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# Comparison of Quantitative Electroencephalography of Depression Patients With Healthy Controls

KEYWORDS: Quantitative electroencephalography -Fast Fourier transformation.

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**ABSTRACT** *The neuro-psycho-physiological disease processes of Depression, an Affective Disorder, were selected in the present study to evaluate and explore the axes that the selected psychopathologies modulate and influence clinically. Depression, influence the individual's cognition, emotions and behavior. The aim of our study is to Compare Absolute Power of qEEG between depression patients and healthy subjects to determine a specific pattern of quantitative EEG for depression which can be useful in early diagnosis and for prognosis. Drug naïve newly diagnosed 31 depression and 32 healthy subjects were gone through 300 seconds eye close supine resting EEG recording over 20 electrodes. Recorded data analyzed by fast Fourier transformation, and then calculated absolute power analyzed by applying unpaired t- test. Depression Patients showed increased alpha and beta activity than healthy subjects. This could be potentially used in early diagnosis and for prognosis of depression.*

## INTRODUCTION

Depression is an affective disorder characterized by a state of low mood and aversion to activity that can affect a person's thoughts, behavior, feelings and sense of well – being. In depression, loss of interest in daily activities results in social withdrawal, decreased ability to function in occupational and interpersonal areas and decreased involvement in previously pleasurable activities. In severe depression, there may be complete anhedonia (inability to experience pleasure). [(1)]

In India, the prevalence of depression is 2.6 per 1000 and incidence is 0.44 per 1000 population at risk. An estimated 5.8% of men and 9.5% women will experience a depressive episode in any given year. [(1)]

Recently, the World Mental Health Report 2001 has identified unipolar depression as the fourth cause of Disability Adjusted Life Years (DALYs) in all ages, and the second cause in the age group of 15 – 44 years. Unipolar depression is also the first cause of YLD (Years of Life Lived with Disability) in all ages. The life – time risk of depression in males is 8 – 12% and in females is 20 – 26%. However, the life-time risk of major depression (or depressive episode) is about 8 percent. [(2)]

The incidence of abnormal conventional EEG findings in mood disorders appears to be substantial, ranging from 20% to 40%. qEEG holds a promise in generating information through the EEG signals in depressed patients. It has been observed that depressed patients have an enhanced power spectral density of alpha and distributed beta activity with less distributed delta activity as compared to that observed in non – depressed control. [(3)]

A decrease in the power spectral density of slow waves of the delta-theta bands and an increase in the beta activity in depressed patients have also been shown.[(4)]

Numerous qEEG studies have found increased alpha and/or theta power in a high percentage of depressed patients.

Decreased current density in the delta band has been observed in the right temporal lobe and a similar trend was also observed in theta, alpha and beta bands. The diagnostic value of EEG in depression is still a topic under discussion. [(5)]

More recent studies have confirmed that depression differ in their qEEG indicators, but results are not consistent Thus, the significance of qEEG parameters as possible biological markers of depression would be confirmed.

The present study was undertaken to evaluate the quantitative electroencephalographic (qEEG) pattern in the patients of depression.

## MATERIAL AND METHODS

The study was conducted in the Upgraded department of Physiology in association with the department of Psychiatry, S.M.S. Medical College, Jaipur. The study design was Hospital based comparative type of observational study.

### Participants

Thirty one depression patients and thirty two healthy controls of age and sex matched were recruited in the present study. The study group was recruited from the O.P.D. of Psychiatry department of S.M.S Medical College and Attached Hospitals, Jaipur. The study was carried out from May 2014 to November 2015 after the approval of institutional ethics committee.

**Inclusion Criteria:**

- Patients between 18 – 40 yrs.
- Drug-naïve or newly diagnosed patients of Depression according to the criteria of Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-iv TR 2000).
- Those who has given written informed consent for this study..

**For Healthy Controls**

- Age and sex matched, who have consented for the study.

The **Exclusion Criteria** for the study (when the subjects would not form part of the study):

- Patients with a personal or family history of neurologi-

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cal/psychotic disturbances, other than schizophrenia and depression.
- Patients on neuroactive substances and/or prophylactic drugs.
- Not Cooperative.

**Procedure**
Following instructions had been given before EEG recording:

The subjects should shampoo hair the night before the test and use of hair cream, oils or spray afterwards was restricted. The controls and patient that comprised the study population was asked to avoid all food and drinks containing caffeine for 2 hrs before the test.

**EEG Recording, Analysis, and Quantification**
EEG was recorded using a stretchable cap which was positioned on the subject's head according to known anatomical landmarks.

QEEG was done on all the patients and controls using BESS (brain electro scan software) of the Axxonet System (India).

