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ECT: Organic Brain Syndrome

The Natural History of Acute Organic Mental Syndrome After Bilateral Electroconvulsive Therapy

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Thirty-one patients undergoing bilateral electroconvulsive therapy (ECT) were followed prospectively for the development of acute organic mental syndrome (AOMS); 15 patients (48.4%) developed AOMS during ECT. For these 15 patients, the average number of ECTs before development of AOMS was 5.5 with average duration of AOMS being 20.1 days. Comparison of these 15 patients to the 16 patients who did not develop AOMS for diagnoses, demographic data, pre-ECT laboratory data, and medications, differed only in exposure to psychoactive medications and prior presence of major medical illness.

INTRODUCTION

Memory deficits and confusional states are said to be characteristic side effects of electroconvulsive therapy (Harper and Wiens, 1975; Harwitz, 1974). Nevertheless, there are few published data on the frequency and duration of mild organic mental syndrome after electroconvulsive therapy (ECT). It is assumed that such confusional states are caused by the electroconvulsive therapy. Other possible causes of postelectroconvulsive therapy confusion, such as metabolic or drug-induced confusional states, have not been excluded.

This study reports on the incidence, duration, and associated risk of acute organic mental syndrome in patients receiving ECT.

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METHOD

Thirty-one randomly selected patients undergoing bitemporal electroconvulsive shock treatments at Renard Hospital (St. Louis, Missouri) agreed to participate in this study. All subjects were older than 16 years and before treatment were medically screened with an electrocardiogram, spine radiographs, complete blood count, serum electrolyte, blood urea nitrogen, and serum glucose, calcium, phosphorus, glutamic-oxaloacetic transaminase, lactic dehydrogenase, bilirubin, alkaline phosphatase, and creatine phosphokinase. All 31 subjects were interviewed before initiation of ECT and a minimum of four times after initiation of therapy. The initial interview consisted of a routine medical history and a standardized research questionnaire based on psychiatric diagnostic criteria of Feighner *et al.* (1972). By the diagnostic method of Feighner *et al.*, it is possible for the same individual to have more than one psychiatric diagnosis (Robins *et al.*, 1977). Standardized evaluation of orientation, recent memory, and remote memory was done initially and on each follow-up visit. The method used was that of Irving *et al.* (1970). Memory testing of all subjects was done 30–36 hr after every second ECT (second, fourth, sixth, etc.) and the last ECT. Subjects who developed confusion were tested daily until the acute organic mental syndrome (AOMS) cleared or the patient was discharged from the hospital. Charts were reviewed for observations of confusion, disorientation, or other symptoms of organic mental syndrome. Exposure to psychoactive drugs was estimated by means of drug risk number (DRN) calculation. The DRN method is described in detail elsewhere (Summers, 1978). Essentially, the higher the DRN number, the greater the exposure to drugs known to induce or enhance anticholinergic organic mental syndromes.

The terms post-ECT confusion and AOMS are used interchangeably in this paper. Acute organic mental syndrome was defined in this study by the presence of either of the following: (i) Acute deterioration of intellectual functioning as measured by "names learning" and remote memory test (Irving *et al.*, 1970); or (ii) Acute loss of orientation to time (greater than 3 days error), place, or person. The term "delirium" was reserved for patients who met criteria for AOMS and had two of the following: delusions, hallucinations, depersonalization, rapid fluctuation of affect, or bizarre psychomotor activity.

ECT procedure was bifrontal with seizure precipitated by a Reiter MOLAC II electroshock device. A 60-cycle a-c flow set at "low" milliamperage at 105–125 V for 1.0–2.0 sec between bifrontal electrodes was usually adequate to induce a grand mal seizure. Pre-ECT medications were: atropine, 1.0 mg subcutaneously (30-60 min before treatment); methohexital, 0.3 mg/lb iv; and succinylcholine, 0.2 mg/lb iv (Pitts, 1972). Only three subjects received pretreatment oxygen. Duration of seizures were 15–30 sec.

Table I. Characteristics of Postelectroconvulsive Therapy Confusion[a]

	Mean (days)	Range (days)
ECT of onset	5.5 ± 1.3	1-19
Day of onset	10.5 ± 2.2	1-33
Day of termination	30.5 ± 2.4	19-44
Duration	20.1 ± 3.2	5-43

[a] Data from 13 subjects with no evidence of organic mental syndrome prior to ECT.

RESULTS

Of 31 subjects completing the study, 15 (48.4%) developed AOMS. No subject met criteria for delirium. Two subjects had mild chronic organic mental syndrome and affective disorder before ECT. Both developed acute organic mental syndrome which had a duration of 45+ and 65+ days after the first ECT. Characteristics of AOMS in 13 subjects with no previous history of memory impairment are given in Table I.

There were no significant differences between the 16 comparison subjects and the 15 AOMS subjects for age, sex, or race. Table II gives data on factors previously associated with organic mental syndromes. Comparison between AOMS patients and comparison patients for preelectroconvulsive therapy laboratory values showed no significant differences. The psychiatric diagnoses prior to treatment are given in Table III.

