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Brief Report

The Effects of ECT Modifications on Autobiographical and Verbal Memory

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INTRODUCTION

Electroconvulsive therapy (ECT) produces memory impairment which may be modified (Valentine *et al.*, 1968; Squire, 1977; Weiner, 1979) by a choice of stimulus electrode placement (bilateral *vs.* unilateral nondominant) or electrical stimulus wave form (sinusoidal *vs.* brief-pulse). Regarding electrical stimulus wave form, it has been suggested that more amnesia may follow sinusoidal than brief-pulse ECT because more total electrical energy is delivered by the former than the latter treatment modality (Medlicott, 1948; Kendall *et al.*, 1956; Cronholm and Ottosson, 1963; d'Elia, 1974).

Several investigations have revealed that personal information inventories are sensitive means of assessing ECT-induced amnesia (Janis, 1950; Janis and Astrachan, 1951; Stieper *et al.*, 1951; Squire *et al.*, 1981; Weiner *et al.*, 1982). To date, however, no investigation has examined the effects of the aforementioned ECT modifications on memory for a specific autobiographical *episode* (e.g., "How did you celebrate your last birthday?"). These effects are examined in the present investigation.

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MATERIAL AND METHODS

Subjects

A group of 16 male inpatients, all meeting Research Diagnostic Criteria (Feighner *et al.*, 1972) for major depressive disorder, was studied. The Hamilton Interviewer-Rated Depression Scale (Hamilton, 1960) was administered before each patient's first ECT to measure severity of depression. Patients with any evidence or history of neurological dysfunction were excluded. No patient was tested who had received ECT within 12 months prior to his present ECT course. Dominance was determined by a battery modified from d'Elia (1970). All patients were strongly right-body dominant.

ECT Technique

Patients received either standard *bilateral* frontotemporal ECT or *unilateral nondominant* ECT (d'Elia, 1970, placement). Electrical stimulation was either bidirectional *brief pulse* (800-mA peak amplitude, 60 pulse-pairs/sec, 0.75- to 1.5-msec pulse duration, 1.25- to 2.00-sec pulse train duration; MECTA Corp. device) or bidirectional *sinusoidal* (140-170 V rms, 60 Hz, 0.5- to 1.0-sec train duration; Medcraft B-24 Mark III device). Thus four treatment groups were formed (unilateral nondominant pulse, unilateral nondominant sine, bilateral pulse, bilateral sine). Patients were randomly assigned to one of these four groups.

ECT was administered three times a week (M,W,F). Patients were premedicated with atropine (mean of 0.6 mg im) 30 min before ECT. Anesthesia was produced by intravenous methohexital, and subtotal muscle relaxation was achieved by intravenous succinylcholine. Ventilation with 100% O_2 was begun shortly after methohexital injection and was continued (except for several seconds during electrical stimulation) until satisfactory spontaneous respiration was achieved.

Seizures were monitored electroencephalographically. Seizure length was taken as time until cessation of epileptiform activity. The number of joules of electrical energy was measured with a custom-made integrating watt-second meter (Indiana University). Table I illustrates patient and ECT variables. The four groups were balanced with respect to all of these variables except electrical energy. Sinusoidal stimulation delivered more joules of electrical energy than did pulse stimulation (means: sine = 68.6 joules, pulse = 30.6 joules; $F = 13.6$, df = 1, 12, $p < 0.01$), a difference which is consistent with that reported elsewhere (e.g., Weiner, 1980).

Table I. Patient and ECT Variables

Variable	Range	Mean	Standard deviation
Age (years)	28-73	58.2	13.2
Hamilton score	30-62	47.7	11.8
Education (years)	4-16	10.2	3.1
Methohexital (mg)	60-80	65.6	8.9
Succinylcholine (mg)	60-120	73.8	18.2
Seizure length (sec)	25-195	57.2	41.7
Joules of energy	13-129	49.6	31.3

Memory Testing

Base-line memory testing was attempted 45 min (mean: 50 min) before each patient's sixth ECT. At this time, patients were read the "Airplane List" (Crovitz, 1979) three times. This story contains ten target words structured in a bizarre-imagery chain-mnemonic format to encourage deep and elaborate encoding (Crovitz, 1979). After each reading, free-recall memory was tested. Following the third free-recall testing, multiple-choice recognition memory was tested. The correct word was randomly interspersed with four distractor words. The last testing mode (story-cued recognition) involved reading each sentence of the story one at a time, with a missing blank(s) where the target word belonged. The same choices used in multiple-choice testing were printed below each sentence. Patients were instructed to guess on both recognition tests if they did not know the correct word.

Twenty-four hours after ECT, each patient was first asked "Do you remember being told a story containing ten words yesterday morning before your treatment?" The patient's "yes" or "no" response was accepted on face value as indicating the presence or absence of autobiographical memory for having heard the Airplane List. Each patient was informed that he *was* told a story before his treatment, and was asked to free-recall words from the story. Multiple-choice and story-cued recognition testing were then performed exactly as was done before ECT.

RESULTS

Table II displays autobiographical memory as a function of electrode placement and stimulus wave form. An exact Mantel-Haenszel Test (Thomas, 1975) revealed less autobiographical memory following bilateral than unilateral

Table II. Autobiographical Memory as a Function of Electrode Placement and Stimulus Wave Form

Autobiographical memory present?	Treatment modality: Bilateral sine ($n = 3$)	Bilateral pulse ($n = 4$)	Unilateral sine ($n = 5$)	Unilateral pulse ($n = 4$)
Yes	0	0	4	3
No	3	4	1	1

nondominant ECT ($p < 0.01$), but no effect due to stimulus wave form ($p > 0.20$). There was no difference in joules of electrical energy ($t = 0.87$, $p > 0.20$) or seconds of seizure length ($t = 0.49$, $p > 0.20$) between patients with and without autobiographical memory.