The 20 electrodes were positioned on the scalp according to the International 10-20 System with biauricular reference. EEG was recorded for 300 seconds in eye closed resting supine position from frontal (Fz/Fp1/Fp2, F3/F4, F7/F8), temporal (T3/T4, T5/T6), central (C3/C4/Cz), parietal (P3/P4/Pz), and occipital (O1/O2/Oz) regions. Impedance was kept below 5 KΩ and electrical activities, amplified with a band – pass filter of 0.5 Hz – 30.0 Hz, digitized at sampling rate 256 Hz. Artifact – free epochs of 2 seconds each was chosen and their spectral content evaluated by means of Fast Fourier Transform analysis.[6] The following parameters were observed and evaluated:

Absolute power values in microvolt for individual segments of EEG spectrum of delta (0.5 – 4.0 Hz), theta (4.0 – 8.0 Hz), alpha (8.0 – 13.0 Hz) and beta (13.0 – 30.0 Hz) waves frequency were calculated. Absolute power is the logarithmic value of a variable in each frequency band of the EEG signals, calculated with a root mean square algorithm.

**Statistics**
The unpaired-t test was used for comparison.

**Results**
**Table 1 Absolute Power Of EEG waves in 20 Electrodes, In depression And Healthy Control.**

| LEADS | BANDS | DELTA | | THETA | | ALPHA | | BETA | |
|---|---|---|---|---|---|---|---|---|---|
| | | MEAN | S.D. | MEAN | S.D. | MEAN | S.D. | MEAN | S.D. |
| FP1 | DEP. | 15.31 | 8.44 | 5.28 | 2.81 | 4.22 | 2.44 | 4.08 | 1.95 |
| | HEALTHY | 13.76 | 10.99 | 4.75 | 3.27 | 3.71 | 2.23 | 3.45 | 2.16 |
| FP2 | DEP. | 19.16 | 8.14 | 6.59 | 2.36 | 5.46 | 1.91 | 5.47 | 1.97 |
| | HEALTHY | 15.66 | 9.16 | 5.41 | 3.64 | 4.17 | 2.43 | 4.26 | 2.43 |
| F3 | DEP. | 22.36 | 28.67 | 7.72 | 9.75 | 5.80 | 6.13 | 6.08 | 7.42 |
| | HEALTHY | 12.79 | 6.51 | 5.10 | 2.97 | 4.01 | 2.12 | 3.53 | 1.59 |
| F4 | DEP. | 11.15 | 9.78 | 3.45 | 3.00 | 2.64 | 2.59 | 2.90 | 2.43 |
| | HEALTHY | 9.06 | 4.36 | 3.18 | 1.73 | 2.61 | 1.67 | 2.54 | 1.24 |
| C3 | DEP. | 15.00 | 7.53 | 5.61 | 2.34 | 4.42 | 1.87 | 4.50 | 1.93 |
| | HEALTHY | 10.38 | 5.65 | 4.21 | 2.75 | 3.31 | 2.04 | 3.02 | 1.63 |
| C4 | DEP. | 13.32 | 8.40 | 4.18 | 2.73 | 3.17 | 2.32 | 3.46 | 2.17 |
| | HEALTHY | 10.56 | 5.37 | 3.94 | 2.61 | 3.29 | 2.24 | 2.90 | 1.56 |
| P3 | DEP. | 13.02 | 8.70 | 4.37 | 2.87 | 3.85 | 2.92 | 3.50 | 2.30 |
| | HEALTHY | 10.03 | 4.95 | 4.47 | 3.66 | 3.82 | 2.58 | 2.92 | 1.53 |
| P4 | DEP. | 19.92 | 21.79 | 7.37 | 6.90 | 6.32 | 3.90 | 5.53 | 4.29 |
| | HEALTHY | 9.99 | 4.49 | 4.98 | 3.36 | 4.17 | 2.36 | 3.09 | 1.39 |
| O1 | DEP. | 15.02 | 6.71 | 6.90 | 2.86 | 8.25 | 4.39 | 5.31 | 1.84 |
| | HEALTHY | 10.71 | 5.35 | 5.58 | 4.76 | 6.36 | 4.11 | 3.65 | 1.95 |
| O2 | DEP. | 24.86 | 33.59 | 10.52 | 13.39 | 10.94 | 8.11 | 7.70 | 7.96 |
| | HEALTHY | 12.20 | 5.70 | 6.34 | 4.51 | 7.97 | 4.25 | 4.24 | 1.98 |
| F7 | DEP. | 16.36 | 7.26 | 5.92 | 1.94 | 5.47 | 3.04 | 5.00 | 1.98 |
| | HEALTHY | 14.17 | 7.87 | 5.16 | 2.88 | 4.18 | 2.53 | 4.19 | 2.32 |
| F8 | DEP. | 17.71 | 7.61 | 6.57 | 2.27 | 5.63 | 1.95 | 5.44 | 2.31 |
| | HEALTHY | 15.10 | 8.30 | 5.41 | 3.11 | 4.16 | 2.02 | 4.35 | 2.31 |
| T3 | DEP. | 15.36 | 9.44 | 5.49 | 2.06 | 4.68 | 1.61 | 5.05 | 2.19 |
| | HEALTHY | 11.16 | 6.53 | 4.33 | 2.41 | 3.42 | 1.84 | 3.43 | 1.90 |
| T4 | DEP. | 15.90 | 8.87 | 5.92 | 2.09 | 5.14 | 1.56 | 5.02 | 2.20 |
| | HEALTHY | 10.49 | 5.26 | 4.38 | 2.57 | 3.57 | 1.93 | 3.30 | 1.73 |

