

Unveiling Electroencephalographic Changes in Frontal Brain Activity among Alcohol-Dependent Individuals

Khan Rashid¹, Yadav Anuradha², Sharma Preeti³, Sankhla Manisha⁴, Yadav Kavita⁵

¹MD Student, Department of Physiology, SMS Medical College, Jaipur (Rajasthan) India- 302004

²Ph.D, Sr. Professor, Department of Physiology, SMS Medical College, Jaipur (Rajasthan) India- 302004

³Ph.D, Assistant Professor, Department of Physiology SMS Medical College, Jaipur (Rajasthan) India- 302004

⁴Ph.D, Associate Professor, Department of Physiology SMS Medical College, Jaipur (Rajasthan) India- 302004.

⁵Ph.D, Assistant Professor, Department of Physiology, SMS Medical College, Jaipur (Rajasthan) India- 302004

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Corresponding author: Dr. Sankhla Manisha

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Abstract:

Introduction: Alcoholism is a significant social and health problem, and its impact brain function, serving as a crucial risk factor for numerous health problems. Therefore, the present study was undertaken to observe the changes in forebrain activity among alcoholics.

Method: A case-control analytic observational study comprised 30 alcoholics (AUDIT score > 7) and 30 age-matched healthy control (non-alcoholic) male subjects, aged 25 to 50 years. Alcoholic subjects were categorized into hazardous (21 subjects), harmful (9 subjects), and high-risk alcoholic groups based on AUDIT scores. Electroencephalogram recordings of absolute power in delta, theta, alpha, beta, and gamma were recorded in the frontal lobe (FP1, FP2, F3, F4, F7, F8, and FZ) during the eye-open and eye-closed states. Data were analyzed via the unpaired sample "t" test and one-way ANOVA test. Statistical significance was set at P < 0.5 considered significant.

Results: In alcoholic subjects, heightened absolute power in high-frequency waves (alpha, beta, and gamma) across all channels, with significant increases in FP2 and F8 during the eye-open resting state, indicates distinct neurophysiological changes. Low-frequency waves (delta, theta) were notably altered, particularly in F4, during both eye states, providing evidence of an inhibitory state of the frontal area of the brain in alcoholic subjects. The harmful alcohol subgroup demonstrated a significant increase in the beta wave absolute power at FP2.

Conclusions: The study revealed that alcoholic subjects exhibited significantly elevated absolute power in high-frequency waves (alpha, beta, gamma) in FP2 and F8, and altered low-frequency waves (delta, theta), particularly in F4, suggesting asymmetrical EEG patterns in alcoholics.

Keywords: Alcoholism, Electroencephalography, Frontal lobe.

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Introduction

Alcoholism is a significant social and health problem that affects various aspects of human life. Its impact extends beyond brain function and, is a crucial risk factor for numerous health problems. Consequently, alcohol consumption contributes significantly to the global burden.[1] According to the world health Organization in the year 2016 the worldwide 380 million population consuming alcohol and 5.1 % population of higher than 15 years of age.[2] A national health survey of India in the year, 2015-2016 reported that the prevalence of alcohol use disorder was 9 % in the adult males. In India, the alcohol-attributable fraction of all causes of death was 5.4 %, out of which 62.9 % cause of

death was liver cirrhosis. [3] The impact of alcohol on the brain manifests in cognitive disturbances and structural functional changes in the central nervous system. Consuming 0.8 % to 0.15 % alcohol results in slurred speech, impaired balance, short-term memory lapses, slowed thinking, reasoning, and loss of voluntary muscle control. At 0.20 % to 0.30 %, individuals experienced stupor, confusion, reduced muscle control, altered pain perception, heightened risk of injury, increased nausea, vomiting, and diminished gag reflex. Beyond 0.30 %, the risk of unconsciousness and death increases, reaching dangerous levels of 0.41 % and above, potentially inducing coma and

