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ALCOHOL DEPENDENCE RELATED CENTRAL BRAIN DEPRESSION: AN ELECTRO-ENCEPHALOGRAPHIC STUDY

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ABSTRACT: Introduction: Chronic alcoholism has been associated with significant neurophysiological alterations, particularly affecting the central nervous system (CNS). Electroencephalography (EEG) studies have provided insights into these changes, revealing patterns of brain activity that differ markedly from those of non-alcoholics. **Materials and Methods:** The study involved EEG power spectral analysis of alcohol-dependent patients compared to healthy controls. The analysis focused on various EEG bands (Delta, Theta, Alpha, Beta, Gamma) across central brain regions (FZ, CZ, PZ, OZ) during eye-open and eye-closed states. **Results:** The findings indicated increased theta and beta power in alcoholics, particularly at central regions (CZ), suggesting alterations in cognitive processing and attention mechanisms. Alpha power was reduced, indicating impaired resting-state cortical activity. Gamma power showed decreased absolute values but increased relative power in posterior regions, suggesting compensatory mechanisms. **Conclusion:** EEG power differences in alcoholics provide evidence of significant neurophysiological alterations, particularly in theta, beta, and gamma bands. These findings could serve as potential electrophysiological signatures for alcohol-induced brain dysfunction and aid in monitoring recovery or treatment.

INTRODUCTION: Alcohol dependence is a chronic neurological disorder characterized by compulsive alcohol consumption, tolerance, and withdrawal symptoms. Chronic alcohol use leads to significant neurophysiological changes, particularly affecting the central nervous system (CNS). Electroencephalography (EEG) is a valuable non-invasive tool for assessing these changes, providing insights into the neurophysiological underpinnings of central brain depression in alcohol-dependent individuals.

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Chronic alcohol use causes widespread cortical dysfunction, evident in EEG changes such as increased slow-wave (theta, delta) and reduced fast-wave (beta, gamma) activity [1, 2], reflecting impaired cortical excitability and neuronal synchrony [3]. Reduced alpha power and coherence further indicate disrupted thalamocortical connectivity, contributing to cognitive impairments [4].

These EEG abnormalities correlate with disease severity and are more pronounced with prolonged alcohol use and repeated withdrawal episodes [5]. Neuroimaging and neurophysiological studies have consistently demonstrated widespread cerebral changes, particularly involving the frontal and central regions, which are crucial for executive function, motor coordination, and cognitive processing [6, 7].

The central brain region, encompassing motor and sensorimotor areas, is particularly vulnerable to alcohol-induced neurotoxicity, which may explain deficits in psychomotor performance and higher cognitive dysfunction observed in alcoholics [8]. Spectral power analysis of EEG frequency bands is crucial for the early detection and monitoring of alcohol dependence, as well as for evaluating therapeutic interventions. The present study explores central brain depression in alcoholics through EEG power spectrum analysis, comparing it with non-alcoholic controls.

MATERIAL AND METHODS:

Study Design and Participants: This case-control analytical study was conducted after obtaining approval from the Institutional Research Review Board and Ethics Committee (352/MC/EC/2020). Written informed consent was obtained from all participants prior to recruitment. The study population comprised 30 male subjects with alcohol use disorder (AUD) and 30 age-matched healthy male controls, aged 25–50 years, recruited from the same population.

Inclusion and Exclusion Criteria: AUD patients were identified using the Alcohol Use Disorders Identification Test (AUDIT) [9]. Participants with an AUDIT score more than seven were classified as having AUD. Controls were non-alcoholic, free from acute or chronic medical conditions, and within the same age group. Exclusion criteria included neuropsychiatric disorders, history of head injury, use of psychotropic drugs or other treatments, any chronic disease, other addictions, and uncooperative behavior.

EEG Recording Procedure: Participants were instructed to wash their scalp the night before the recording and refrain from applying oil, sprays. The subjects were also asked to avoid all foods and drinks containing caffeine for at least two hours before the initiation of the test procedure. EEG recording was conducted in mornings hours of the day between 8 to 10 am., in quiet, comfortable, temperature controlled, dim light room conditions, in sitting posture with clear instructions (eye open or close, with relaxation) to ensuring accurate research data. Resting-state EEG was recorded under two physiological conditions eyes closed (5 min) and eyes open (5 min). Scalp electrodes were placed according to the International 10–20 System at midline frontal FZ, central CZ, parietal PZ, occipital OZ sites, using saline-soaked Ag/AgCl electrodes.

