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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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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

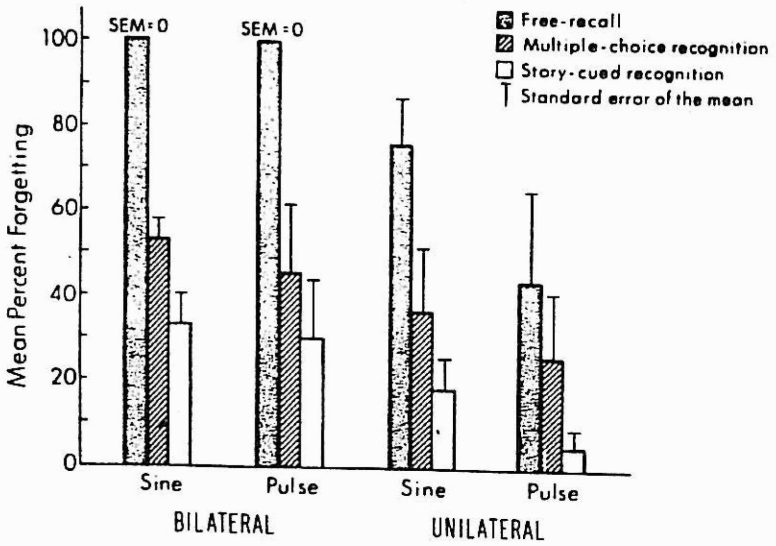

Fig. 1. Mean percentage of words forgotten before and after ECT in relation to treatment group.

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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Brief Report

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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