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# P 300 Event Related Potential in Patients Receiving ECT

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## *Abstract*

Modified bilateral sinewave ECTs were administered to 14 patients (7 males) with manic depressive psychosis (10 patients with depressive episode and 4 patients with manic episode). P 300 event related potential (ERP) was recorded using auditory oddball paradigm. No changes in the latency or amplitude of the P 300 ERP were detected form pre-ECT occasion to later occasions viz
(a) post-sham ECT,
(b) post-true ECT (first) or
(c) post4ast ECT (5-14 ECTs).

All patients had recovered by the time last ECT was given. P 300 ERP recorded using auditory oddball paradigm did not detect cerebral dysfunction 12-24 hours after ECT.

Key words -
**Bilateral ECT,**
**Affective disorder,**
**P 300 event related potential,**
**Cerebral dysfunction**

ECT is known to result in transient cerebral dysfunction. Electrophysiological measures of such cerebral dysfunction have employed EEG and averaged evoked potentials (AEP). Early components of evoked potentials are uninfluenced by ECT or are affected for a very brief postictal period [1]. Early components of AEP reflect integrity of brainstem and thalamocortical projections and it is likely that longer latency potentials putatively of cortical origin [2] are more vulnerable to ECT related dysfunction. Shaw [3] while demonstrating normal brainstem auditory evoked potential even during seizure, observed that a longer latency component (P8) was affected with electroconvulsive shock [4]. Among the longer latency potentials is the P 300 event related potential (ERP). Recognition of a novel/rare stimulus amidst the more commonly occurring non-novel stimuli, is associated with a positive wave at about 300 msec latency. This study examined the influence of ECT on this net of AEP the P 300 ERP.

## Method

## Patients

Patients diagnosed as manic depressive psychosis (both mania and depression) according to the 9th edition of International Classification of Diseases (ICD9, 296), were prescribed ECT by the treating consultant after obtaining informed consent. Consent was again sought for additional procedures in the study which included one sham-ECT, recording of event related potential and clinical assessments. Consenting patients who were right handed were included. Clinical evidence of hearing impairment, history of middle ear infection, use of ototoxic drugs, past history of head trauma with post traumatic amnesia of more than 48 hours or CSF leak/bleeding through ear, history of alcohol or other drug abuse, history of epilepsy, were the exclusion criteria. Patients in whom concurrent psychotropic medication tion were prescribed, were expected to remain on the same dosage and changes if any should not exceed 50% of the initial dose during the course of ECT. Fourteen patients were included in this study.

## ECT

ECTs were administered on alternate days. The first session included only sham ECT which was anaesthesia (vide infra). Thiopentone (5 mg/kg), succinylcholine (0.75 mg/kg) and atropine (1 mg) were used as intravenous anasethesia. Sinewave stimulus of 110-150 Volts RMS, passed for 0.5 to 0.8 seconds was applied bifrontotemporaly. Seizure duration was recorded by BP cuff method [5]. Twenty seconds of motor seizure was considered adequate. Patients were supported with 100% positive pressure artificial ventilation, until resumption tion of regular spontaneous respiration. The judgment to stop ECTs was made by the treating consultant.

## Assessments

The severity of mania or depression was rated on the appropriate sections of Minicompendium of Rating Scales (MCRS) [6] before the starting of ECTs, and at weekly intervals 48 hours after the ECT. P 300 was recorded before ECTs, 8-24 hours after sham ECT, 8-24 hours after first ECT and 24-48 hours after the last ECT.

## P 300 stimulus

P 300 stimulus was generated using a commercial stimulator. The stimulus was applied binaurally using ear phones. The repetition rate was 0.6 Hz at 35 db above hearing threshold. The stimulus had 7 msec rise and fall time, with 100 msec plateau time. The frequent stimulus was 1 kHz pure tone. The novel/infrequent stimulus was 1.5 .5 kHz pure tone. The infrequent tone occurred randomly at 20% chance.

