



Decoding Brainwaves: Electroencephalographic Power Alterations in Alcoholics Compared to Non-Alcoholics in Multimodal Association Areas

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ABSTRACT

Chronic alcoholism poses a significant public health concern, often leading to and functional and structural brain changes. These changes in the multimodal area of the brain can be evaluated using electroencephalographic (EEG), which reflects underlying neurophysiological dysfunctions. This case-control analytic study involved 30 alcoholic males aged 25 to 50 years, compared with 30 age-matched non-alcoholic controls. Alcoholics were identified using the alcohol use disorder identification test (AUDIT) criteria. EEG recordings were obtained from the temporoparietal and occipital regions during eye-open and eye-closed states. Continuous variables were expressed as mean \pm SD, with a significance level set at $p < 0.05$.

In the alcoholic group, alpha and beta band power were significantly higher in the temporal region. In contrast, gamma power was significantly reduced in the parieto-occipital regions during the eye-open state. Similarly, alpha and beta band power were elevated in the temporal regions during the eye-closed state, whereas gamma power was significantly lower in the occipital region (O_2). The radar charts revealed evenly distributed brain wave powers in controls, whereas alcoholics showed reduced overall power, except in the left temporoparietal region.

The findings indicate increased alpha and beta power in the left temporoparietal region, reduced gamma power, and a generalized slowing of higher cognitive functions. These changes reflect impaired neurophysiological processes, particularly those associated with language, emotional regulation, and cognitive flexibility, which are linked to alcohol-induced brain dysfunction.

Keywords: Alcoholism, Brain, Cognition, Electroencephalography, Emotional Regulation, Public Health

INTRODUCTION

Alcohol use disorder (AUD) is a significant public health issue with far-reaching consequences for cognitive and neurological functioning. AUD is a chronic and relapsing condition defined by compulsive alcohol consumption, impaired control over drinking, and distressing emotional states during withdrawal.[1] Prolonged alcohol consumption leads to functional and structural brain changes, including grey matter atrophy, decreased white matter integrity, and disrupted functional connectivity, particularly in the temporo-parieto-occipital (TPO) multimodal association area.[2,3] The TPO region integrates sensory inputs across visual, auditory, and somatosensory modalities, supporting essential cognitive functions such as attention, memory, spatial reasoning, and decision-making.[4-6]

Electroencephalography (EEG), a non-invasive method for measuring brain activity, offers valuable insights into neural oscillations and connectivity. Studies of EEG power spectra in alcoholics have consistently reported alterations across alpha, beta, theta, and gamma bands, indicating disrupted neural dynamics and impaired cortical communication.[7,8] These EEG changes reflect underlying neurophysiological dysfunctions, highlighting

the cumulative impact of chronic alcohol exposure on the brain.[9] While many studies have examined EEG abnormalities in individuals with AUD, limited attention has been given to the TPO region—a critical hub for multimodal information processing. This study aims to investigate and compare EEG power in the TPO region of individuals with AUD and non-alcoholic controls to identify specific neural deficits associated with chronic alcohol use. By analyzing differential EEG activity patterns, this research seeks to deepen our understanding of the neurophysiological effects of AUD and their implications for cognitive health. Insights from these findings could illuminate the pathophysiology of AUD and guide the development of targeted interventions and neurorehabilitation strategies.

MATERIAL AND METHODS

Study Design

This study was a case-control type of analytical study conducted after obtaining clearance from the Institutional Research Review Board (IRRB) and Ethics Committee (352/MC/EC/2020). Written informed consent was obtained from all participants prior to inclusion.

Study Population

The study group comprised 30 male alcoholics and 30 age-matched healthy control subjects, aged 25 to 50 years, recruited from the same population. Inclusion and exclusion criteria were strictly followed.

Inclusion criteria

Alcoholic subjects were identified based on the AUDIT criteria.[10] Participants with an AUDIT score greater than seven were classified as having AUD. Healthy control subjects were non-alcoholic individuals within the same age group and free from any acute or chronic medical conditions.

Exclusion criteria

Participants with neuropsychiatric illnesses, other addictions, a history of head injury, drug or treatment history, or non-cooperative behaviour were excluded.

EEG Data Recording and Analysis

The EEG procedure was explained thoroughly to participants before data collection. Participants were instructed to shampoo their scalp the night before the test and to avoid using hair oil or spray. Additionally, they were asked to abstain from consuming caffeine-containing foods and drinks for at least two hours before the test.