Raw EEG signals were recorded using the Brain Electro Scan System (BESS, version 4.0; Axxonet Systems Technologies Ltd., India). Electrode impedance was maintained below 5 KΩ. Signals were amplified and filtered using a bandpass filter (0.5–70 Hz) and notch filters (50 Hz) to eliminate electrical noise. Data were digitized at a sampling rate of 512 Hz. Artifact removal was performed after EEG recording by manually selecting and rejecting noisy epochs that could interfere with the results.

EEG Feature Analysis: Power spectral analysis was performed using the Welch method to estimate EEG power across frequency bands. The continuous EEG signal was segmented into 1000-ms overlapping epochs, windowed with a Hamming function to reduce spectral leakage, and transformed using the fast Fourier transform (FFT). Periodograms from individual epochs were then averaged across epochs for each electrode and experimental condition to obtain a smooth, low-variance power spectrum.

Absolute spectral power (μV^2) was computed across five frequency bands: Delta (0.5–4 Hz), Theta (4–7 Hz), Alpha (7–13 Hz), Beta (13–30 Hz), and Gamma (30–70 Hz). Data were processed using BESS software, with averaged values calculated separately for each electrode site and condition.

Relative power calculated in percentage by following formula:

$$\text{Relative Power}_{\text{Brand}} = \text{Absolute Power}_{\text{Brand}} / \text{Total absolute power}$$

Statistical Analysis: Data normality was evaluated using the Shapiro–Wilk test, and homogeneity of variances was tested using Levene's test. All variables included in the parametric analyses satisfied these assumptions. The continuous variables were expressed as mean ± standard deviation (SD). Statistical analyses were conducted using Epi Info software, version 7.2.1.0. Between-group comparisons were performed using the

unpaired t-test, with a significance threshold of $p \leq 0.05$.

RESULTS: The present study was a case–control comparative study conducted on 30 male alcohol-dependent subjects (cases) and 30 age-matched healthy male subjects (controls).

The **Table 1** depict that in the central brain region during the eye-open state, alcoholics showed significantly reduced absolute beta (2.09 ± 1.44 vs. 2.9 ± 1.53, *p*=0.039) and gamma power (0.47 ± 0.28 vs. 0.87 ± 0.74, *p*=0.008) at CZ compared to controls. Relative power analysis revealed highly significant elevation of delta (66.74 ± 7.16 vs. 56.38 ± 14.32, *p*=0.001) and significant reduction of theta, beta, and gamma at CZ, along with decreased gamma relative power at OZ (*p*=0.007).

Alcoholics exhibited abnormal EEG activity, characterized by increased relative power of slow-wave (delta) and reduced fast-wave (beta and gamma) power in central regions.

TABLE 1: COMPARISON OF THE ABSOLUTE AND RELATIVE POWER OF EEG (MEAN ± SD) IN CASES (ALCOHOLICS) AND CONTROLS (NON-ALCOHOLICS) DURING EYE OPEN STATE IN CENTRAL BRAIN REGION

EEG	Delta		Theta		Alpha		Beta		Gamma	
Channels	Controls	Cases	Controls	Cases	Controls	Cases	Controls	Cases	Controls	Cases
Absolute power (μV^2)										
FZ	17.68 ± 8.97	17.4± 11.93	4.38 ± 2.28	4.64± 5.63	2.35 ± 1.31	2.61± 2.13	2.2 ± 1.11	2.75± 2.47	0.6 ± 0.36	0.66± 0.48
p-value	0.920		0.818		0.580		0.267		0.608	
CZ	21.43 ± 18.4	21.96± 9.94	8.43 ± 8.35	5.16± 4.97	3.42 ± 2.52	2.44± 1.82	2.9 ± 1.53	2.09± 1.44	0.87 ± 0.74	0.47± 0.28
p-value	0.890		0.070		0.090		0.039*		0.008*	
PZ	18.94 ± 8.02	21.81 ± 10	5.64 ± 4.9	6.27± 4.62	2.86 ± 1.23	3.17± 2.17	2.63 ± 1.16	2.95 ± 2.5	0.84 ± 0.72	0.8 ± 0.68
p-value	0.225		0.611		0.504		0.532		0.849	
OZ	18.61 ± 10.96	25.01± 21.38	5.17 ± 3.82	7.06± 8.75	2.83 ± 1.58	4.31± 6.37	3.05 ± 2.08	4.14± 7.66	0.98 ± 0.7	0.97± 1.58
p-value	0.150		0.283		0.222		0.454		0.976	
Relative power										
FZ	62.63 ± 8.01	60.93 ± 9.07	16.19 ± 3.34	15.67± 4.53	9.04 ± 3.08	9.76± 2.71	9.29 ± 3.06	10.6 ± 3.9	2.85 ± 1.63	3.0± 1.65
p-value	0.444		0.617		0.337		0.154		0.651	
CZ	56.38 ± 14.32	66.74 ± 7.16	21.79 ± 12.77	15.45 ± 3.9	9.7 ± 3.14	8.16± 2.96	9.2 ± 2.53	7.71± 2.71	2.94 ± 1.85	1.9± 0.66
p-value	0.001**		0.012*		0.057		0.032*		0.007*	
PZ	59.38 ± 13.15	60.9± 12.89	18.04 ± 10.03	17.83 ± 9.69	10.05 ± 3.72	9.5 ± 3.1	9.48 ± 2.8	9.0± 2.76	3.06 ± 1.95	2.6± 1.29
p-value	0.635		0.935		0.540		0.570		0.310	
OZ	56.99 ± 13.7	61.6± 10.15	18.75 ± 9.51	16.91 ± 7.36	9.97 ± 4.02	10.01± 2.42	10.67 ± 4.43	8.94 ± 2.5	3.62 ± 2.01	2.4± 1.21
p-value	0.136		0.405		0.957		0.068		0.008*	

