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Immediate and Intermediate Effects of Intensive Alpha Neurofeedback Training on Pain Symptoms and Mood States in Tension Type Headache: A Randomized Sham Controlled Study

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ABSTRACT

Aim: The current study aims to explore the effects of neurofeedback training on primary and associated symptoms of tension type headache (TTH) using a randomized sham control study design. **Methods:** 20 participants with the diagnosis of tension type headache with or without mild to moderate level of depression were recruited for the study after which they were randomly allocated to either the active intervention or the sham group. They underwent ten sessions of alpha enhancing or sham neurofeedback respective to their groups within a span of two weeks. Participants completed an assessment on three baselines i.e. pre intervention, after 5th session and post intervention on domains of mood states, anxiety levels, pain intensity and level of depression in both groups. The groups were compared using repeated measures ANOVA and spearman correlation coefficient was also computed. **Results:** The active neurofeedback group was associated with significant changes in mood states, state anxiety, affective and sensory domains of pain and depression levels when compared to the sham group ($p<0.05$).In addition significant negative correlations were found between the sensori-motor rhythms (SMR) and reported sensory pain. **Conclusion:** The present study provides evidence for efficacy of using alpha neurofeedback training in tension type headache as it has shown to be effective in reducing levels of anxiety, depression and pain in addition to being a non-invasive and time efficient process with minimum placebo effects.

Keywords: *Alpha Neurofeedback, Tension type headache, Mood states*

INTRODUCTION

Headache, an almost universal human experience, is one of the most common complaints encountered in medicine and neurology (Rizzoli et al., 2018). Chronic tension type headache is one of the most prevalent conditions with a lifetime prevalence of 30% to 78% (Kaniecki, 2015) affecting 0.5% to 4.8% of the world population (Yu and Han., 2015). A meta-analysis found that the overall pooled prevalence of headache in India was found to be 438.8 per 1,000 population, which was higher than previously reported data (Dhiman et al., 2021).Chronic tension type headache is also said to be involved in causing emotional difficulties and other co morbidities out of which depression is the most common and next in line are hypertension and anxiety disorders (Caponnetto et al., 2021). A cross sectional study conducted by Ghogare and Patil (2020) in a tertiary health care centre in central rural India found that tension type headache was comorbid with depression (found in 54.1%) and generalized anxiety disorder (found in 70.6%) when majority of the study participants were employed, married, literate and had rural residence. Further a case control study found that depression, negative affectivity, state and trait anxiety were the most co-morbid conditions with chronic tension type headache, therefore indicating in addition to management of pain symptoms, attention should be paid to these conditions as well for better control (Godoy et al., 2022). When sufferers of tension type headache also have a co-morbid conditionof anxiety, depression or underlying personality vulnerabilities it can seriously affect their quality of life, subjective happiness and overall satisfaction with life (Ashina et al., 2020).

Given the costs associated, a well-established, short term treatment plan with associated long term benefits is a key element to effective and holistic treatment of such patients (Jimenez et al., 2015).The pharmacotherapy remains the main resort to headache treatment and that may further lead to feelings of despair and uncontrollability in patients about their conditions. To bridge this gap techniques of neuromodulation and bio-behavioural therapy as a treatment modality may serve as a great benefit for patients suffering from headache (Ailana et al., 2021).

Neurofeedback is a non-invasive kind of biofeedback which targets the imbalanced electrical impulses of the cerebral neurons and is a reward based treatment lying on principles of conditioning. It can help the client learn how to independently manage their pain symptoms, therefore boosting their self- esteem and creating an optimistic view of alleviating their symptoms. A wide variety of neurofeedback (NF) types and protocols have been used for pain management aiming to either increase, decrease or regulate brain activity in certain areas theoretically associated with pain but there is sparse literature on the

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effectiveness of neurofeedback in headache syndromes, especially tension type headache (Roy et al., 2020).There is a need to understand the modulation of EEG activity after intensive neurofeedback training as a method to investigate the neuromodulatory effects due to the relaxation training using alpha protocol. Furthermore, neurofeedback has been found to be effective in wide ranges of conditions like depression (Melnikov.,2021), fibromyalgia (Wu et al., 2021), post-traumatic stress disorder (Steingrimsson et al., 2020), anxiety(Gadea et al., 2020) and other emotional problems (Boland et al., 2020).

