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Alpha and Beta Powers in EEG: How Audio-Visual Stimulation Influences Anxiety

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Abstract

This study aimed to reduce alpha and beta brainwave power through external audio-visual stimulation (AVS) and to evaluate its clinical effect on anxiety levels, as measured by the State-Trait Anxiety Inventory (STAI), alongside electroencephalography (EEG) data. Thirty participants received AVS, while 12 received audio-visual white noise as a control. EEG recordings were obtained before, during, and after the stimulation. A digital FFT-based power spectrum analysis was performed, and raw sum values (sum of spectral line values) within defined frequency ranges were extracted. The STAI was administered to assess both trait and state anxiety. Statistical analysis was conducted using SPSS. In the AVS group, significant reductions in alpha and beta power were observed between the pre-, during-, and post-stimulation phases ($p < 0.05$). Comparison of pre- and post-STAI scores revealed a statistically significant decrease in anxiety levels within the AVS group ($p < 0.001$), with no similar effect observed in the control group. Audio-visual stimulation significantly reduced alpha and beta EEG power during exposure and led to a marked decrease in self-reported anxiety. These findings provide both neurophysiological and clinical support for the use of AVS as a nonpharmacological method to alleviate anxiety symptoms.

Keywords Alpha wave · Beta wave · Power spectrum · Anxiety · Audio-visual stimulation

Introduction

Anxiety disorders rank among the most prevalent mental health conditions globally, affecting an estimated 275 million people each year (Vos et al., 2017). These disorders are typically characterized by excessive fear responses, heightened anxiety, and a strong tendency to avoid perceived threats (Penninx et al., 2021). Their etiology involves a complex interplay of genetic, environmental, and neurological factors. Fear is understood as a conscious emotional response to immediate or perceived threats, while anxiety relates to the anticipation of future dangers—real or imagined.

Anxiety is not merely a biological phenomenon; it is deeply entwined with mental and cognitive processes (Curtiss et al., 2021; Penninx et al., 2021). Dysfunctional thought patterns and maladaptive cognitive mechanisms, particularly those focused on future uncertainties, play a central role in both the development and persistence of anxiety symptoms. Consequently, modern approaches to treating anxiety disorders adopt a multidisciplinary perspective that accounts for both biological and psychological influences (Antos et al., 2024; Curtiss et al., 2021).

Pharmacological treatments aim to alleviate symptoms by addressing neurotransmitter imbalances. However, there has been growing interest in non-pharmacological interventions. Recent reviews have identified a range of effective complementary treatments for anxiety, including mindfulness-based techniques, physical activity, and immersive virtual reality experiences, highlighting their potential as valuable therapeutic tools (Antos et al., 2024).

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Brain waves are defined as oscillatory electrical voltages generated within the cerebral cortex, typically measuring only millionths of a volt. Five primary frequency bands of human electroencephalography (EEG) are clinically recognized. EEG signals arise from the superposition of these frequency components such as delta, theta, alpha, beta and gamma and vary across individuals due to factors such as age, gender, and health status (Nunez, 2011; Valdés-Hernández et al., 2010). These variations may manifest across different brain regions or within specific oscillatory components. To quantify and analyze frequency intensities in various regions, frequency-domain analysis using Fast Fourier Transform (FFT) is commonly applied (Zhang et al., 2023).

Historically, considerable research has focused on the impact of external stimulation on cortical EEG activity. Beta waves are linked to active mental states, external focus, emotional processing, and anxiety (Hendrayana et al., 2020; Huebl et al., 2016; Ribas et al., 2018). Alpha waves are generally related to relaxation and passive attention (Benwell et al., 2022; Cho et al., 2011; Knyazev et al., 2004).Reductions in alpha and beta power have been associated with improved cognitive functions, such as memory retrieval, language comprehension, and attentional control (Hanslmayr et al., 2012; León-Cabrera et al., 2022; Griffiths et al., 2019). Interest in rhythmic visual and auditory stimulation as a method to induce relaxation or hypnosis emerged in the mid-twentieth century. More recently, devices delivering light and sound at specific frequencies have been employed to 'entrain' or influence brainwave activity.

