biological Foundations and Clinical Applications*. R. Abrams & W. B. Essman, Eds.: 129-156. SP Medical and Scientific Books, New York, N.Y.
8. LIEBERSON, W. T. & P. H. WILCOX. 1945. Electric convulsive therapy: comparison of "brief stimuli technique" with Friedman-Wilcox-Reiter technique. *Digest Neurol. Psychiatry* **13**: 292-302.
9. KENDALL, B. S., W. B. MILLS & T. THALE. 1956. Comparison of two methods of EST and their effects on cognitive functions. *J. Consult. Psychol.* **20**: 423-429.
10. CRONHOLM, B. & J. O. OTTOSON. 1963. Ultrabrief stimulus techniques in ECT. I. Influence on retrograde amnesia. *J. Nerv. Ment. Dis.* **137**: 117-123.
11. CRONHOLM, B. & J. O. OTTOSON. 1963. Ultrabrief stimulus techniques in ECT. II. Comparative studies of therapeutic effects and memory disturbances in treatment of endogenous depression with the Elther ES apparatus and Siemens Konvulsator 111. *J. Nerv. Ment. Dis.* **137**: 268-276.
12. VALENTINE, M., K. M. G. KEDDIE & D. DUNNE. 1968. A comparison of techniques in electroconvulsive therapy. *Br. J. Psychiatry* **114**: 989-996.
13. GOLDSTEIN, S. G., S. B. FILSKOV, L. A. WEAVER & J. O. IVES. 1977. Neuropsychological effects of electroconvulsive therapy. *J. Clin. Psychol.* **33**: 798-806.
14. WARREN, E. W. & D. H. GROOMER. 1984. Memory test performance under three different waveforms of ECT for depression. *Br. J. Psychiatry* **144**: 370-375.
15. DANIEL, W. F., H. F. CROVITZ, R. D. WEINER & H. J. ROGERS. 1982. The effects of ECT modifications on autobiographical and verbal memory. *Biol. Psychiatry* **17**: 919-924.
16. DANIEL, W. F., R. D. WEINER & H. F. CROVITZ. 1983. Autobiographical amnesia with ECT: an analysis of the roles of stimulus waveform, electrode placement, stimulus energy, and seizure length. *Biol. Psychiatry* **18**: 121-126.
17. DANIEL, W. F., H. F. CROVITZ & R. D. WEINER. 1984. Perceptual learning with right unilateral versus bilateral electroconvulsive therapy. *Br. J. Psychiatry* **145**: 394-400.
18. DANIEL, W. F., H. F. CROVITZ, R. D. WEINER, H. S. SWARTZWELDER & E. M. KAHN. 1985. ECT-induced amnesia and postictal EEG suppression. *Biol. Psychiatry* **20**: 379-382.
19. DANIEL, W. F. & H. F. CROVITZ. 1986. Disorientation during Electroconvulsive Therapy. *Ann. N.Y. Acad. Sci.* (This volume.)
20. LAMINGTON, J. & D. GILL. 1978. A controlled comparison of simulated and real ECT. *Br. J. Psychiatry* **133**: 514-519.
21. PRICE, T. R. P. 1981. Unilateral electroconvulsive therapy for depression. *N. Engl. J. Med.* **304**: 53.
22. WEINER, R. D., II, J. ROGERS, J. R. T. DAVIDSON & E. M. KAHN. 1986. Effects of electroconvulsive therapy upon brain electrical activity. *Ann. N.Y. Acad. Sci.* (This volume.)
23. FEIGHNER, J. P., E. ROVINS, S. B. GUZE, R. A. WOODRUFF, A. WINOKUR & R. MUNOZ. 1972. Diagnostic criteria for use in psychiatric research. *Arch. Gen. Psychiatry* **26**: 57-63.
24. D'ELIA, G. 1970. Unilateral ECT. *Acta Psychiatr. Scand. Suppl.* **215**: 5-98.
25. HOLLINGSHEAD, A. B. & F. C. REDLICH. 1958. *Social Class and Mental Illness: A Community Study*. John Wiley & Sons, New York, N.Y.
26. SHIPLEY, W. C. 1940. A self-administering scale for measuring intellectual impairment and deterioration. *J. Psychol.* **9**: 371-377.
27. HAMILTON, M. 1960. A rating scale for depression. *J. Neurol. Neurosurg. Psychiatry* **23**: 56-62.
28. OVERALL, J. E. & D. R. GORHAM. 1962. The Brief Psychiatric Rating Scale. *Psychol. Rep.* **10**: 799-812.
29. ZUNK, W. W. K. 1970. A self-rating depression scale. *Arch. Gen. Psychiatry* **12**: 63-70.
30. SQUIRE, L. R. & P. C. SLATER. 1978. Bilateral and unilateral ECT: effects on verbal and nonverbal memory. *Am. J. Psychiatry* **135**: 1316-1320.
31. WECHSLER, D. 1945. A standardized memory scale for clinical use. *J. Psychol.* **19**: 87-95.
32. SQUIRE, L. R., C. D. WETZEL & P. C. SLATER. 1979. Memory complaint after electroconvulsive therapy: assessment with a new self-rating instrument. *Biol. Psychiatry* **14**: 791-801.
33. JANIS, I. 1950. Psychological effects of electroconvulsive treatments. I. Post-treatment amnesias. *J. Nerv. Ment. Dis.* **111**: 359-382.
34. CRONHOLM, B. & J. O. OTTOSON. 1963. The experience of memory function after electroconvulsive therapy. *Br. J. Psychiatry* **109**: 251-258.
35. STERNBERG, D. E. & M. E. JARVIK. 1976. Memory functions in depression: improvement with antidepressant medication. *Arch. Gen. Psychiatry* **33**: 219-224.
36. SQUIRE, L. R., P. C. SLATER & P. L. MILLER. 1981. Retrograde amnesia following ECT: long-term follow-up. *Arch. Gen. Psychiatry* **38**: 89-95.
37. STRATON, R. D., P. J. HASS & R. A. BRUMBACK. 1981. Electroencephalographic recording during bilateral and unilateral nondominant hemisphere (Lancaster position) electroconvulsive therapy. *J. Clin. Psychiatry* **42**: 264-269.
38. OTTOSON, J. O. 1960. Experimental studies on the mode of action of electroconvulsive therapy. *Acta Psychiatr. Neurol. Scand.* **36**(Suppl. 145): 1-141.