sudden death due to halted heart rate and breathing. [4] Electroencephalography (EEG) serves globally as a non-invasive tool to comprehend neurocognitive phenomena and is a sensitive indicator of pathophysiological processes in alcohol use disorders. EEG signals reflect the electrical activity of cerebral tissue and the functional status of the brain. Distinct brain states associated with specific signals help to understand the functional properties of the human brain. The frontal region is associated with reasoning, motor skills, and higher cognition, with FP1, FP2, and Fz focusing on attention and judgment. F3 and, F4 govern motor planning, whereas F7 and F8 are linked to emotional and verbal expressions. [5] Alcohol addiction is a growing public health concern and, the focus of significant therapeutic and preventive efforts. Understanding the mechanisms of Alcohol Use Disorder (AUD) is crucial to enhance treatment outcomes. This study utilized electroencephalography in patients with AUD to gain insights into the mechanisms of the disorder. Specifically, the study analyzed and compared the power spectrum of the frontal lobe in alcoholic individuals and age-matched healthy controls.

Material and Method

This case-control, analytic, observational study was conducted after clearance from the Institutional Research Review Board and Ethics Committee. The study involved 30 patients (alcoholics) (Audit score > 7) and 30 age-matched healthy controls (non-alcoholic), aged 25 to 50 years, who were recruited after obtaining participant consent and adhering to the inclusion/exclusion criteria. Individuals with neurological or psychotic illness, a history of head injury, any drug or treatment use, and addiction other than alcohol were excluded.

Data Collection

Alcoholic subjects should meet the diagnostic criteria defined by the alcohol use disorder identification test (AUDIT). [6] Study participants with an AUDIT score greater than seven were categorized as Alcohol use disorder (AUD) patients. The Alcohol Used Disorders Identification Test (AUDIT) score developed by the world health organization, to identify person hazardous and harmful pattern of alcohol consumption, it is a simple method of screening alcoholic patient by their AUDIT score and categorize as hazardous, harmful, and high-risk alcoholic group.

AUDIT score categorizes as follow:

- Hazardous level (8-15) = 21 subjects
- Harmful level (16-19) = 9 subjects
- High-risk level (More than 20) = no subjects

The procedure of the experiment was thoroughly explained before recording the electroencephalogram (EEG). Raw EEG data were captured using the Brain Electro Scan System (BESS) version 4.0, from Axxonet Systems Technologies Ltd., India. The electrode impedance was maintained below 5 Ω and the electrical activities were amplified using an amplifier. To eliminate electrical line noise, a bandpass filter 0.5 70 Hz and notch filters (50 Hz and 60 Hz) with a delta of six were applied. Resting-state EEG data were recorded during two physiological conditions: 5 min with eyes closed and 5 min with eyes open. Saline-soaked Ag/AgCl surface electrodes were placed according to the international 10–20 system. [7]

The absolute power of EEG frequency bands (delta, theta, alpha, beta, and gamma) was recorded from seven electrodes in the frontal lobe (FP1, FP2, F3, F4, F7, F8, and FZ). The data were further analyzed using the BESS software, with epochs separated for each trial and a length of 1000 ms. Selective averaging was performed for each electrode site for each condition across all subjects recorded with the BESS software.

Statistical Analysis

Continuous variables were computed and presented as means and standard deviations (SD). Mean differences were analyzed using the unpaired sample " t-test, with a P-value of <0.05 considered statistically significant. Statistical analyses were conducted using the Epi Info version 7.2.1.0 software. To assess the statistical differences among the means of the three groups (hazardous, harmful, and control) based on the AUDIT Score, a one-way ANOVA test was employed.

Results

The high-frequency waves (alpha, beta, and gamma) showed an increase in absolute power among alcoholic subjects in all channels, and statistical significance was observed in FP2 and F8 as compared to the control in the eyes-open resting state. The low-frequency waves (delta, theta) were altered in these cases, and a significant increase was observed for the F4 channel only. (Table 1)

Table 1: Comparison of the absolute power of EEG (mean ± SD) in cases (alcoholics) and controls ((non-alcoholics)) during eye open state in frontal lobe

