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To assess whether Resting Anterior EEG Alpha Asymmetry can be considered as a Trait Marker for Depression

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Original Research Article

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Abstract: Our finding of anterior EEG alpha power in patients may be unstable. Several reasons may contribute to this instability. First, there is preliminary evidence on an association of state affect and EEG alpha power asymmetry in depression. For this reason, we have not explored the relation of the subjects' self-reported mood and anterior EEG asymmetry, but neither in healthy controls nor in depressed patients did we find a significant relation between positive affect, negative affect or a combined measure of mood and anterior EEG asymmetry for methodology. Thus, we did not find evidence that state mood is linked to variations in frontal EEG asymmetry, but, of course, we cannot exclude this possibility. To sum up, the findings reported in this study add to the growing body of evidence to show alterations of anterior brain activation in depression. Since anterior EEG asymmetry lacked temporal stability in depressed patients in this study, it appears too premature to consider anterior EEG alpha power asymmetry a trait marker for depression. Further research is needed to uncover the temporal characteristics of anterior EEG asymmetry to clinical features of depression. It is in this spirit that the present remarks are offered.
Keywords: Anterior EEG, Depression & Alpha Asymmetry.

INTRODUCTION

The human brain is the control centre of the body. It is responsible for perception, cognition, attention, emotion, memory and action .When a person is thinking, reading or watching television different parts of the brain are stimulated. This creates electrical signals, which, together with chemical reactions, let the parts of the body communicate.

These electrical signals can be monitored through scientific techniques such as electroencephalography (EEG) etc. Electroencephalography is a medical imaging technique that reads scalp electrical activity generated by brain structures. The electroencephalogram (EEG) is defined as electrical activity of an alternating type recorded from the scalp surface after being picked up by metal electrodes and conductive media [1].

The EEG measured directly from the cortical surface is called electro cortiogram while when using depth probes it is called electrogram. In this thesis, we will refer only to EEG measured from the head surface [2].

In 1929 Hans Berger reported in a publication his observations on what he termed '*das Elektrenkephalogramm*', which became the seminal paper highlighting the beginning of research on the human electroencephalogram also abbreviated as EEG. In his first experiments he recorded the EEG from his son Klaus – among others – and described extensively the methods he used and what he observed. Figure 1 below demonstrates 2 graphs from that first publication recorded from his son. The bottom figure represents the rhythm he would eventually call the 'alpha EEG rhythm' and the top figure represents what he would eventually call the 'beta EEG rhythm'. Since that time much research has been dedicated to measuring EEG under different conditions as well as measuring EEG in a variety of disorders ranging from neurological to psychiatric[3,4].

MATERIALS & METHODS

Ethical clearance from the institutional ethics committee was obtained before starting the study.

CONDUCT OF STUDY

The study was jointly conducted in the Department of Physiology& Psychiatry of N.S.C.B. Medical College, Jabalpur M.P.

DURATION OF STUDY

The study was conducted from March 2016 to August 2017 for data collection, after which data was analysed.

SAMPLE SIZE

In this study the sample size is 100 (N= 60 depressive patients and N= 40 controls).

SELECTION CRITERIA OF SUBJECTS

The participants in this study had been offered to voluntarily participate in the study and they had to give the written informed consent before enrolment in the study. The study will be carried out on a group of 60 patients with major depressive disorder and 40 age-matched control subjects. The diagnosis of Depression will be established by an experienced Psychiatrist and those already diagnosed patients were recruited from the Department of Psychiatry, NSCB Medical College to the Department of Physiology. Subjects with non-psychotic depressive disorder as defined by ICD-10 criteria and determined by 17-item Hamilton Depression Rating Scale (HAM-D) score higher than 14 will be eligible.

Forty adults with no psychiatric, alcohol/drug abuse or dependence history (assessed with non-patient version of the SCID [SCID-IV-I/NP]), and no history of seizures or brain trauma were tested. Controls were included only if they scored ≤13 on the Beck Depression Inventory, had no Psychiatric history and no psychiatric history in first-degree relatives.

INCLUSION CRITERIA

- Patients of average adult age (18-60yrs) of either gender.
- Both new and old diagnosed outpatients of Depression.
- Patients who gave written informed consent.
- Patients with good physical health as determined by physical examination
- Controls who are physically, mentally and socially healthy with no past medical history and scored <13 on the Beck Depression Inventory-II.

EXCLUSION CRITERIA

- Patients who do not give informed consent for participation in the study.
- Patients who received Lithium as medication.
- Patients unable to respond to verbal questions.
- Patients with a history of Bipolar disorder, Schizophrenia, alcohol or drug dependence within last 5 years or significant suicidal ideation.
- BDI point < 10

METHODS AND EQUIPMENT

All study participants underwent conventional EEG registration .All enrolled patients who were diagnosed cases of Depression were registered and provided with a date on which the EEG is supposed to be done. The procedure for a subject included continuous EEG recording for 30 mins duration between time interval 9 a.m. to noon. The subjects lay in relaxed position with closed eyes during the recording. The room was dark but no other special conditions were provided. Electrodes were placed according to the International "10-20" system using linked-ears as a reference. EEG recordings were performed with 16 channel Neurograph machine (Neurocompact). All EEG recordings were performed by the same EEG technician.

The sampling rate was 256 Hz, the amplitude bandwidth between 0.3 and 70 Hz, and impedance levels were ≤5 kΩ. Epochs contaminated by blinks, eye movements, and movement-related artefacts were excluded from analyses by direct visual inspection.