Audio-visual stimulation (AVS) has gained traction as an effective, non-pharmacological method for alleviating anxiety, promoting hypnagogic states, and supporting cognitive and behavioral improvements in specific populations (Teplan et al., 2011). This study aimed to investigate the effects of a standardized AVS protocol on anxiety reduction through both neurophysiological (EEG) and clinical (STAI) measures.

The AVS protocol utilized mandala videos with green hues for visual stimulation, chosen for their calming and emotionally positive associations (Lee, 2018; Wilms & Oberfeld, 2018). Binaural beats were selected for the auditory component, given their documented effectiveness in reducing anxiety through non-invasive means (Yusim & Grigaitis, 2020). It was hypothesized that this combined AVS approach would lead to significant reductions in alpha and beta EEG power, reflecting decreased anxiety, along with a measurable decline in STAI scores as clinical validation.

Materials and Methods

Study Design and Ethics

This prospective study was conducted in accordance with the 2009 Declaration of Helsinki. Ethical approval was granted by the Muğla Sıtkı Koçman University Medicine and Health Sciences Ethics Committee (Decision 7/XX, dated 23.03.2023).

Subjects

A total of 42 healthy volunteers (14 females, 28 males), aged between 18 and 60 years (mean age: 22 ± 1.73), with no history of medical conditions, medication use, or substance addiction, were recruited at the Department of Clinical Neurophysiology, Faculty of Medicine, Muğla Sıtkı Koçman University. EEG recordings were conducted in a temperature-controlled (25 °C), quiet, and dimly lit room in accordance with the International Federation of Clinical Neurophysiology guidelines (Babiloni et al., 2020). Participants were seated in a comfortable, half-reclined armchair throughout the session.

Exclusion criteria included pregnancy and physical or mental disabilities. Participants were randomly assigned to either the AVS (subject) group (n = 30; 10 females, 20 males) or the control group receiving audio-visual white noise (n = 12; 4 females, 8 males). Three participants were excluded due to technical issues during EEG recording. No adverse effects were reported, and all tests were successfully completed.

Psychometric Assessment

Anxiety levels were assessed using the State-Trait Anxiety Inventory (STAI), a validated tool for measuring both state and trait anxiety. The Turkish-validated version was used (Oner & Le Compte, 1985), administered before and after the AVS session. All assessments were performed by a trained clinical specialist.

Neurophysiological Assessment

EEG recordings followed the standards of the International Federation of Clinical Neurophysiology and the International League Against Epilepsy (Peltola et al., 2023). A Nicolet EEG device with 19-channel electrode placement conforming to the International 10–20 system was used. Data were recorded using an ear-reference montage at a sampling rate of 256 Hz, with filters set at 1 Hz (low-pass), 70 Hz (high-pass), and a 50 Hz notch filter. Each recording consisted of three 8-min sessions:

(1) Pre-stimulation (baseline)
(2) During AVS or white noise stimulation
(3) Post-stimulation

Data were exported in.edf format and analyzed using BrainVision Analyzer. Artifacts from eye movements were removed via independent component analysis (ICA), while muscle artifacts were eliminated manually. A digital FFT-based power spectrum analysis using a 10% Hanning window was conducted on each section. The raw sum values for specific EEG frequency bands were extracted: delta (0.1–4 Hz), theta (4–8 Hz), alpha (8–13 Hz), beta (13–30 Hz), gamma (30–48 Hz).

Audio-Visual Stimulation (AVS) Protocol

AVS was administered using a virtual reality headset.