In the current study was a sham controlled intensive Alpha neurofeedback training in tension type headache patients by providing ten sessions over the span of two weeks. In addition to the pain symptoms the levels of depression, state and trait anxiety and mood states were also studied. Studies have found that neurofeedback sessions had the potential of reducing pain symptoms and other related outcomes like depression and fatigue (Roy et al.,2020) and its efficacy remained stable over the period of almost 14 months (Nestroniuc et al., 2008). The previous studies have shown mixed outcomes of this training due to which the possible placebo effects of neurofeedback has also been an issue for its treatment efficacy; this has been addressed in this study by giving sham treatment to the control group, further enhancing the utility of the results.

METHODOLOGY

Participants and experimental designs:

The study included 20 patients. The inclusion criteria used for thr selection of the sample were: (i) Those fulfilling the criteria for tension type headache according to the International Classification of Headache disorders- 3rd edition; (ii) Age range of 18-45 years; (iii) who provided consent for the study; and (iv) those who had at least primary level of education and could comprehend English or Hindi.

Purposive sampling of patients with random allocation to either the experimental or control group was done from the Out-patient department (OPD) of the hospital (CIP, Ranchi).

The study protocol was approved by the Institute Ethical Committee (IEC) and Informed consent forms were filled and signed by both the participants and the caregivers and any questions queries related to the procedure were answered before starting the procedure.

The study was composed of three phases (I)Baseline assessment (II)Mid-line assessment (after the 5th session) and (III) Post treatment assessment. The patients were randomly assigned to two groups as follows- (I) the experimental group and (II) the Control group. Every patient was first tested on a baseline according to their moods, state anxiety levels, depression levels, and intensity of pain; then were given ten sessions of intensive neurofeedback training in three rounds of fifteen minutes each which was either real feedback or a sham feedback according to the group they belong to. The patients were blinded to the sham treatment.

Neurofeedback Procedure:

The present study used the Deymed neurofeedback system and the alpha protocol which is used in relaxation. It focused on the sensori motor, beta 1A and theta rhythms. The patient was brought to the laboratory and seated comfortably; the gel was used to place the electrodes on the skin. The electrodes were placed on the C3 and C4 channels, out of which C4 was the main focus of the study because it is involved in emotional processing and relaxation (Warner et al., 2013). The reference electrode was placed at Cz location and the ground electrodes were placed on the forehead.

The real neurofeedback included a video game in which there were rewards in the form of points flashed on the screen, the patient had to focus on the car and be relaxed; therefore, the more the patient was relaxed the better was their alpha activity. When the patient was relaxed, there was rise in alpha activity after which theywere rewarded by a point on the screen which further worked as reinforcement. Initially after 5 sessions the patient was tested on profile of mood states and pain intensities. Soon after the ten sessions over a period of 2 weeks they were again tested on the variables of their mood states, state-trait anxiety levels, pain intensity and depression levels. In the sham neurofeedback the patients were not provided with any visual stimuli but the electrodeswere placed on the same locations and the patients were seated in front of the system after which they relaxed with closed eyes but were not shown the video game and a 45 minute recording was done. They were also tested at the baseline, after the 5th session and after the 10th session in order to rule out the placebo effect and knowing the actual effectiveness of the intervention.

Assessment Methods

The participants were made to fill the following questionnaires: Profile of mood states (POMS) **(Terry et al., 2003),** State- Trait anxiety Inventory (STAI) **(Spielberger., 1983),** Mc Gill Pain Questionnaire **(Melzack& Raja 2005),** Headache Impact test (HIT) **(Kosinki et al., 2003),** Beck's Depression Inventory (BDI) (**Beck, 1961)**and Hamilton's Rating Scale for Depression(HAM-D) **(Hamilton, 1960).**

In addition the Sidedness Bias Schedule **(Mandal et al., 1992)**was used to determine the laterality of the patients and a well standardized Side effect questionnaire called

the discontinuation- emergent signs and symptoms (DESS) **(Rogel et al., 2015)**was administered before the first, fifth and tenth session of neurofeeedback. It includes the emotional, behavioral, cognitive and physical conditions that can be considered possible adverse side effects and is a checklist of 43 symptoms.