Patients were tested in a sound attenuated chamber in a sitting position. During the testing they were asked to keep the vision fixed on a point two meters in front of them. They were familiarised with the tones and the response key 10-15 minutes before the testing. The recording electrodes were applied during this time. Testing began only when patients were recognizing 80% or more of the odd (novel) tones.

EEG was recorded using (MEE 4108 - Neuropack 8 from Nihon Kohden) a commercially available averager. Recordings were made from Pz site of 10-20 system referenced to right mastoid. The electrode impedence was less than 5 kiloohms. The EEG was filtered between a band width of 0.5 and 30 Hz with filter rolloff at 3 db per octave. EEG epochs of 700 msec duration beginning with the onset of the infrequent / novel stimulus were averaged. Epochs containing artefacts were automatically rejected by the averager. 30 epochs were averaged. Patients were expected to detect the odd tone (1.5

kHz) by pressing a key button. All patients detected 80% or more of the novel stimuli. Latency of the maximal positive peak between 250 and 600 msec was measured. Its absolute amplitude in relation to an isoelectric DC line was also estimated.

## Results

Sample consisted of 14 patients (7 males). Present episode was depressive in ten patients and mania in four patients. Mean (SD) age of the sample was 28.7 (6.6) years (range 20-40 years). Patients received 5-14 ECTs (mean and SD=8 and 2.7). All patients were rated as recovered by end of five weeks after starting the treatment according to the MCRS. Mean (SD) seizure duration of the first ECT was 32.5 (9.0) seconds and that of the last ECT 32.1 (8.8) seconds.

Mean latency (milliseconds) and amplitude (microvolts) of the P 300 ERP recorded at pre, post-sham ECT, post-1st ECT and post-last ECT are presented in Table I. Paired 't' test failed to identify changes in the ERP measures from pre ECT to post-sham ECT suggesting that anaesthetic agents had no influence on the ERP. Nor were theERP measures different at either of the post-ECT occasions from pre ECT values. The results do not indicate any influence of ECT on the P 300 ERP (Table I).

***Table I - Mean (SD) latency and amplitude of P 300 event related potential***

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Paired 't' test was applied with Bon Ferroni correction criteria. Comparing Pre ECT scores of latency and amplitude with the three consecutive post treatment occasions, viz., post-sham, post 1st ECT and Postlast ECT none of the post treatment values either of latency of amplitude differed significantly ($P > 0.05$) from the pre treatment values.

## Discussion

P 300 ERP is a sensitive index of cerebral dysfunction. Delay in latency and reduction amplitude are detected in disorders such as dementia [7], Parkinsons's disease [8] and post traumatic amnesia [9]. It is claimed that P 300 is a useful tool in differentiating true dementia from depressive pseudodementia [7]. P 300 is mooted to represent short term memory [10] with its origins implicated in hippocampus [2]. Memory dysfunction is a well demonstrated side effect of ECT [11]. Hippocampal damage is associated with prolonged seizures or status epilepticus [12].Therefore, ECT was expected to influence this component of ERP the P 300.

In this study however, P 300 ERP measures were unaffected by ECT. No changes were detectable 8-24 hrs after the first ECT. Cumulative effects of consecutive ECTs (5-14 ECTs) on this measure too, were not detectable 24-48 hours after the last ECT, when the patients were declared clinically improved.

Failure to demonstrate effect of ECT on this supposedly sensitive index of cerebral dysfunction, while reassuring, needs to be viewed with caution. It is possible, that a testing during earlier postical period - perhaps first 4-6 hours - was more likely to identify changes in P 300 ERP. Testing was done 24-48 hours after the last ECT. Clinicians could not decide stopping of treatment before this period.