*P ≤0.05 (Significant), **P=0.001 (Highly Significant)

In the **Table 2** shows that during the eye-closed state, alcoholics showed significantly reduced absolute theta power at FZ (4.02 ± 2.31 vs. 5.6 ± 3.37, *p*=0.038) and at CZ (3.94 ± 1.78 vs. 7.39 ± 6.52, *p*=0.07).

At CZ, absolute alpha (2.26 ± 1.36 vs. 4.17 ± 2.28, *p*=0.001), beta (1.78 ± 1.1 vs. 2.86 ± 1.29, *p*=0.001), and gamma (0.39 ± 0.17 vs. 0.79 ± 0.63, *p*=0.002) powers were also significantly reduced. Relative power analysis revealed significantly increased delta with reduced theta, alpha, beta, and gamma at CZ, along with decreased beta and gamma at OZ.

Alcoholics exhibited reduced absolute and relative fast-wave activity (theta, alpha, beta, gamma) particularly in central regions.

TABLE 2: COMPARISON OF THE ABSOLUTE AND RELATIVE POWER OF EEG (MEAN ± SD) IN CASES (ALCOHOLICS) AND CONTROLS (NON-ALCOHOLICS) DURING EYE CLOSE STATE IN CENTRAL BRAIN REGION

EEG Channels	Delta		Theta		Alpha		Beta		Gamma	
	Controls	Cases	Controls	Cases	Controls	Cases	Controls	Cases	Controls	Cases
Absolute power (μV^2)										
FZ	19.64 ± 11.37	15.28 ± 6.33	5.6 ± 3.37	4.02± 2.31	3.5 ± 2.39	2.71± 1.41	2.8 ± 1.82	2.1 ± 1.73	0.67 ± 0.46	0.48 ± 0.33
p-value	0.071		0.038*		0.124		0.130		0.061	
CZ	16.49 ± 5.89	18.15 ± 6.64	7.39 ± 6.52	3.94± 1.78	4.17 ± 2.28	2.26± 1.36	2.86 ± 1.29	1.78 ±1.1	0.79 ± 0.63	0.39 ± 0.17
p-value	0.310		0.007*		<0.001**		0.001**		0.002*	
PZ	18.94 ± 9.04	19.59 ± 11.74	5.45 ± 3.36	6.5 ± 4.72	3.72 ± 1.96	3.72± 1.87	2.7 ± 1.11	2.82± 1.71	0.84 ± 0.62	0.83 ± 0.62
p-value	0.812		0.323		0.991		0.736		0.959	
OZ	18.54 ± 10.52	21.59 ± 15.7	4.74 ± 2.26	5.47± 5.37	3.4 ± 1.9	4.07± 3.75	3.11 ± 1.89	2.92± 4.08	0.92 ± 0.63	0.65 ± 0.75
p-value	0.380		0.496		0.384		0.817		0.131	
Relative power										
FZ	58.87 ± 10.95	60.32 ± 8.99	17.37 ± 3.29	17.05± 6.56	11.58 ± 5.06	11.32± 3.69	9.71 ± 3.55	8.96± 3.82	2.46 ± 1.33	2.36 ± 1.26
p-value	0.577		0.808		0.815		0.435		0.756	
CZ	52.48 ± 13.49	65.88 ± 8.95	21.87 ± 10.98	16.07 ± 5.77	13.27 ± 5.31	8.67 ± 3.6	9.7 ± 2.61	7.48± 2.98	2.68 ± 1.62	1.91 ± 0.61
p-value	0.003*		0.013*		0.002*		0.003*		0.018*	
PZ	56.78 ± 13.93	55.18 ± 15.2	17.94 ± 7.64	19.94 ± 12.1	12.8 ± 6.13	12.21 ± 5	9.53 ± 2.87	9.65 ± 2.9	2.95 ± 1.84	3.02 ± 1.58
p-value	0.674		0.446		0.683		0.869		0.889	
OZ	56.17 ± 11.28	60.56 ± 10.38	17.88 ± 6.53	16.37 ± 7.58	11.95 ± 5.4	12.4 ± 4.76	10.68 ± 4.51	8.49± 2.87	3.32 ± 1.95	2.18 ± 0.85
p-value	0.122		0.412		0.734		0.029*		0.005*	