Exposure to psychoactive drugs is given in Fig. 1. AOMS subjects received more exposure to psychoactive agents than comparison subjects during the course

Table II. Risk Factors Associated with Acute Organic Mental Syndrome

	N	Age (years)	PMD[a]	PSD[b]	FmH[c]	ECT[d]
AOMS	15	47.0 ± 12.4	9[e]	9	8	9.7 ± 0.9
Comparison	16	36.5 ± 7.1	3[e]	9	9	8.8 ± 0.3

[a] PMD = Presence of significant medical illness in past.
[b] PSD = Previous major surgery.
[c] FmH = Family history of psychiatric disease.
[d] ECT = Mean number of electroconvulsive treatments given. For age and ECT, 1.96 SE is given.
[e] Refers to $p < 0.05$.

Table III. Psychiatric Diagnosis of Comparison and Postelectroconvulsive Therapy Confusion Subjects (AOMS)[a]

Diagnosis	AOMS[b]	Comparison
Unipolar affective disorder (primary)	6	4
Unipolar affective disorder (secondary)	5	4
Bipolar affective disorder (primary)	3	2
Bipolar affective disorder (secondary)	0	2
Antisocial personality	0	2
Briquet's syndrome	0	1
Anxiety neurosis	0	3
Obsessive-compulsive neurosis	0	1
Alcohol abuse	1	1
Drug abuse	1	0
Schizophrenia	1	0
Schizophrenia affective	1	1
Chronic organic mental syndrome	2[c]	0
Undiagnosed	1	2

[a] $p > 0.05$ (not significant) for all diagnoses.
[b] 6 AOMS subjects had 2 diagnoses, 5 comparison subjects had 2 diagnoses, 1 comparison subject had 3 diagnoses.
[c] Post-ECT confusion in these two subjects was markedly prolonged (see text).

of ECT. This difference reached significance of $p < 0.05$ on days 5, 7, 14, 16, 18, and 19 after the initiation of ECT.

DISCUSSION

Shortly after Cerletti and Bini (1938) described electroconvulsive therapy, the problem of memory deficit was recognized (Sherman *et al.*, 1941; Stainbrook, 1946; Huston and Strother, 1948; Stone, 1947; Wilcox, 1949). Initially, memory deficit was felt to be a necessary side effect of electroconvulsive therapy (Brengelmann, 1959). Korin *et al.* (1956), Cronholm and Ottosson (1961), and Ottosson (1969; 1962; 1967) demonstrated that memory deficit after ECT was not necessary for clinical improvement. Since the recognition of electroconvulsive therapy induced memory deficit as a side effect, and since the advent of unilateral ECT, there have been many studies and reviews of post-ECT memory deficits (Cronholm and Molander, 1957; Cronholm and Blomquist, 1959; Fink, 1977; Halliday *et al.*, 1970; Dornbush, 1972; Squire, 1974, 1975; Dornbush and Williams, 1974; Squire and Chace, 1975; Harper and Weins, 1975; Reichert *et al.*, 1976; Squire *et al.*, 1975, 1976; Small *et al.*, 1977; D'Elia and Raotma, 1977; Squire and Stater, 1978; Frankel *et al.*, 1978). Few of these studies are pertinent to these data, as they concern only the single symptom of memory deficit, and many of these studies concern only effects immediately after ECT. The symptom of memory disturbance is an integral part of AOMS, but it is possible to have mild memory disturbance without the presence of clinical AOMS. This paper concerns the clinical syndrome of acute organic mental syndrome.

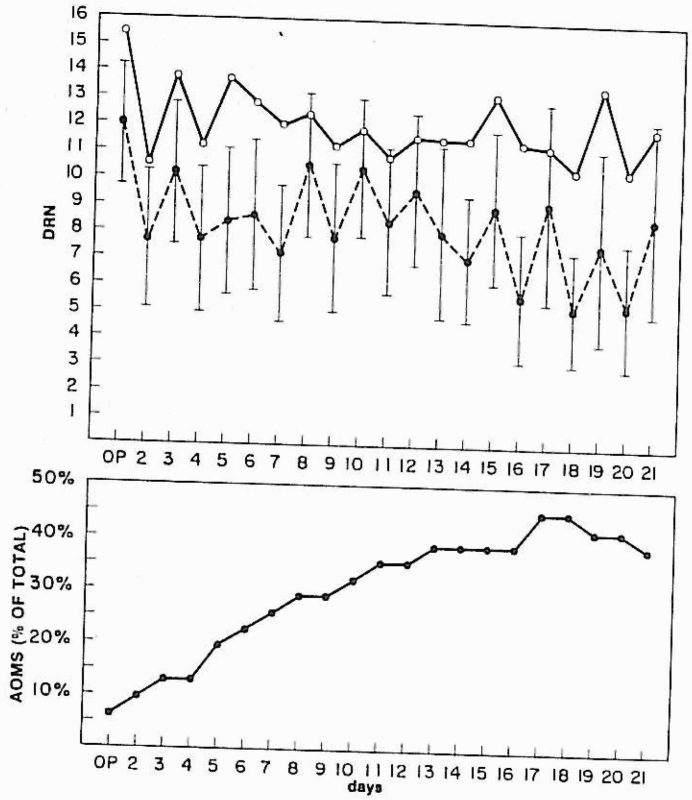

Fig. 1. Exposure to psychoactive drugs estimated by drug risk number (DRN) calculation.