Figure 1 displays the amount of pre–post ECT forgetting of Airplane List words as a function of treatment group. Analysis of variance revealed a significant main effect for electrode placement ($F = 9.2$, df = 1, 12, $p < 0.05$), with greater forgetting following bilateral than unilateral ECT. There was no main effect for stimulus wave form ($F = 1.9$, df = 1, 12, $p > 0.10$), and there was no

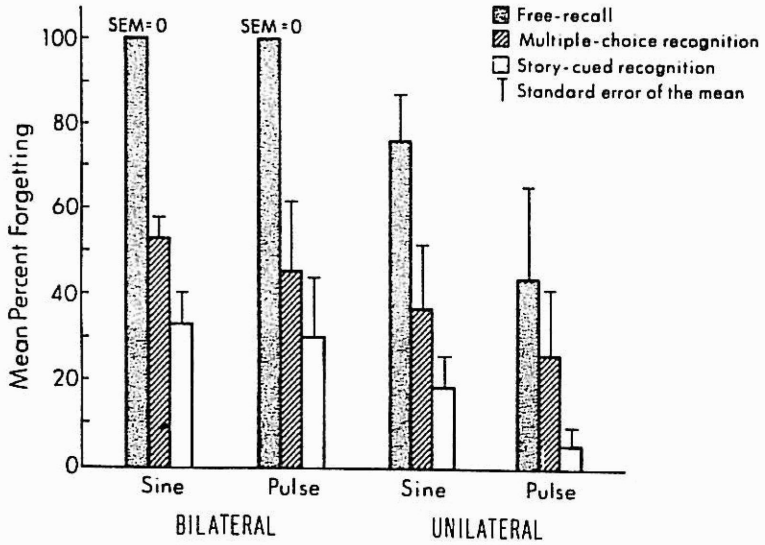

Fig. 1. Mean percentage of words forgotten before and after ECT in relation to treatment group.

interaction of electrode placement with stimulus wave form ($F = 0.9$, df = 1, 12, $p > 0.20$). Pairwise Tukey tests revealed that bilateral ECT produced more forgetting than unilateral ECT on free-recall testing ($p < 0.05$), but not on multiple-choice or story-cued recognition testing ($p > 0.05$).

DISCUSSION

Sinusoidal stimulation did not produce significantly greater autobiographical or verbal amnesia than did brief-pulse stimulation. Other studies have reported more amnesia following sinusoidal than pulse stimulation, but these studies contain the following serious methodological inadequacies: failure to establish statistical significance for alleged intertreatment amnestic differences (Medlicott, 1948; Epstein and Wender, 1956; Valentine *et al.*, 1968); confounding of results by postictal confusion (Medlicott, 1948; Valentine *et al.*, 1968); failure to specify whether patients were oxygenated (Medlicott, 1948; Kendall *et al.*, 1956; Valentine *et al.*, 1968); intertreatment difference in hypoxia (Epstein and Wender, 1956); and intertreatment differences in treatment number and spacing (Kendall *et al.*, 1956). Our study contains none of these methodological inadequacies, and no statistically significant effect of stimulus wave form on memory functions was observed.

Regarding electrode placement, our results are consistent with other reports of greater retrograde amnesia following bilateral than unilateral nondominant ECT (e.g., Lancaster *et al.*, 1958; Cannicott and Waggoner, 1967; Costello *et al.*, 1970; d'Elia, 1970; Weiner *et al.*, 1982). However, this is the first investigation to demonstrate a statistically significant greater impairment in memory for an autobiographical *episode* following bilateral than unilateral nondominant ECT.

The forgetting of an autobiographical episode as simple as having heard the Airplane List before ECT is not a trivial phenomenon. Similar ECT-induced autobiographical memory failures, if added across a course of ECT, may produce *gross* autobiographical memory gaps that may be disconcerting to a patient and a patient's family, because the patient's sense of continuity with his or her own past may be disrupted. It is not yet known how far back in time autobiographical deficits extend. Nor is it known whether low-energy brief-pulse ECT will reduce these deficits if autobiographical memory is evaluated more thoroughly than in the present investigation.

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Glucose-6-Phosphate Dehydrogenase Deficiency in a Psychiatric Population: A Preliminary Study

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Glucose-6-phosphate dehydrogenase (G6PD) is the rate-limiting enzyme of the hexose monophosphate shunt. Deficiency of G6PD is a recessive X-linked metabolic disorder (Beutler, 1974). Erythrocytes are particularly affected but other tissues have also been found to be deficient in this enzyme. G6PD deficiency may result in hemolytic anemia, particularly after the ingestion of certain drugs, fave beans, or after conditions of stress like bacterial infections. Hemolysis has also been known to occur following exposure to pollen. It is estimated that this enzyme deficiency affects around 100 million people around the world, but mainly blacks, Mediterraneans, and Sephardic Jews.

Following the report of two black men with G6PD deficiency who developed transient psychosis following the administration of primaquine sulfate (Dern *et al.*, 1963), G6PD deficiency was surveyed in hospitalized chronic schizophrenic patients (Dern *et al.*, 1963, Bowman *et al.*, 1965; and Fieve *et al.*, 1965). These studies showed no association between G6PD deficiency and psychosis, but there are questions about the diagnostic and assay reliability used in these studies. Heller *et al.* (1979) studied sickle cell disease and G6PD deficiency in over 65,000 admissions to Veterans Administration hospitals. They also found no correlation between G6PD deficiency and any psychiatric diagnosis.

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