Subject Group Protocol

- Auditory Stimulus: Binaural beats began at 14 Hz (114 Hz in the right ear, 100 Hz in the left ear) and decreased to 7.83 Hz over 4 min (97.83 Hz and 90 Hz respectively), then maintained at 7.83 Hz for the remaining 4 min. The stimulus was generated using Mistikist: AI Assisted Brainwave Regulator (Mistikist, 2023).
- Visual Stimulus: Kaleidoscopic mandala videos with green hues (~526–605 THz) were used, designed to feature continuous, non-repetitive motion. This component was also delivered using the Mistikist platform (Mistikist, 2023).

Control Group Protocol

- Visual White Noise: A looped 8-min grayscale video (1080 × 1920 resolution) was generated using FFmpeg, consisting of randomly assigned grayscale pixel patterns (Tomar, 2006). The red channel intensity varied (0–255), while green and blue channels were fixed at 128.
- Auditory White Noise: White noise audio was encoded in PCM S16 LE (WAV) format, with a 44.1 kHz sample rate, 16-bit resolution, and amplitude of 0.5, creating an 8-min stimulus with uniform average power across all frequencies (Söderlund GBW, 2021).

Statistical Analysis

Statistical analysis was performed using IBM SPSS Statistics for Windows, Version 27.0 and Statistica Version 12. Data normality was assessed using the Shapiro–Wilk test.

- For normally distributed continuous variables, independent samples t-tests were used; otherwise, the Mann–Whitney U test was applied.
- Pre-, during-, and post-stimulation values were compared using repeated measures ANOVA (for parametric data) or the Friedman test (for non-parametric data).
- Pre- and post-STAI scores were analyzed using paired samples t-tests or Wilcoxon signed-rank tests, depending on distribution.

Descriptive statistics are presented as mean ± standard deviation or median (25th-75th percentiles). A $p\text{-value} < 0.05$ was considered statistically significant. A larger subject group was used to account for potential variability in response to AVS and to improve variance estimation. Between-group comparisons employed non-parametric tests to address unequal sample sizes and minimize bias.

Results

Table 1 presents the descriptive statistics for pre-stimulation alpha wave raw sum values in both control and subject groups. No statistically significant difference was observed between groups at baseline ($p > 0.05$).

In Table 2, the descriptive statistics for control and subject groups in relation to pre-area raw sum measurements of beta waves are presented. A statistically significant difference was not found between the control and subject groups concerning pre-area raw sum measurements of beta waves ($p > 0.05$).

Table 3 presents the descriptive statistics and comparative analysis of alpha wave raw sum measurements for the control group across pre-, during-, and post-stimulation phases. Overall, no statistically significant differences were observed across the three time points ($p > 0.05$), with the exception of measurements at electrode sites O2 and T6.

At site O2, pairwise comparisons indicated that the pre-stimulation alpha wave raw sum was significantly higher than during-stimulation values ($p < 0.05$). At site T6, the pre-stimulation measurement was significantly greater than the during-stimulation value ($p < 0.01$) and significantly lower than the post-stimulation value ($p < 0.05$). These site-specific changes suggest minor localized fluctuations in alpha activity during the control condition, but no consistent or widespread pattern of change was evident across the control group.

Table 4 presents the descriptive statistics and comparative results for beta wave raw sum measurements in the control group across the pre-, during-, and post-stimulation phases. No statistically significant differences were found across

Table 1 Descriptive statistics of control and subject groups in terms of pre area raw sum measurements by alpha wave