DATA ANALYSIS

Statistical analysis was done using the Statistical Package for Social sciences (IBM SPSS Version 25.0) with different parametric and non-parametric measures being used, wherever applicable as follows:

- Chi Square test for comparing discrete/ Categorical variables.
- Mann Whitney U Test for comparing continuous variables.
- Repeated measures ANOVA for comparing various variables between the active and sham over time and group.
- Spearman Correlation Coefficient for finding correlation among the continuous variables under study

In this study two levels of significance ($\alpha <= 0.01$ and $\alpha <= 0.05$) were considered to be statistically significant.

RESULTS

Socio Demographic profile and Comparative description:

The analysis shows that no significant difference was found between the experimental and control groups on variables of sex ($\chi2= 0.952$), age (U= 45.0), marital status ($\chi2= 0.0001$), education ($\chi2= 1.053$), occupation ($\chi2= 0.952$) and habitat ($\chi2= 0.952$).

Table 1: Comparative description of socio demographic variables

Variable		Experimental Mean ± SD/n (%)	Control Mean ± SD/n (%)	χ2/Mann Whitney U	df	p value
Sex	**Male**	4 (40%)	2 (20%)	0.952	1	0.628
	Female	6 (60%)	8 (80%)			
Age	-	27.70±6.83	27.70±4.21	45.00	1	0.722
Marital status	**Married**	2 (20%)	2 (20%)	0.0001	1	1.000
	Unmarried	8 (80%)	8 (80%)			
Education	**Primary**	0	1 (10%)	1.053	1	1.000
	Intermediate	10 (100%)	9 (90%)			
Occupation	**Employed**	6 (60%)	8 (80%)	0.952	1	0.628
	Unemployed	4 (40%)	2 (20%)			
Habitat	**Rural**	2 (20%)	4 (40%)	0.952	1	0.628
	Urban	8 (80%)	6 (60%)			

Clinical Profile and comparison between the groups:

On comparing the clinical variables it was found that there was no significant difference between the experimental and control groups because of drug status ($\chi2= 0.952$), psychiatric co morbidity ($\chi2= 0.267$), family history of psychiatric disorders ($\chi2= 1.250$) or handedness ($\chi2= 0.0001$).

Table 2: Clinical Variable comparison

Variable		Experimental Mean ± SD/n (%)	Control Mean ± SD/n (%)	χ2/Mann Whitney U	df	p value
Drug Status	**On drugs**	8 (80%)	6 (60%)	0.952	1	0.628
	Not on drugs	2 (20%)	4 (40%)			
Psychiatric	**Present**	3 (30%)	2 (20%)	0.267		1.000
Comorbidity	**Absent**	7 (70%)	8 (80%)			
Family	**Present**	3 (30%)	1 (10%)	1.250	1	0.582
History	**Absent**	7 (70%)	9 (90%)			
Handedness	**Right**	9 (90%)	9 (90%)	0.0001	1	1.000
	Left	1 (10%)	1 (10%)			

Table 3: Comparison of clinical scales between the experimental and control group

Variable		Experimental (Mean ± SD)	Control (Mean ± SD)	Time		Time*Group	
				Greenhouse Giesser (F)	Significance	Greenhouse Giesser (F)	Significance
POMS	**Baseline**	15.80 ± 7.8	11.50 ± 5.359	**20.626**	**>0.001***	**14.603**	**>0.001***
	5th session	14.40 ± 7.749	11.50 ± 5.359				
	Post	10.80 ± 6.63	11.10 ± 4.818				
STAI-A	**Baseline**	39.90 ± 12.60	25.80 ± 6.713	**42.722**	**>0.001***	**22.975**	**>0.001***
	Post	33.40 ± 13.14	24.80 ± 5.750				
Trait A	**Baseline**	29.40 ± 12.42	15.70 ± 8.367	1.068	0.315	0.545	0.470
	Post	28.80 ± 11.98	15.60 ± 7.777				
Pain (Affective)	**Baseline**	4.20 ± 1.874	3.40 ± 1.897	**5.784**	**0.015****	**5.784**	**0.015****
	5th session	3.60 ± 1.838	3.40 ± 1.897				
	Post	2.50 ± 1.080	3.40 ± 1.897				
Pain (Sensory)	**Baseline**	3.80 ± 2.044	4.00 ± 1.563	**7.950**	**0.005****	0.127	0.719
	5th session	4.10 ± 2.079	4.20 ± 1.549				
	Post	2.80 ± 1.549	3.20 ± 1.229				
BDI	**Baseline**	12.60 ± 8.07	9.80 ± 7.208	**6.826**	**0.018****	3.303	0.086
	Post	8.70 ± 6.201	9.10 ± 6.983				
HAM-D	**Baseline**	11.0 ± 7.630	8.90 ± 7.695	3.973	0.062	2.584	0.125
	Post	8.20 ± 5.613	8.60 ± 7.058				