*P ≤0.05 (Significant), **P=0.001 (Highly Significant)

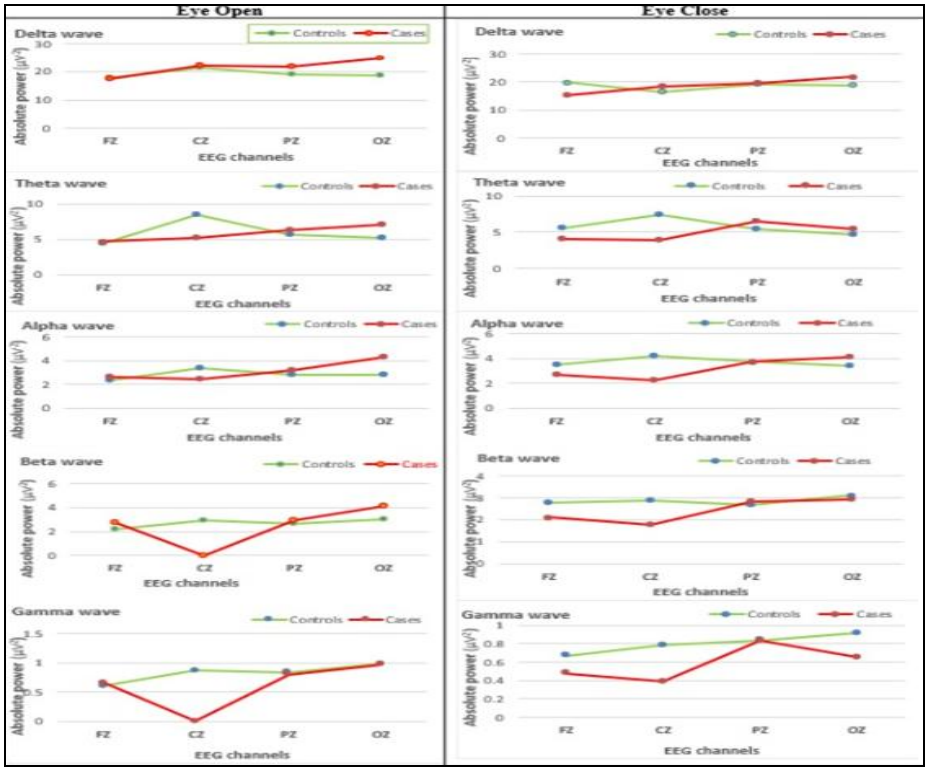

FIG. 1: EEG ABSOLUTE POWER SPECTRUM ANALYSIS IN CENTRAL BRAIN REGIONS IN ALCOHOLICS (CASES) AND NON-ALCOHOLICS (CONTROLS) DURING EYE-OPEN AND EYE-CLOSED STATE

The graphical comparison **Fig. 1** of EEG absolute power spectra between alcoholics and controls during eye-open and eye-closed states highlights distinct alterations. For delta waves, cases consistently demonstrated higher power, particularly at CZ and OZ, with a pronounced increase during the eye-closed state. Theta, alpha, beta, and gamma waves were generally lower in cases compared to controls, most evident at CZ in both conditions. In the eye-open state, alcoholics showed marked reductions in beta and gamma power at CZ, whereas in the eye-closed state, significant declines in theta, alpha, beta, and gamma power at CZ were observed. Overall, controls maintained balanced spectral activity across regions, while cases showed a shift toward slower-wave predominance. The figure indicates that alcoholics reduced fast-wave (theta, alpha, beta, gamma) activity, particularly in central regions.