PRE measurement	Group		Test statistic	p
	Control (n = 12)	Subject (n = 27)		
F3	1.15 (0.61–1.93)	1 (0.71–1.66)	− 0.213	0.845
F4	2.18 (0.68–4.60)	1.12 (0.61–2.23)	− 1.674	0.098
C3	1.49 (0.57–4.24)	2.23 (1.25–7.27)	− 1.248	0.221
C4	1.62 (0.67–5.59)	2.63 (0.83–8.51)	− 0.761	0.461
P3	2.86 (0.96–15.18)	5.33 (2.91–17.05)	− 1.339	0.188
P4	2.30 (1.24–12.05)	6.27 (2.58–18.61)	− 1.308	0.199
O1	3.61 (2.33–16.64)	10.45 (4.22–18.28)	− 1.491	0.142
O2	4 (2–16.96)	9.58 (4.31–23.96)	− 1.795	0.075
F7	0.67 (0.50–2.65)	2.06 (0.99–3.35)	− 1.795	0.075
F8	1.05 (0.52–2.10)	1.58 (0.80–3.55)	− 0.669	0.518
T3	1.67 (1.08–8.71)	4.08 (2.16–7.98)	− 1.248	0.221
T4	2.37 (0.94–7.30)	3.17 (1.87–9.86)	− 0.822	0.425
T5	2.77 (2.07–17.41)	7.94 (4.33–19.20)	− 1.552	0.126
T6	3 (1.72–16.11)	12.43 (3.35–20.72)	− 1.674	0.098
FZ	0.64 (0.37–1.84)	0.99 (0.45–1.35)	− 0.061	0.964
CZ	1.23 (0.64–2.34)	2.03 (0.79–4.53)	− 0.822	0.425
PZ	2.27 (0.98–10.49)	4.71 (2.44–15.71)	− 1.156	0.258

Data are presented as median (25th–75th percentiles). Test statistic refers to the Mann Whitney U test (Z), which was used due to non-normal distribution of variables. Significant results are shown in bold ($p < 0.05$).

Table 2 Descriptive statistics of control and subject groups in terms of pre-area raw sum measurements by beta wave

PRE measurement	Group		Test statistic	p
	Control (n = 12)	Subject (n = 27)		
F3	2.49 (1.07–8.29)	3.41 (1.75–6.04)	− 0.365	0.730
F4	4.40 (1–7.94)	2.08 (1.34–4.13)	− 0.669	0.518
C3	2.55 (1.39–5.11)	2.41 (1.83–4.44)	− 0.548	0.599
C4	1.98 (1.47–4.37)	2.92 (1.51–5.16)	− 0.548	0.599
P3	3.20 (1.65–5.47)	5.44 (2.45–6.83)	− 1.339	0.188
P4	2.57 (2.32–5.96)	4.09 (2.67–6.68)	− 1.217	0.233
O1	4.77 (3.07–7.84)	6.46 (3.58–10.77)	− 1.278	0.210
O2	4.95 (2.89–8.60)	5.86 (3.86–12.16)	− 0.974	0.343
F7	1.85 (0.97–2.53)	2.94 (1.13–4.39)	− 1.674	0.098
F8	2.11 (1.18–4.55)	1.86 (1.27–3.96)	− 0.061	0.964
T3	5.54 (2.54–10.69)	5.26 (3.51–10.05)	− 0.213	0.845
T4	3.48 (2.49–10.23)	4.79 (2.67–10.12)	− 0.609	0.558
T5	5.98 (3.29–9.73)	7.32 (3.97–12.06)	− 0.730	0.480
T6	4.86 (2.08–12.89)	6.95 (4.08–10.47)	− 1.035	0.313
FZ	1.13 (0.59–1.76)	1.17 (0.60–1.80)	− 0.274	0.799
CZ	1.67 (0.92–3.74)	1.94 (1.36–3.96)	− 0.761	0.461
PZ	2.40 (1.31–6.85)	3.64 (1.94–6.15)	− 0.852	0.408

Data are presented as median (25th–75th percentiles). Test statistic refers to the Mann Whitney U test (Z), which was used due to non-normal distribution of variables. Significant results are shown in bold ($p < 0.05$).

time points in the majority of electrode sites ($p > 0.05$), with the exception of site O2.

At O2, a significant increase in beta wave activity was observed: the pre-stimulation raw sum measurement was significantly lower than both the during- and post-stimulation values ($p < 0.05$ for both comparisons). However, there was no significant difference between the during- and post-stimulation measurements ($p > 0.05$).