On comparing the clinical scales between experimental and control group (From Baseline to post assessment) it was found that there was a significant difference between the mood states (F=20.62) and state anxiety (F = 47.722). There were no significant differences found in trait anxiety and the affective domain of pain but the sensory domain showed a significant difference (F= 7.950). The depression levels had reduced from the different assessment levels but were not significant enough.

Comparison of EEG frequencies (Alpha and Theta) between active and sham group:

When comparison wasdone with the frequencies of alpha and theta waves between the experimental group which received the active intervention to the sham group who did not receive an intervention; there was no significant difference between the two groups on either the Alpha waves (F=0.432) or the Theta waves (F= 0.329).

Table 4: EEG Frequency comparison between groups

Variable		Experimental (Mean ± SD)	Control (Mean ± SD)	Time		Time*Group	
				Greenhouse Giesser (F)	Significance	Greenhouse Giesser (F)	Significance
	Baseline	13.020 ± 2.07	12.80 ± 5.41				
ALPHA	**5th session**	13.30 ± 2.11	11.610 ± 2.84	0.432	0.652	2.099	0.141
	Post	14.59 ± 1.66	11.560 ± 5.10				
	Baseline	32.10 ± 8.50	21.52 ± 4.25				
THETA	**5th session**	31.84 ± 8.82	23.21 ± 4.86	0.329	0.695	4.055	0.031
	Post	28.80 ± 6.14	24.500 ± 4.12				

Table 5: Differences between EEG frequencies (Beta 1a and SMR) within the experimental group

Variable		Experimental (Mean ± SD)	Time	
			Greenhouse Giesser (F)	Significance
BETA 1a	**Baseline**	8.230 ± 3.54	0.018	0.955
	5th session	8.420 ± 1.51		
	Post	8.300 ± 1.64		
SMR	**Baseline**	8.130 ± 2.018	2.568	0.122
	5th session	8.70 ± 1.31		
	Post	9.60 ± 1.61		

The differences in Beta 1a and SMR (Sensori Motor Rhythms) within the experimental group itself from baseline (1st session) to the last session (10th) did not reveal a significant difference with F ratio of 0.018 and 2.568 respectively.

Correlations among the clinical variables and EEG frequencies of pre assessments:

The correlation between the clinical scales and the wave frequencies at the baseline (1st session) reveal that there was a significant negative correlation between the sensory domain of pain and the EEG frequency of theta and Sensori Motor Rhythm (SMR) which shows that the greater the theta inhibition and increase in sensori motor rhythm (SMR), the lesser will be the sensory pain. There was also a significant negative correlation between the depression scores and the EEG frequency of theta which reveals that the greater the theta inhibition the lesser will be the score of depression.

Table 6: Correlations at baseline:

	Alpha Baseline	**Beta1A Baseline**	**Theta Baseline**	**SMR Baseline**
POMS Baseline	.090	.234	-.551	-.111
STAI-A Baseline	-.342	.513	.094	.388
TraitA Baseline	-.316	.015	.338	.151
Pain A Baseline	-.024	.356	-.188	.092
Pain S Baseline	.445	-.608	**-.727****	**-.720****
HIT	-.0162	0.128	-.335	-.296
BDI Baseline	.195	.167	**-.668****	-.219
HAMD Baseline	.0113	.049	-.622	-.306

Correlations among the clinical variables and EEG frequencies of post assessments:

The correlation between the clinical scales and the wave frequencies at the post assessment (10th session) reveal that there was a significant negative correlation between the theta wave and the profile of mood questionnaire ($p <= 0.05$) which indicates that the more the theta inhibition the lesser will be the anxiety/ depression mood state. There was also a significant positive correlation between the sensory pain and Sensori Motor Rhythm (SMR) after the 10th session ($p<=0.05$) which reveals that the more the SMR the lesser will be the sensory pain experienced.