There are only two studies addressing AOMS after ECT (Kalinowsky and Hoch, 1946; Stainbrook, 1946). Both using less precise methods note that the AOMS clears within "2 weeks." If one assumes that post-ECT confusion represents an extension of the process causing minimal memory deficits, crude comparison with the literature is possible. The frequency of no discernible memory deficit after ECT has been reported to be 12.6–27.0% (Bidder *et al.*, 1970; Brunschwig *et al.*, 1971). In this study, 51.6% of subjects did not meet criteria for AOMS. The average onset of ECT-related AOMS reported here was 5.5 ± 1.3 with a mean duration of 20.1 ± 3.1 days. Brunschwig *et al.* (1971) tested memory function 36 hr after ECT and found an average onset of memory deficit after the fourth ECT. Follow-up studies of memory deficits are consistent with the duration of AOMS observed here (Strain *et al.*, 1968; Bidder *et al.*, 1970; Squire,

and Chace, 1975; Squire *et al.*, 1976). Squire and associates demonstrated memory deficits 12 days after completion of ECT, but not at 6–9 month follow-up (Squire, 1975; Squire *et al.*, 1976). Strain *et al.* (1968) and Brunschwig *et al.* (1971) noted a trend of improvement in recent memory between the last ECT and 10 days of follow-up. Follow-up of these patients at 30 days and 1 year showed memory to be better than pre-ECT testing (Bidder *et al.*, 1970).

The etiology of post-ECT confusion is elusive. Holmberg (1953) postulated anoxia as a cause in unmodified ECT. Ottosson (1960) proposed that excessive electrical current was causal. Others have proposed psychologic phobic reactions as causal (J. Wortis, personal communication). It is known that other forms of AOMS are associated with drug toxicity, increasing age, preexisting chronic organic mental syndromes, and prior major medical problems (Summers and Reich, 1979).

In this study, there were no apparent significant differences in environmental factors, demographic data, number of treatments, and pre-ECT laboratory studies. There was definite association between prior medical illness and probable association with prior chronic organic mental syndromes.

The finding of higher drug exposure in AOMS subjects is of theoretical and possibly practical interest. Drachman (1977) has postulated that memory is a function of central nervous system cholinergic neurons and that acute memory loss represents dysfunction of these neurons or an anticholinergic state. The DRN data would support this theory, because high DRN values reflect increased exposure to anticholinergic drugs (Summers, 1978). It seems unlikely that anticholinergic drugs alone are responsible for the AOMS seen in this study. The DRN values noted here are considerably less than that noted in other types of AOMS (Summers, 1978; Summers and Reich, 1979). Further, convulsive therapy alone is known to cause AOMS. Anticholinergic drugs, then, may only be contributory to AOMS after ECT.

If Drachman's hypothesis is correct, ECT itself should exert an anticholinergic effect. The effect of ECT on neurotransmitters has been reviewed by Essman (1973). In this review, there is evidence that ECT induces rupture or increased permeability of presynaptic vesicles containing bound acetylcholine. The liberated acetylcholine is reflected by elevation of extracellular acetylcholine after ECT. Because of reduced availability of "bound" intracellular acetylcholine, ECT may cause a synaptic acetylcholine deficit. If this hypothesis is correct, anticholinesterase drugs may improve or reverse AOMS after ECT. Further, the incidence of AOMS after ECT could be reduced by minimizing psychoactive drug exposure during ECT.

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Effects of Rate of Repetitive Stimulus Presentation on the Visual Evoked Brain Potentials of Young Adults with Down's Syndrome

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Visual evoked brain potentials (VEP) to repeated stimuli of several interstimulus intervals (ISI) were recorded from young adults with Down's syndrome (DS). The following results were obtained: (i) An ISI effect previously observed in normals: VEP amplitudes increased with increase in ISI; some ISI effect on latency was also observed; (ii) VEP amplitudes of DS subjects were larger than VEP amplitudes of normals; (iii) VEP peak latencies of DS subjects were longer than VEP peak latencies obtained from normals; (iv) ISI had a more pronounced effect on VEP amplitudes of DS than normal subjects. These results are discussed with respect to CNS differences and issues of attention and information processing.

INTRODUCTION

A number of studies have demonstrated a relationship between evoked potentials (EP) and stimulus presentation rate or its complimentary interstimulus interval (ISI). This effect (recovery function, ISI effect) has been noted in humans for the early components (e.g., Shagass, 1977) and long-latency components of the EP, and for ISIs of less than 1 sec (e.g., Surwillo, 1977) and greater

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