Depressives can have P 300 ERP abnormalities [13] and its amplitude improves after successful antidepressant therapy [14]. In this study (depressives 70%) successful ECT treatment did not change P 300 ERP in either direction despite clinical recovery. It is likely that P 300 ERP improvements with clinical recovery compensated the changes produced by ECT. Comparison of P 300 ERP changes after a fixed number of ECTs between patients who recovered and those who did not recover might detect changes attributable to ECT. Patients may differ from healthy controls when they are ill as well as when they have recovered after ECT. Alternatively if antidepressants are used in place of ECT, improvements in P 300 may be identified after recovery as opposed to no change after ECT induced recovery.

Topographical variations in P 300 ERP before and after ECT merit attention. For example, P 300 ERP is known to vary across and within hemispheres in patients with schizophrenia [15] and dementia [16]. Detecting P 300 from one scalp site perhaps failed to detect P 300 ERP changes following ECT.

In summary, this preliminary study demonstrated no change in auditory P 300 ERP in patients with affective disorders 8-48 hours after bilateral modified ECT.


1.Kris A, Halliday A M & Pratt R T C, Evoked potentials following unilateral ECT II. The flash evoked potentials
*Electroencephalography & Clinical Neurophysiology* Page: 65: 141-149, 1980

2.Halgren E, Squires N K, Wilson C L, Rohrabaugh J W, Babb T L & Crandall P H, Endogenous potentials generated in the human hippocampal formation and amygdala by infrequent events
*Science* Page: 210: 803-805, 1980

3.Shaw N A, The effect of electroconvulsive shock on the brain stem auditory evoked potential in the rat
*Biological Psychiatry* Page: 21:1327-1331, 1986

4.Shaw N A, Effects of electroconvulsive shock on the slow component of the brain stem auditory evoked potential
*Experimental Neurology* Page: 100: 242-247, 1988

5.Jain S, Sheshadri S & Gangadhar B N, [Monitoring the seizure during electroconvulsive therapy]
*NIMHANS Journal* Page: 7: 115-117, 1989

6.Beach P, Kastrup M & Rafaelsen O J, Mini-compendium of rating scales for states of anxiety, depression, mania and schizophrenia with corresponding DSM III syndromes
*Acta Psychiatrica Scandinavica* Page: Suppl. 326, 73:1-37, 1986

7.Goodin S D & Aminoff M, Electrophysiological differences between demented and nondemented patients with Parkinson's disease
*Annals of Neurology* Page: 21: 90-94, 1987

8.Gordon E, Krainhin C, Harris A, Meares R & Howson A, The differential diagnosis of dementia using P 300 latency
*Biological Psychiatry* Page: 21:1123-1132, 1986

9.Papincolau A C, Levin H S, Eisenberg H M, Moore B D, Goethe K E & High W M, Evoked potential correlation of post traumatic amnesia after closed head injury
*Neurosurgery* Page: 14: 676-678, 1984

10.Hammon E J, Meador K J, Augustine R & Wilder B J, Cholinergic modulation of human P300 event related potential
*Neurology* Page: 346-349, 1987

11.Weiner R D, Does electroconvulsive therapy cause brain damage?
*The Behavioural & Brain Sciences* Page: 7: 1-53, 1984

12.Meldrum B S, Neuropathological consequences of chemically and electrically induced seizures
*In: Electroconvulsive Therapy: Clinical and Basic Research Issues. (Eds.) Malitz S and Sackeim H A.* Page: 462, 186-193, 1986
13.Diner B C, Holcomb P J & Dykman R A, P 300 in major depressive disorder
*Psychiatry Research* Page: 15: 175-184, 1985
14.Blackwood D H R, Whalley L J, Christie I E, Blackburn I M, St. Clair D M & McInnes A, Changes in auditory P 3 event related potential in schizophrenia and depression
*British Journal of Psychiatry* Page: 150: 154-160, 1987
15.Morstyn R, Duffy H & McCurley R W, Altered P 300 topography in schizophrenia
*Archives of General Psychiatry* Page: 40: 729-734, 1983
16.Maurer K, Dierks T & Ihl R, Quantitative P 300 data and their topography in dementia
*Statistics and Topography in Quantitative EEG* Page: 6: 243-250, 1988