Channels	Delta		Theta		Alpha		Beta		Gamma	
	Controls	Cases	Controls	Cases	Controls	Cases	Controls	Cases	Controls	Cases
FP1	31.26 ± 22.44	27.19 ± 11.6	7.31 ± 4.41	6.42 ± 3.32	3.06 ± 1.41	3.44 ± 1.57	2.99 ± 1.23	3.84 ± 2.3	0.98 ± 0.54	1.14 ± 0.85
FP2	28.49 ± 15.09	34.31 ± 10.3	7.74 ± 4.96	9.09 ± 4.87	3.24 ± 1.28	3.95 ± 0.97*	3.6 ± 1.27	4.77 ± 2.19*	1.26 ± 0.63	1.71 ± 0.86*
F3	18.86 ± 7.95	17.37 ± 9.81	5.61 ± 3.04	5.93 ± 5.01	2.79 ± 1.64	2.71 ± 2.07	2.8 ± 1.76	2.79 ± 1.76	0.89 ± 0.64	0.89 ± 0.54
F4	20.13 ± 8.74	37.55 ± 26.76**	4.71 ± 1.9	10.62 ± 15.82 *	2.54 ± 1.21	5.5 ± 8.2	2.55 ± 1.09	5.16 ± 8.18	0.73 ± 0.43	1.09 ± 1.56
F7	20.18 ± 12.48	21.53 ± 8.14	4.87 ± 3.85	5.74 ± 6.68	2.78 ± 2.49	2.68 ± 1.63	2.44 ± 1.27	2.93 ± 2.74	0.79 ± 0.64	0.72 ± 0.78
F8	23.43 ± 12.36	21.09 ± 12.42	5.3 ± 4.54	4.09 ± 3.39	2.44 ± 1.2	1.98 ± 1.58	2.59 ± 1.19	2.12 ± 1.78	0.83 ± 0.59	0.55 ± 0.45*
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=0.01(Significant),**P=0.001(Highly Significant)

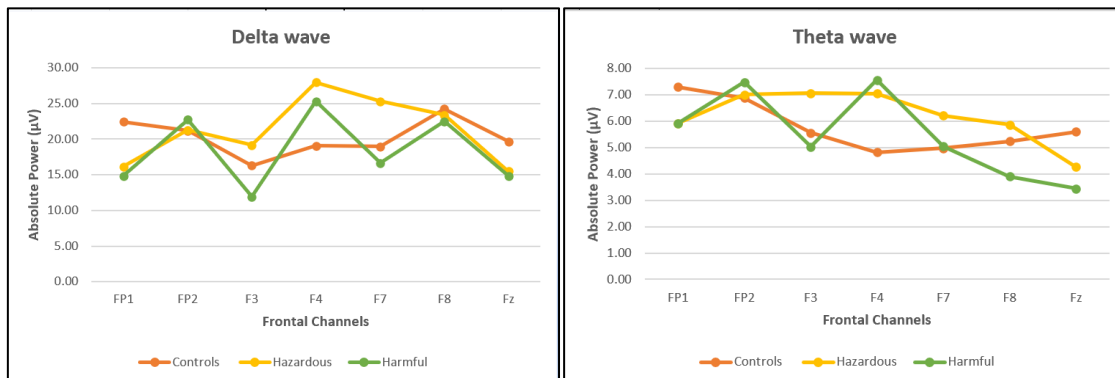
The low-frequency waves (delta, theta) were altered in cases during the eye-close state, but a highly significant increase was observed for the F4 channel of the delta frequency band and a decrease in the Fz channel of the theta frequency band. (Table 2)

Table 2: Comparison of the absolute power of EEG (mean ± SD) in cases (alcoholics) and controls (non-alcoholics) during eye close state in frontal lobe