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| LEADS | BANDS | DELTA | | THETA | | ALPHA | | BETA | |
|---|---|---|---|---|---|---|---|---|---|
| | | MEAN | S.D. | MEAN | S.D. | MEAN | S.D. | MEAN | S.D. |
| P7 (T5) | DEP. | 14.07 | 6.94 | 5.84 | 2.30 | 6.38 | 3.18 | 4.87 | 1.76 |
| | HEALTHY | 10.48 | 4.97 | 4.59 | 2.74 | 4.70 | 2.35 | 3.28 | 1.59 |
| P8 (T6) | DEP. | 14.67 | 7.23 | 6.19 | 3.06 | 6.78 | 3.48 | 5.03 | 2.03 |
| | HEALTHY | 10.76 | 5.34 | 4.84 | 3.19 | 5.06 | 3.13 | 3.37 | 1.71 |
| Fz | DEP. | 14.86 | 6.80 | 6.20 | 2.77 | 5.11 | 2.36 | 4.48 | 1.82 |
| | HEALTHY | 10.62 | 5.32 | 4.48 | 2.99 | 3.50 | 2.01 | 3.07 | 1.50 |
| Cz | DEP. | 15.48 | 7.40 | 6.41 | 2.49 | 5.29 | 2.27 | 4.66 | 1.93 |
| | HEALTHY | 11.72 | 5.53 | 5.07 | 3.39 | 3.88 | 2.12 | 3.24 | 1.50 |
| PZ | DEP. | 15.38 | 7.21 | 5.99 | 2.50 | 5.55 | 2.81 | 4.55 | 1.99 |
| | HEALTHY | 11.21 | 5.44 | 5.60 | 5.11 | 4.40 | 3.18 | 3.28 | 1.77 |
| OZ | DEP. | 5.65 | 3.46 | 1.56 | 1.53 | 1.22 | 2.07 | 1.47 | 1.10 |
| | HEALTHY | 5.38 | 3.69 | 1.63 | 1.54 | 1.28 | 1.77 | 1.41 | 0.88 |

**Table 2. Statistically significant differences of EEG rhythms between depression patients, and healthy subjects.**

| | Rhythm | | | |
|---|---|---|---|---|
| electrode | Delta | Theta | Alpha | Beta |
| Fp1 | | | | |
| Fp2 | | | *** | |
| F3 | | | | |
| F4 | | | | |
| C3 | *** | | | *** |
| C4 | | | | |
| P3 | | | | |
| P4 | *** | | *** | *** |
| O1 | *** | | | *** |
| O2 | *** | | | *** |
| F7 | | | | |
| F8 | | | *** | |
| T3 | | | *** | *** |
| T4 | *** | | *** | *** |
| P7 (T5) | | | | |
| P8 (T6) | | | | *** |
| FZ | | | *** | *** |
| CZ | | | *** | *** |
| PZ | *** | | | *** |
| OZ | | | | |

(*** p value <0.05)

In depression group mean age was 33.29 years and in healthy control group it was 30.40 years. Out of the 31 of depression patients 19 were male and 12 were female. Out of the 32 of healthy controls 20 were male and 12 were female.

Absolute Power Of EEG Bands Of Delta, Theta, Alpha And Beta In 20 Electrodes, **[Fp1, Fp2, F3, F4, C3, C4, F7, F8, P3, P4, O1, O2, T3, T4, T5, T6, Fz, Cz, Pz and Oz]** In Depression, And Healthy Control shown in table 1 as mean and standard deviation.

**Depression-healthy control**

Patients with depression had significantly higher alpha power over Fp2 ($p<0.051$), P4 ($p<0.015$), F8 ($p<0.017$), T3 ($p<0.027$), T4 ($p<0.003$), Fz ($p<0.012$), and Cz ($p<0.023$) regions than healthy subjects.