Procedure

Resting-state EEG data were recorded under two physiological conditions for the temporo-parieto-occipital (TPO) region at electrode sites T3, T4, T5, T6, P3, P4, O1, and O2, as per the 10-20 International System[11]:

Eyes Closed (EC): 5 minutes

Eyes Open (EO): 5 minutes

Raw EEG data were recorded using the Brain Electro Scan System (BESS) version 4.0 (Axxonet Systems Technologies Ltd, India). Electrode impedance was maintained below 5 Ω, and signals were amplified using an amplifier. A bandpass filter (0.5–70 Hz) and notch filters (50 and 60 Hz) were applied to eliminate electrical noise and smoothen waveforms. The EEG recordings were digitized at a sampling rate of 512 Hz.

EEG Feature Analysis

Spectral powers were analyzed across five frequency bands: Delta (0.05–4 Hz), theta (4.10–7 Hz), alpha (7.10–13 Hz), beta (13.10–30 Hz), and gamma (30.10–70 Hz).

AUDIT Scoring Levels

The AUDIT scoring levels used were hazardous Level (8–15), harmful level (16–19), and high-risk level (>20).

Statistical Analysis

Continuous variables were expressed as mean ± standard deviation (SD). The mean differences between groups were analyzed using an unpaired t-test, with a *p-value* ≤ 0.05 considered statistically significant. Statistical analysis was performed using Epi Info version 7.2.1.0.

RESULTS

The present study group was conducted on 30 male alcoholics and 30 age-matched healthy control subjects of the age group 25 to 50 years. Table 1 displays EEG absolute power (mean ± SD) in different

Table 1: Comparison of the absolute power (μV_2) of EEG (mean ± SD) in cases (alcoholics) and controls (non-alcoholics) during eye open state in temporo-parietal occipital region

Channels	Delta (Hz)		Theta (Hz)		Alpha (Hz)		Beta (Hz)		Gamma (Hz)	
	Controls	Cases	Controls	Cases	Controls	Cases	Controls	Cases	Controls	Cases
T3	19.48 ± 7.59	24.01 ± 16.02	5.2 ± 3.11	5.42 ± 4.83	2.6 ± 1.64	3.12 ± 2.7	2.43 ± 1.3	2.72 ± 1.76	0.92 ± 0.59	0.75 ± 0.54
T4	23.77 ± 9.66	31.03 ± 27.38	4.59 ± 2.17	6.91 ± 6.22	3.02 ± 2.91	2.97 ± 2.08	2.5 ± 1.06	2.65 ± 1.51	0.7 ± 0.42	0.7 ± 0.47
T5	22.18 ± 10.66	21.16 ± 13.1	4.61 ± 1.98	5.4 ± 3.82	2.45 ± 1.11	3.48 ± 1.92*	2.31 ± 0.98	3.47 ± 1.97*	0.82 ± 0.56	0.83 ± 0.38
T6	21.14 ± 10.9	21.15 ± 11.27	6.24 ± 5.44	5.64 ± 4.43	2.9 ± 2.15	2.76 ± 1.87	2.9 ± 2.31	2.73 ± 2.07	0.96 ± 0.77	0.71 ± 0.47
P3	20.66 ± 14.13	19.34 ± 10.24	5.5 ± 3.66	6.22 ± 4.56	2.92 ± 1.66	3.4 ± 1.58	2.57 ± 1.18	2.99 ± 1.8	0.85 ± 0.63	0.77 ± 0.67
P4	21.12 ± 15.9	17.46 ± 12.03	7.23 ± 7.25	4.35 ± 3.18	3.07 ± 1.99	2.69 ± 1.74	2.95 ± 1.79	2.14 ± 1.51	0.92 ± 0.72	0.46 ± 0.31*
O1	18.84 ± 8.67	19.72 ± 9.89	4.08 ± 1.7	5.33 ± 5.69	2.63 ± 1.49	2.97 ± 1.57	2.53 ± 1.45	2.46 ± 1.5	0.77 ± 0.52	0.67 ± 0.58
O2	19.38 ± 11.98	19.5 ± 9.35	4.66 ± 2.68	4.81 ± 2.66	3.01 ± 1.62	3 ± 1.89	3.07 ± 1.6	2.57 ± 1.98	0.92 ± 0.63	0.63 ± 0.44*

Significance levels: **p* = 0.01 (Significant), ***p* = 0.001 (Highly Significant)

frequency bands (Delta, Theta, Alpha, Beta, Gamma) across channels (T3, T4, T5, T6, P3, P4, O1, O2) for alcoholics (cases) and non-alcoholics (controls) during the participants' eye-open state.

In individuals with chronic alcohol use, distinct patterns of brain activity are observed across various EEG frequency bands. The delta band and theta band power cases exhibit higher power compared to controls in most channels, though these differences are not statistically significant. Significant alterations appear in the alpha band and beta band, where an increase in power is noted at T5 ($p \leq 0.01$). Meanwhile, the gamma band shows decreased gamma power in cases, with significant reductions at P4 ($p \leq 0.001$) and O2 ($p \leq 0.01$). Overall, alcoholics demonstrate increased alpha, beta power and reduced gamma power, indicating altered brain activity patterns likely tied to the neurophysiological effects of prolonged alcohol use.