These findings indicate that while a localized increase in beta activity occurred at O2, the control group showed no consistent or widespread changes in beta wave power across the brain.

Table 5 presents the descriptive statistics and comparative results for alpha wave raw sum measurements in the subject group across the pre-, during-, and post-stimulation phases. Statistically significant differences were observed at multiple electrode sites, including F3, C3, P3, P4, O1, O2, F7, T3, T4, T5, T6, and PZ ($p < 0.05$; see Fig. 1).

Multiple comparison analyses revealed the following:

1. For C3, P3, P4, O1, O2, F7, T3, T4, T5, T6, and PZ, the during-stimulation measurements were significantly lower than both pre- and post-stimulation values ($p < 0.05$). No significant differences were observed between pre- and post-stimulation values at these sites ($p > 0.05$), indicating a temporary reduction in alpha power limited to the stimulation phase.
2. At F3, the during-stimulation measurement was significantly lower than the pre-stimulation value ($p < 0.05$).

Table 3 Descriptive statistics of the control group in terms of pre-during-post area raw sum measurements by alpha wave and comparison results

Measurement	Pre	During	Post	Test statistic	P
F3	1 (0.64–1.87)	0.73 (0.53–1.83)	1.17 (0.66–2.05)	1.167	0.558
F4	1.16 (0.61–2.68)	0.85 (0.51–2.02)	1.16 (0.58–2.34)	0.167	0.920
C3	2.09 (0.93–4.57)	1.92 (1.09–3.62)	2.26 (1.14–4.80)	4.500	0.105
C4	2.04 (0.83–8.36)	1.95 (0.99–4.59)	2.31 (0.99–5.83)	1.167	0.558
P3	5.10 (1.71–16.60)	5.06 (1.48–9.37)	5.13 (2.66–12.37)	0.500	0.779
P4	4.31 (2.02–14.74)	3.88 (1.94–7.83)	6.02 (2.14–14.97)	3.500	0.174
O1	9.05 (3.15–18.06)	6.28 (3.38–11.59)	8.59 (3.83–20.18)	4.167	0.125
O2	9.34 (3.83–21.84)^{a}	6.45 (3.66–12.32)^{b}	8.84 (4.57–24.50)$^{a.b}$	8.167	**0.017**
F7	1.64 (0.64–3.23)	1.03 (0.50–2.45)	1.63 (0.96–3.08)	5.167	0.076
F8	1.48 (0.74–3.43)	1.38 (0.41–2.65)	1.78 (0.80–3.70)	2.167	0.338
T3	3.24 (1.48–7.98)	2.46 (1.24–5.23)	3.20 (1.57–8.16)	4.500	0.105
T4	2.99 (1.44–7.35)	2.37 (1.56–5.41)	2.93 (1.64–7.41)	1.167	0.558
T5	6.40 (2.48–17.69)	4.58 (3.06–11.56)	8.13 (3.63–17.94)	5.167	0.076
T6	7.10 (2.65–17.99)^{a}	5.17 (3.20–11.16)^{b}	7.94 (3.34–21.76)^{b}	13.167	**0.001**
FZ	0.87 (0.45–1.57)	0.55 (0.44–1.28)	0.79 (0.52–1.78)	0.000	>0.999
CZ	1.83 (0.70–3.67)	1.97 (0.93–3.45)	2.01 (1.05–4.60)	2.667	0.264
PZ	4.59 (1.86–10.72)	3.19 (1.75–8.01)	5.15 (2–12.17)	0.667	0.717

Data are presented as median (25th–75th percentiles). Test statistic refers to the Friedman test (χ^2), which was used due to non-normal distribution of variables. Significant results are shown in bold ($p<0.05$). Upper letters that are similar represent the similarity between groups, that are different represent significant differences between groups.