EEG AND DEPRESSION

Several articles have appeared on EEG screening and indications for referral in Psychiatric populations. An annotated bibliography of significant EEG-Psychiatric references was also published Hughes 1995. In addition a new organization, the American Psychiatric Electrophysiological Association (APEA), was formed in 1993 for purposes of reviewing current knowledge of EEG applications in Psychiatry, fostering education and generating guidelines for training requirement for clinicians and technologists working in the field. The primary objective of APEA is to improve the standards of EEG practice by Psychiatrists to enable them to acquire subspeciality certification by the American Board of Electroencephalography and Electrophysiology. EEG provides important information about Depression, more so than in most other psychiatric illnesses [2].

A growing body of evidence strongly suggests that the right and left cerebral hemispheres are differentially involved in the regulation and processing of emotion [5]. During the past two decades, anterior electroencephalographic (EEG) alpha power asymmetry, an inverse measure of relative brain activation, has been related to hemispheric specialization of fundamental dimensions of affect, resulting in a model of anterior asymmetry and emotion [5, 6].

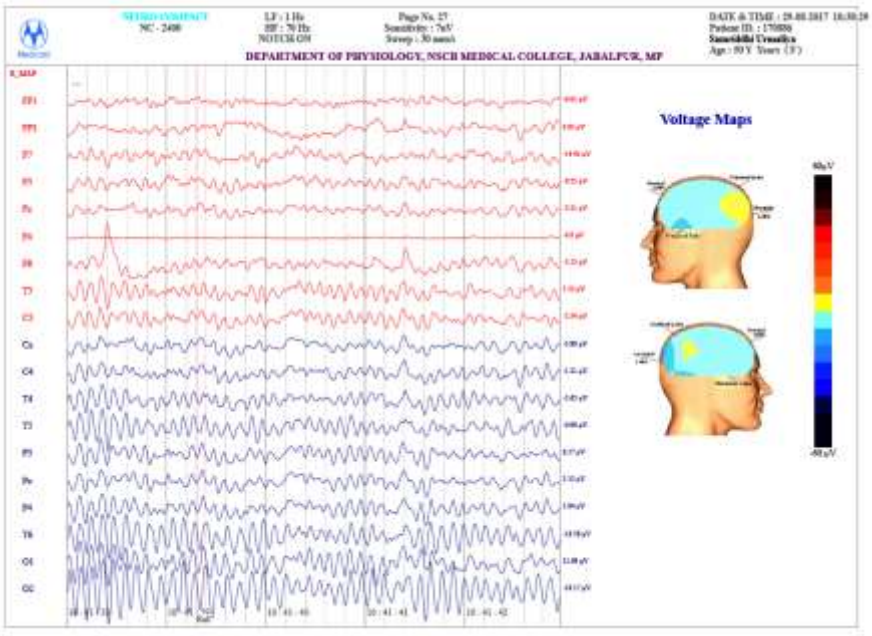
DATA ANALYSIS AND STATISTICS

EEG analysis was performed using Matlab software using EEG Lab toolbox. The statistics were computed using software IBM SPSS Statistics version 23.0.

STATISTICAL DESCRIPTION

An EEG signal can be characterized by the distribution of the amplitude and its moments. For each segment of an EEG signal

OBSERVATION & RESULTS



Fig–1: Eeg recording of a patient

Asymmetry scores for depressive patients (n=60)

Table-2 (A): Alpha power for frontal region (fp_1, f_3, f_7)

Variable	Descriptive statistics (Frontal asymmetry-spectra power) Include cases 1 : 60				
	Valid N	Mean	Minimum	Maximum	Std. Dev.
Fp_1	60	1.891	0.166	12.987	1.879
F_3	60	2.831	1.256	12.354	2.338
F_7	60	3.354	0.276	16.957	2.289

Table– 2 (B): Alpha power for frontal region (fp_2, f_4, f_8)

Variable	Descriptive statistics (Frontal asymmetry-spectra power) Include cases 1 : 60				
	Valid N	Mean	Minimum	Maximum	Std. Dev.
Fp_2	60	2.387	0.287	12.092	2.014
F_4	60	3.198	0.178	18.557	2.468
F_8	60	2.763	0.296	13.537	2.059

DISCUSSION

The present study evaluated regional characteristics and their statistical properties of asymmetric brain activation in depression by assessing EEG alpha power during rest. In agreement with prior findings, depressed patients differed from healthy controls in resting anterior EEG asymmetry when data were collapsed. Depressed patients failed to show more left than right anterior alpha activity, whereas healthy controls had markedly more right than left anterior alpha activity, i.e. a relative greater activation of left anterior regions.

Asymmetry scores conceptually simplify certain analyses, such as those involving correlations between frontal asymmetries (as a difference score) and other individual difference measures (e.g., behavioural activation. Additionally, difference scores based on alpha power asymmetries tend to show high internal consistency and acceptable test-retest reliability, dispelling fears about reduced reliability attributable to difference scores per se.

CONCLUSION

Our finding of anterior EEG alpha power in patients may be unstable. Several reasons may contribute to this instability. First, there is preliminary evidence on an association of state affect and EEG alpha power asymmetry in depression. For this reason, we have not explored the relation of the subjects' self-

reported mood and anterior EEG asymmetry, but neither in healthy controls nor in depressed patients did we find a significant relation between positive affect, negative affect or a combined measure of mood and anterior EEG asymmetry for methodology. Thus, we did not find evidence that state mood is linked to variations in frontal EEG asymmetry, but, of course, we cannot exclude this possibility.

To sum up, the findings reported in this study add to the growing body of evidence to show alterations of anterior brain activation in depression. Since anterior EEG asymmetry lacked temporal stability in depressed patients in this study, it appears too premature to consider anterior EEG alpha power asymmetry a trait marker for depression. Further research is needed to uncover the temporal characteristics of anterior EEG asymmetry to clinical features of depression. It is in this spirit that the present remarks are offered.

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