Channels	Delta		Theta		Alpha		Beta		Gamma	
	Controls	Cases	Controls	Cases	Controls	Cases	Controls	Cases	Controls	Cases
FP1	22.45 ± 23.12	15.76 ± 6.67	7.3 ± 4.31	5.91 ± 2.67	3.54 ± 1.87	4.14 ± 1.78	3.11 ± 1.25	3.52 ± 1.22	1.01 ± 0.55	1.05 ± 0.42
FP2	21.19 ± 9.32	21.7 ± 9.03	6.88 ± 3.56	7.15 ± 4.95	3.6 ± 1.66	4.3 ± 1.72	3.26 ± 0.99	3.84 ± 1.23	1 ± 0.49	1.2 ± 0.48
F3	16.29 ± 6.6	16.98 ± 3.41	5.56 ± 2.57	6.46 ± 5.91	3.15 ± 1.95	2.87 ± 1.79	2.71 ± 1.7	2.49 ± 1.23	0.74 ± 0.41	0.83 ± 0.59
F4	19.08 ± 11.15	27.16 ± 6.69*	4.83 ± 2.21	7.2 ± 7.09	2.98 ± 1.55	4.16 ± 3.85	2.78 ± 1.3	3.18 ± 3.91	0.74 ± 0.48	0.67 ± 0.75
F7	18.98 ± 12.88	22.7 ± 11.92	4.98 ± 3.36	5.86 ± 6.62	2.91 ± 1.92	3.21 ± 1.58	2.36 ± 1.29	2.69 ± 2.38	0.64 ± 0.35	0.65 ± 0.77
F8	24.24 ± 15.23	23.12 ± 14.6	5.24 ± 5.84	5.28 ± 5.67	3.08 ± 3.41	3.45 ± 5.01	2.97 ± 3.04	2.82 ± 3.22	0.77 ± 0.59	0.59 ± 0.56
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=0.01(Significant)

The absolute power of EEG waves in different frequency bands was altered among the hazardous, harmful, and control groups, but there was a statistically significant increase in the beta wave absolute power of the EEG recorded at the FP2 channel. (Figure 1)



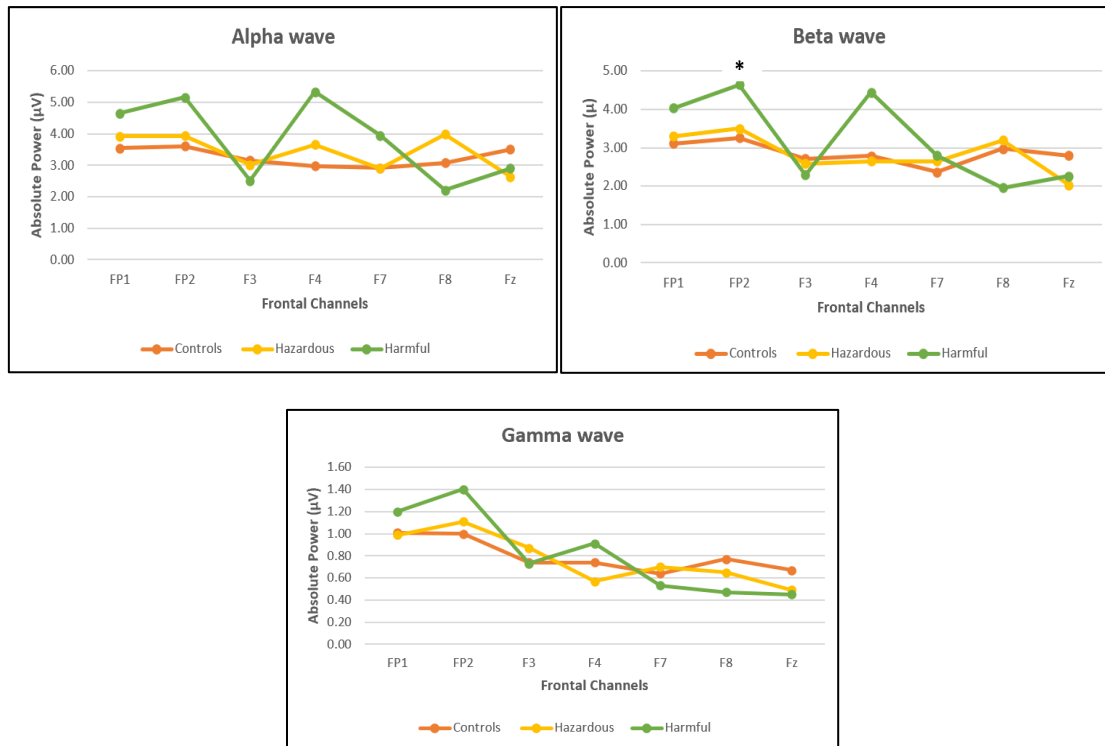


Figure 1: Comparison of the absolute power of EEG in different frequency bands among control (non-alcoholics) and cases (alcoholic) groups (Hazardous and harmful).