Table 4 Descriptive statistics of the control group in terms of pre-during-post area raw sum measurements by beta wave and comparison results

Measurement	Pre	During	Post	Test statistic	P
F3	2.49 (1.07–8.29)	1.26 (0.83–3.42)	3.34 (2.68–4.67)	4.500	0.105
F4	4.40 (1–7.94)	1.83 (1.13–3.07)	2.76 (1.41–5.03)	6.667	0.717
C3	2.54 (1.39–5.11)	2.46 (1.58–3.83)	2.57 (1.23–5.03)	0.167	0.920
C4	1.98 (1.47–4.37)	2.90 (1.16–4.81)	2.30 (1.37–3.34)	0.500	0.779
P3	3.20 (1.65–5.47)	3.75 (1.82–5.67)	2.84 (2.35–4.84)	0.167	0.920
P4	2.57 (2.32–5.96)	3.69 (1.84–5.92)	3.08 (2.48–5.89)	0.920	0.920
O1	4.77 (3.07–7.84)	6.03 (2.87–11.73)	4.92 (3.67–5.88)	3.500	0.174
O2	4.95 (2.89–8.60)^{a}	5.23 (3.41–11.13)^{b}	5.51 (4.17–7.48)^{b}	6.167	**0.046**
F7	1.85 (0.97–2.53)	2.22 (0.89–4.39)	3.25 (1.53–4.87)	2.667	0.264
F8	2.11 (1.18–4.55)	2.10 (0.91–2.76)	3 (2.11–3.35)	1.167	0.558
T3	5.54 (2.54–10.69)	3.73 (2.79–8.05)	4.62 (2.61–10.80)	0.500	0.779
T4	5.63 ± 4.37	5.86 ± 4.42	4.95 ± 2.93	0.250	0.781
T5	5.98 (3.29–9.73)	6.14 (3.98–10.69)	5.72 (2.85–9.82)	0.667	0.717
T6	4.86 (2.08–12.89)	9.64 (3.99–15.34)	6.14 (3.43–10.69)	5.167	0.076
FZ	1.13 (0.59–1.76)	0.95 (0.66–1.70)	1.20 (0.88–1.69)	0.167	0.920
CZ	1.67 (0.92–3.74)	1.97 (1.28–4.27)	1.78 (1.31–3.07)	1.500	0.472
PZ	2.40 (1.31–6.85)	2.42 (1.37–6.86)	2.38 (1.78–3.98)	0.667	0.717

Data are presented as median (25th–75th percentiles), except for T4 (mean ± SD). Friedman test was used for non-parametric variables (test statistic = χ^2); repeated measures ANOVA was applied for normally distributed variables (test statistic = F). Significant results are in bold ($p<0.05$). Upper letters that are similar represent the similarity between groups, that are different represent significant differences between groups.

Table 5 Descriptive statistics of the subject group in terms of pre-during-post area raw sum measurements by alpha wave and comparison results