DISCUSSION: The present study examined electroencephalographic (EEG) activity in alcohol-dependent individuals compared with healthy controls during eye-open and eye-closed resting states, with a particular focus on the central brain regions. Our findings reveal a characteristic slowing of EEG rhythms in alcoholics, manifested as increased delta power and concomitant reductions in theta, alpha, beta, and gamma power, particularly at central (CZ) and occipital (OZ) sites. These alterations provide evidence of central brain depression associated with chronic alcohol consumption. A prominent observation was the significant elevation in relative delta power among alcoholics, evident in both eye-open and eye-closed states. This finding is consistent with previous studies that reported enhanced slow-wave activity in alcohol-dependent individuals, reflecting cortical hypoarousal and impaired neuronal synchronization [10, 11]. Increased delta activity has been interpreted as an indicator of diffuse cortical dysfunction, possibly related to alcohol-induced structural and functional brain changes, including reduced gray matter volume and neuronal loss [12, 13].

Conversely, we observed significant reductions in fast-frequency oscillations (beta and gamma) in alcoholics, particularly at CZ during both resting states. Beta and gamma rhythms are closely linked to higher-order cognitive processing, attention, and sensorimotor integration [14]. Decrements in these bands suggest impaired thalamocortical connectivity and disrupted excitatory-inhibitory balance in alcoholism [1]. These alterations may underlie cognitive deficits such as reduced executive function, working memory impairment, and attentional deficits commonly documented in alcohol dependence [15, 16].

Theta and alpha bands also showed reduced absolute power in alcoholics, particularly at CZ during the eye-closed state. Alpha oscillations are considered markers of cortical idling and inhibitory control, while theta is associated with memory encoding and cognitive control [17]. Attenuation of these rhythms may reflect disrupted oscillatory coordination between cortical and subcortical structures. Previous studies have reported diminished alpha and theta in alcoholics, correlating with poor neuropsychological performance and increased relapse risk [18, 19].

The predominance of delta activity, coupled with suppression of theta, alpha, beta, and gamma, suggests a shift toward slower-wave dominance in alcoholics. This pattern is indicative of central brain depression, consistent with the neurotoxic effects of prolonged alcohol exposure on cortical circuits [20]. Functional MRI studies also corroborate our EEG findings, showing hypoactivation in prefrontal and central regions in alcoholics during cognitive tasks [21].

Overall, our findings provide robust evidence that alcohol dependence is associated with central brain depression, reflected in spectral slowing and impaired fast-frequency activity. These EEG signatures may serve as potential electrophysiological signatures for assessing the severity of alcohol-related brain dysfunction and monitoring therapeutic outcomes. Future longitudinal studies are warranted to determine whether normalization of EEG rhythms accompanies abstinence or treatment, thereby offering insights into neuroplastic recovery in alcoholism.

Strengths and Limitations:

Strengths: A major strength of this study is the use of quantitative EEG analysis across both eye-open and eye-closed resting states, which allowed us to

capture state-dependent alterations in spectral dynamics among alcohol-dependent individuals. The inclusion of both absolute and relative power analyses provided a comprehensive understanding of frequency-specific changes, particularly in central brain regions. Moreover, the case-control design with age-matched participants minimized potential confounding related to age-related EEG variability. The focus on central regions of the brain, which are highly relevant for cognitive and sensorimotor integration, adds neurophysiological validity to the findings.

Limitations: Several limitations should be acknowledged. First, the sample size was relatively modest, which may limit the generalizability of the findings. Second, the cross-sectional design precludes causal inferences regarding whether EEG alterations are a consequence of chronic alcohol use or represent predisposing vulnerability markers. Third, only male participants were included, which restricts the applicability of results to female alcoholics who may exhibit different neurophysiological patterns. Additionally, potential confounders such as nutritional deficiencies, co-morbid psychiatric conditions, and duration or severity of alcohol dependence were not fully controlled. The study was limited by a restricted channel montage, absence of abstinence and withdrawal quantification, possible lack of correction for multiple comparisons, no correlation with clinical severity, and incomplete characterization of alcohol-use patterns and withdrawal status.

CONCLUSIONS: The present study demonstrates that alcohol dependence is associated with marked alterations in EEG spectral activity, characterized by increased delta power and reductions in theta, alpha, beta, and gamma rhythms, particularly in central region of the brain. These findings reflect a shift toward slower-wave dominance, indicative of central brain depression and impaired cortical processing. The attenuation of fast-frequency oscillations suggests disrupted thalamocortical connectivity and cognitive dysfunction commonly observed in alcoholism. These EEG waveforms may serve as potential electrophysiological signatures for assessing the severity of alcohol-induced neural impairment and could be valuable in monitoring therapeutic interventions or recovery during abstinence.

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CONFLICTS OF INTEREST: Nil

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