Discussion

In the frontal region of the brain (FP1 and FP2 electrodes), the absolute power of the alpha and beta frequency bands was increased in alcoholic subjects in comparison to non-alcoholic (control) subjects. Increased absolute power (delta and theta) was observed at the F4 electrode region of the frontal region of the. In contrast to the frontal areas (FP1 and FP2) the posterior and central frontal areas of the brain (F7, F8, and Fz) showed decreased absolute power in the gamma and theta frequency bands. When we compared the harmful alcoholic group with hazardous and non-alcoholic subjects by ANOVA, no significant difference was observed in various frequency bands between various groups, except for the increase in beta wave absolute power of EEG at the FP2 channel. Delta frequency band, the absolute power significantly increased in alcoholic subjects ($P=0.001$) during the eyes open resting state and ($p=0.032$) eye closed state as compared to control subjects at F4 electrode of frontal lobe; similar results have been reported by previous study. [8] In contrast other studies have found that the delta power of alcoholic subjects is lower than that of healthy controls. [9,10]

Delta waves on electroencephalography increase pathological brain disturbances and sleep. Oscillations in the delta range are supposed to be generated by the neocortical and thalamocortical systems. Decreased delta activity has been recorded in situations that require attention to the outer

environment; however, increased activity has been manifested during cognitive processes that require attention to internal processing. An increase in the delta band is believed to be related to inhibitory mechanisms.[11] The present study also found that dominant delta waves in the frontal regions of the brain provide evidence of an inhibitory state in the frontal area of the brain in alcoholic subjects. Theta frequency absolute power significantly increased ($p=0.046$) in the F4 electrode of the frontal lobe during the eyes-open resting state in the present study, similar to earlier studies. [9,12,13] Similar to the study conducted by Coutin-Churchman and Moreno, the present study also found decreased absolute theta power in alcoholic subjects during the eye-closed state. [14]

The theta wave ranges from to 4-7 Hz, present in the thalamic region of the brain physiologically recorded during the subconscious state and observed in deep relaxation and meditation. A theta power increase may be an electrophysiological indicator of the imbalance between excitatory and inhibitory neurons in the cortex. Increased tonic theta power in EEG may reflect a deficiency in the information-processing capacity of the central nervous system. [15] Alpha, beta and gamma frequency band showed a statically significant increased ($p=0.019$ ($p=0.014$, $p=0.024$, respectively) absolute power at FP2 channel of the frontal lobe during eye open state in alcoholics when compared with normal healthy subjects, this study proves several previous studies. [8,9,16]

An imbalance between excitatory and inhibitory neurons is involved in the predisposition to developing alcohol dependence and proneness to relapse. Alcoholics have shown deficits in Gamma-aminobutyric acid (GABA) receptors for the chemical benzodiazepine, which facilitates inhibitory GABAergic transmission mediated by Gamma Amino Butyric Acid (GABA), the principal inhibitory neurotransmitter of the mammalian nervous system. [17] Benzodiazepines, drugs that modulate GABA receptors, induce beta waves in EEG recordings from humans. [18] In the present study, EEG depicts more alertness of the brain as beta wave absolute power is increased in the frontal lobe (FP2) of the brain during the eye-open state in alcoholic subjects.

In the present study, high-frequency gamma waves showed a statistically significant increase in alcoholic addict's than in their counterparts at F4 in the frontal lobe during the eye-open state. A decrease in gamma indicates a lack of attention, working memory, or long-term memory. As gamma is responsible for fast processing of brain activity, decreased gamma power is responsible for less attention and low cognition status, and delayed processing of information from various senses (smell, sight, and hearing) results in late perception.

Limitations

In the present study, electrooculogram recordings were not taken therefore, the eye blink artifact effect might have influenced the recording.

Conclusion

Significant alterations were exclusively identified in the EEG channels of the right hemisphere in subjects with alcohol consumption. In alcoholic subjects, heightened absolute power in high-frequency waves (alpha, beta, and gamma) across all channels, with significant increases in FP2 and F8 during the eye-open resting state, indicates distinct neurophysiological changes. Low-frequency waves (delta, theta) were notably altered, particularly in F4 in both eye states. The harmful alcohol subgroup demonstrated a significant increase in the beta wave absolute power at FP2. These findings underscore the asymmetrical impact of alcohol on EEG patterns, thereby emphasizing potential markers for neurophysiological assessment.

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