Table 2 compares the absolute power of EEG waves (Delta, Theta, Alpha, Beta, and Gamma) between controls (non-alcoholics) and cases (alcoholics) during an eyes-closed state across tempo-parietal-occipital EEG channels. There is no statistically significant difference was observed between controls and cases across all channels, except T5, suggesting that delta, theta band power remains largely unaffected by alcohol use in this state. Alpha and beta band power is significantly higher in alcoholics at T5 ($p = 0.01$). This could reflect hyperexcitability or dysregulation in brain activity associated with alcohol use. Gamma band power is significantly reduced in alcoholics at T3 ($p = 0.01$) and O2 ($p = 0.01$). This indicates impaired cognitive processing and neural synchronization in alcoholics, particularly in temporal and occipital regions.

The radar charts (Figure 1) compare the power spectrum of different EEG waves (Delta, Theta, Alpha, Beta, and Gamma) across tempo-parietal-occipital regions of the brain (P3, P4, O1, O2, T3, T4, T5, T6). The groups analyzed are:

- Control (Green) - Individuals with no alcohol addiction.
- Hazardous (Yellow) - Individuals with hazardous levels of alcohol use.
- Harmful (Blue) - Individuals with harmful levels of alcohol addiction.

In the control group, brain wave power is consistent and evenly distributed across all regions. In contrast, the "Hazardous" group shows distinct patterns of brain wave activity. Delta band power is highest at the T4 region, indicating excessive slow-wave activity associated with alcohol addiction. In the "Harmful" group, alpha band power is most pronounced at T5 and P3. The "Hazardous" group also exhibits a slight reduction in beta wave power compared to controls, particularly in the occipital regions (O1, O2), where Beta power measures around 2 units. In the "Harmful" group, beta wave power is significantly reduced across most regions, with the lowest levels observed in the occipital (O1, O2) and parietal (P4) areas, and the highest values recorded in the temporal region (T5). However, both alpha and beta wave power show a marked increase at the P3 and T5 channels. Gamma activity is significantly diminished in the "Harmful" group, except for excessive gamma activity at the T5, T6, and P3 channels.

DISCUSSION

The present study observed increased alpha and beta power and reduced gamma power in alcoholics, suggesting altered brain activity and impaired cognitive processing. Delta and theta bands

Table 2: Comparison of the absolute power (μV_2) of EEG (mean \pm SD) in cases (alcoholics) and controls (non-alcoholics) during eye close state in tempo-parietal occipital region

Channels	Delta (Hz)		Theta (Hz)		Alpha (Hz)		Beta (Hz)		Gamma (Hz)	
	Controls	Cases	Controls	Cases	Controls	Cases	Controls	Cases	Controls	Cases
T3	18.6 \pm 9.4	18.68 \pm 10.32	5.33 \pm 3.9	4.86 \pm 3.56	2.89 \pm 2.2	3.03 \pm 2.03	2.35 \pm 1.2	2.17 \pm 1.24	0.78 \pm 0.46	0.55 \pm 0.28*
T4	22.72 \pm 10.99	31.48 \pm 29.23	5.1 \pm 3.3	5.84 \pm 3.8	3.24 \pm 2.77	2.9 \pm 1.34	2.54 \pm 1.08	2.35 \pm 1.1	0.63 \pm 0.44	0.57 \pm 0.32
T5	20.96 \pm 9.49	16.28 \pm 3.75*	4.52 \pm 1.61	4.94 \pm 1.87	2.65 \pm 1.35	3.79 \pm 1.93*	2.23 \pm 0.87	3.01 \pm 1.45*	0.7 \pm 0.41	0.68 \pm 0.27
T6	22.23 \pm 12.34	19.07 \pm 9.2	5.65 \pm 4	5.38 \pm 3.92	3.28 \pm 2.21	3 \pm 1.6	3.01 \pm 2.19	2.49 \pm 1.44	0.81 \pm 0.63	0.58 \pm 0.31
P3	20.28 \pm 11.45	15.98 \pm 6.58	5.56 \pm 2.69	6.54 \pm 4.69	3.5 \pm 1.96	4.34 \pm 1.86	2.61 \pm 1.01	2.81 \pm 1.61	0.73 \pm 0.43	0.69 \pm 0.65
P4	19.92 \pm 13.59	15.57 \pm 10.04	6 \pm 4.83	5.38 \pm 5.36	3.82 \pm 2.5	3.52 \pm 1.95	2.93 \pm 1.73	2.31 \pm 1.39	0.77 \pm 0.6	0.52 \pm 0.47
O1	20.79 \pm 10.68	19.8 \pm 10.07	4.53 \pm 2.31	4.74 \pm 2.87	3.56 \pm 2.6	3.76 \pm 1.42	2.76 \pm 1.58	2.27 \pm 1.11	0.78 \pm 0.56	0.62 \pm 0.42
O2	19.14 \pm 11.95	18.14 \pm 8.16	4.95 \pm 2.65	5.04 \pm 2.5	4.27 \pm 2.9	4.02 \pm 1.55	3.45 \pm 2.07	2.54 \pm 1.49	0.94 \pm 0.73	0.59 \pm 0.39*