Table 7: Correlations after intervention

	Post Alpha	Post Beta1A	Post Theta	Post SMR
Post POMS	.418	-.117	**-.665****	.225
Post STAI-A	-.196	.345	.151	.285
Post Trait A	-.058	.327	.017	.532
Post Pain A	-.157	-.188	.094	-.235
Post Pain S	-.022	.427	.025	**-.554****
Post BDI	-.203	.070	.166	.110
Post HAMD	.017	.155	-.038	.224
Side effect Q	.339	-.262	-.214	-.412

DISCUSSION

The present study revealed differences in mood states, state anxiety levels and sensory pain as a result of receiving alpha neurofeedback as compared to the sham treatment, thereby indicating the absence of placebo effects to a high extend. The altered negative mood states of the person also cause a significant reduction in the quality of life of the patients. Earlier studies have generally shown the benefits of mood enhancement in fMRI based neurofeedback (Johnston et al., 2010) but the present study shows efficacy of EEG based neurofeedback on mood states which might be beneficial in exploring further treatment options which can be accessible to everyone. Significant improvements in the state level anxieties of experimental group is in synchronization with evidence for effectiveness of neurofeedback on anxiety as a study in which patients underwent 10 sessions of

neurofeedback had significantly reduced state anxiety and altered cortical arousal (Costa et al., 2016).

The reductions in pain and significant differences found are supported by earlier studies which show that there is significant reduction in pain intensity in patients post treatment and even an enhancement in quality of life as it could work as a tool of self regulation through neurofeedback (Jacobs and Jensons., 2015). Further there is drop in depression levels but has not been found to be significant which is supported by studies which used the Deymed neurofeedback found that 15 sessions of 20 minutes each have shown to be efficacious in dropping the depression levels and anxiety in patients (Sahraee., 2016).

The alpha band power increased in the experimental group but it was not a significant difference after 10 sessions; Studies conducted involving eight sessions of neurofeedback have found similar results that the sessions were successful in enhancing the alpha power but the results have not shown a significant improvement (Escolano et al., 2014) which might further indicate that more sessions may be needed for significant training of the alpha bands.

The within experimental group comparison of the SMR frequencies indicates that frequency has increased or enhanced as compared between the 5^{th} session mean and the 10^{th} session means but there is no significant difference between the two baselines. Previous studies involving the use of ten neurofeedback sessions aiming at enhancing the sensori motor rhytm activity have found similar results that there was increase in the amplitudes post intervention but the results were not significant, although there was significant improvement in sleep latency in terms of shortened duration for the same (Hoedlmoser et al., 2008).

The study findings has shown a positive correlation between the mood states and alpha wave frequency, Beta wave frequency and sensori motor rhythms (SMR) at the baselines. With regard to the readings post assessment the correlation coefficient values have increased and have found that there is a significant negative correlation between the theta frequencies and the mood states.

With regard to the affective domain of the pain levels there was a negative correlation to the alpha and theta frequencies whereas a positive correlation was found with beta and Sensori motor rhytms but these were not significant at the baseline or post assessment.Studies have also found that higher EEG frequencies have a negative relationship with pain perception whereas a positive relationship exists with low amplitude EEG brain waves, although the relationships found were not conclusive as a direct causal relationship has not been found (Jenson et al., 2008).

CONCLUSIONS AND FUTURE DIRECTIONS

The study provides evidence for using the Alpha protocol of neurofeedback as an effective treatment for Tension type headache as it helps reducing the situational anxiety and sensory pain across the sessions helping the patients learn self regulation of their symptoms, further enhancing their mood states. However studies with greater number of sessions and follow up of these patients might further help know its long term efficacy and study other significant variables.

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