Power of beta activity was significantly increased over C3 ($p<0.002$), P4 ($p<0.002$), O1 ($p<0.001$), O2 ($p<0.022$), T3 ($p<0.009$), T4 ($p<0.001$), T6 ($p<0.002$), Fz ($p<0.002$), Cz ($p<0.001$) and Pz ($p<0.002$) regions in patients with depression than healthy control.

Whereas power of delta and theta activity was not shown any statistically differences between depression patients and healthy controls.

**Discussion**

In our study, comparison between patients with depression and healthy subjects showed that patients with depression had significantly increased alpha and beta activity then with healthy subjects, shown in Table 2

According to the conventional EEG studies, 20-40% of patients with depression have abnormal EEG findings. [6, 7]

There are many areas of the brain responsible for emotions and mood. The neuronal circuit responsible for emotions consists of dorsolateral and ventromedial prefrontal cortex, nucleus accumbens, basal ganglia, amygdales, temporal and parietal cortex, and hippocampus Lesions to these structures in patients with depression elicit EEG changes. [8]

**Alpha Band**

In our present study Patients with depression had significantly higher alpha power over Fp2 (right superior frontal gyrus) ($p<0.051$), P4 (right inferior parietal gyrus) ($p<0.015$), F8 (right inferior frontal gyrus) ($p<0.017$), T3 (medial temporal gyrus) ($p<0.027$), T4 (medial temporal gyrus) ($p<0.003$), Fz (medial frontal gyrus) ($p<0.012$), and Cz (superior frontal gyrus) ($p<0.023$) than healthy subjects.

These findings are similar to findings of **(Pollock & Schneider 1990)** where they found increased alpha power. Changes in alpha rhythm are associated with thalamic dysfunction, but they are also associated with cortical activity. It is known that alpha power is reversely proportional with cortical activity in patients with depression. [9]

Alpha oscillations are correlated to brain function such as inhibition, attention, consciousness and primarily generat-

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ed in thalamus, hippocampus, and cortical regions. [10]

**Beta Band**

In the present study Power of beta activity was significantly increased over C3 (left precentral frontal gyrus) ($p<0.002$), P4 (right inferior parietal gyrus) ($p<0.002$), O1 ( left medial occipital lobe) ($p<0.001$),O2 right medial occipital lobe ($p<0.022$),T3 ( left medial temporal gyrus) ($p<0.009$), T4 (right medial temporal gyrus) ($p<0.001$), T6 (right medial temporal lobe) ($p<0.002$), Fz (medial frontal lobe) ($p<0.002$), C3 (superior frontal gyrus) ($p<0.001$) and Pz (precuneus parietal lobe) ($p<0.002$) regions in patients with depression than healthy control. These findings are similar to findings of **(Pollock & Schneider 1990)** where they found increased beta power.

In our study results table 2 demonstrated that the effect of the major depression disorder was most pronounced in the posterior cortex of the brain by showing increased beta activity in O1 and O2 leads. This may indicate that depressive subjects had increased arousal [11] which may reflect a prolonged stress and may serve as a background for psychopathology development. This idea is supported by the fact that the whole posterior region of the cortex in our study was characterized by beta brain oscillations together with other oscillations in depressive subjects. Additionally the greater effect of major depression disorder on the posterior cortex may be explained by the contribution of comorbid anxiety. Posterior part of the brain was significantly related to depression.

In our study we found that distributed EEG effects during major depressive disorder can be explained through the "monoamine concept" which proposes that depression is related to a deficit of monoamines, particularly norepinephrine and serotonin, at critical synapses. [12] It has been shown that activation of serotonin signaling can suppress the GABAergic inhibition. Thus, in serotonin deficit diseases such as major depression disorder, GABAergic signaling in the cortex may be overly potent. Because serotonin is one of the widely distributed neurotransmitters in the central nervous system. [13] It is likely that a disturbance of serotonergic neurotransmission would lead to an increase in the GABAergic signaling in many different brain regions.

**Conclusions:**

The finding with depression differed from healthy subjects in absolute power values of alpha and beta waves.

Replication of the results is essential before a firm conclusion could be drawn.

Other methodological problems associated with EEG studies may need to be resolved before it is possible to identify specific EEG markers for schizophrenia In support of this in a recent review of the application of digital EEG quantitative techniques, it was concluded that regardless of developments in EEG technology the use of QEEG in schizophrenia remains investigational.

Thus, evaluation of EEG can be a relatively inexpensive means to assess absolute power of schizophrenia and depression to find pathophysiology behind clinical symptoms of these psychiatric illnesses.

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