Significance levels: * $p = 0.01$ (Significant), ** $p = 0.001$ (Highly Significant)



Fig 1: Comparison of power spectrum analysis of different EEG waves at tempo-parietal-occipital regions of the brain among control subjects, and persons affected by hazardous and harmful levels of alcohol addiction

are increased with no significant differences noted. Radar charts revealed consistent brain activity in controls, whereas alcoholics exhibited reduced activity except at T5 and P3, with higher left-sided temporoparietal high-frequency activation.

Research on EEG patterns in alcoholics shows varied findings. Newson and Thiagarajan [12] reported increased delta and theta power and reduced alpha, beta, and gamma power in psychiatric disorders, including addiction. Coutin-Churchman *et al.* [13] found reduced slow-band power but increased Beta power in alcoholics. Ceballos *et al.* [14] similarly reported higher delta and theta power in alcoholics, though differences were not statistically significant. Hong

et al. [15] linked high-frequency EEG abnormalities in alcoholics to poorer cognitive functioning. Fein and Allen [16] observed higher EEG power across all bands in treatment-naïve alcoholics, with long-term abuse reducing overall power. Galkin and Kisel [17] found lower alpha power in alcoholics across most regions except antero-temporal and mid-temporal leads.

Studies also highlight higher beta power in centroparietal areas in hazardous alcohol consumption [18] and reduced delta power in at-risk individuals [19]. Galkin and Bokhan [20] noted increased alpha and beta power in frontal-central-parietal-occipital regions and reduced theta power in posterior-temporal areas.

These findings highlight the diverse neurophysiological effects of alcohol use, which are linked to behavioral disinhibition and cognitive impairment. EEG patterns may act as biomarkers for assessing the severity of alcoholism and predicting recovery potential. Research indicates significant differences in brain activity between alcoholics and non-alcoholics, reflecting an imbalance in excitation and inhibition processes in alcoholics.[21] Chronic alcohol use is associated with altered resting-state EEG, characterized by neural hyperactivation and reduced neural communication, some of which may improve with sustained abstinence[22].

The study found that all higher brain functions are reduced in alcoholics, except for increased high-frequency alpha and beta waves in the left temporoparietal area. This suggests that the alcoholic brain remains engaged in language processing, comprehension, and semantic integration during tasks. Elevated high-frequency (alpha and beta) band activity in the left temporal region is also associated with heightened emotional states in alcoholics. Conversely, reduced gamma activity may indicate slower processing speed and reduced precision for left hemisphere-dominant functions, reflecting generalized cognitive slowing due to alcohol-induced brain dysfunction. Gamma oscillations, linked to neural plasticity and adaptive brain changes, showed decreased power, suggesting long-term deficits in learning and memory, particularly related to visual stimuli.

Future Implications

Increased high-frequency alpha and beta activity in the temporoparietal area may serve as a marker of language and emotional instability. Similarly, decreased gamma power at the O2 region could act as a potential biomarker for evaluating the extent of alcohol-induced neurophysiological impairments and tracking recovery progress during abstinence or therapeutic interventions. These findings suggest clinical opportunities for therapeutic strategies, such as visual-cognitive training, mindfulness practices, or neurofeedback, to enhance gamma activity in the occipital regions.

Additionally, integrating EEG findings with neuroimaging or neuropsychological assessments could provide a more comprehensive understanding of alcohol-induced brain changes, aiding in both diagnosis and tailored intervention strategies.

CONCLUSION

The study highlights significant alterations in EEG patterns among alcoholics, including increased alpha and beta power in the left temporoparietal region, reduced gamma power, and generalized slowing of higher cognitive functions. These findings suggest impaired neurophysiological processes, including language, emotional regulation, and cognitive flexibility, linked to alcohol-induced brain dysfunction. EEG biomarkers, such as gamma power at the occipital region, offer potential for assessing the severity of alcoholism and monitoring recovery.

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CONFLICTS OF INTEREST

Nil.

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Nil.

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