Measurement	Pre	During	Post	Test statistic	p
F3	1 (0.71–1.66)^{a}	0.71 (0.53–1.56)^{b}	1.05 (0.6–1.90)$^{a.b}$	7.630	**0.022**
F4	1.12 (0.61–2.23)	0.79 (0.45–1.44)	1.07 (0.51–1.97)	2.296	0.317
C3	2.28 (1.25–7.27)^{a}	2.14 (1.09–3.72)^{b}	2.51 (1.36–4.91)^{a}	8.296	**0.016**
C4	2.63 (0.83–8.51)	1.87 (0.99–4.59)	3.12 (1–5.93)	3.556	0.169
P3	5.33 (2.91–17.05)^{a}	4.65 (1.65–9.48)^{b}	7.83 (3.36–14.10)^{a}	11.556	**0.003**
P4	6.27 (2.58–18.61)^{a}	3.88 (2.21–7.83)^{b}	8.30 (2.38–15.89)^{a}	9.852	**0.007**
O1	10.45 (4.22–18.28)^{a}	5.66 (3.38–11.52)^{b}	12.28 (4.24–26.17)^{a}	9.556	**0.008**
O2	9.58 (4.31–23.96)^{a}	6.38 (3.66–10.71)^{b}	10.20 (5.29–27.34)^{a}	8.074	**0.018**
F7	2.06 (0.99–3.35)^{a}	1.02 (0.43–2.37)^{b}	1.68 (1.05–3.08)^{a}	8.000	**0.018**
F8	1.58 (0.80–3.55)	0.82 (0.38–1.86)	1.74 (0.70–3.70)	5.852	0.054
T3	4.08 (2.16–7.98)^{a}	2.46 (1.16–4.64)^{b}	3.22 (1.76–8.61)^{a}	9.556	**0.008**
T4	3.17 (1.87–9.85)^{a}	2.33 (1.11–5.18)^{b}	4 (2.21–8.62)^{a}	24.889	**<0.001**
T5	7.94 (4.33–19.20)^{a}	4.26 (3.06–11.37)^{b}	8.31 (3.88–21.02)^{a}	8.000	**0.018**
T6	14.43 (3.35–20.72)^{a}	5.06 (2.95–10.29)^{b}	11.63 (3.34–23.69)^{a}	11.185	**0.004**
FZ	0.99 (0.45–1.35)	0.54 (0.40–0.91)	0.77 (0.37–1.78)	1.407	0.495
CZ	2.03 (0.79–4.53)	1.50 (0.92–3.18)	2.28 (1.12–4.60)	1.407	0.495
PZ	4.71 (2.44–15.71)^{a}	3.18 (2.45–8.01)^{b}	7.77 (2.94–12.94)^{a}	8.074	**0.018**

Data are presented as median (25th–75th percentiles). Test statistic refers to the Friedman test (χ^2), which was used due to non-normal distribution of variables. Significant results are shown in bold ($p<0.05$). Upper letters that are similar represent the similarity between groups, that are different represent significant differences between groups.

However, no significant differences were found between pre- and post-, or during- and post-stimulation values ($p>0.05$).

These results suggest that AVS led to a transient but widespread decrease in alpha wave activity across multiple brain regions during stimulation, with activity levels returning to baseline post-stimulation.

Table 6 presents the descriptive statistics and comparative analyses of beta wave raw sum measurements for the subject group across the pre-, during-, and post-stimulation phases. Significant differences were observed at multiple electrode sites, including F3, F4, P3, P4, O2, F7, F8, T3, T4, T5, T6, and PZ ($p<0.05$; see Fig. 2).

Multiple comparison results revealed the following:

1 At F3, P4, O2, F7, F8, T3, T5, T6, and PZ, during-stimulation beta wave measurements were significantly lower than both pre- and post-stimulation values ($p<0.05$). No significant differences were found between pre- and post-stimulation measurements at these sites ($p>0.05$), indicating a temporary suppression of beta activity during AVS.

2 At F4, P3, and T4, during-stimulation measurements were significantly lower than pre-stimulation values ($p<0.05$, $p<0.01$, and $p<0.05$, respectively). However, no significant differences were found between pre- and post- or during- and post-stimulation measurements ($p>0.05$), suggesting a similar transient reduction effect.

These findings demonstrate that AVS induced a temporary but widespread reduction in beta wave activity across multiple brain regions, consistent with a reduction in cognitive arousal or anxiety during the stimulation period.

Table 7 presents a comparative analysis of State-Trait Anxiety Inventory (STAI) scores recorded before and after the intervention for both the subject and control groups. A statistically significant reduction in anxiety was observed in the subject group, with post-intervention STAI scores significantly lower than pre-intervention scores ($p<0.001$; see Fig. 3). In contrast, the control group exhibited no significant change between pre- and post-intervention STAI scores ($p>0.05$; see Fig. 4).

Additionally, gender distribution across both groups was statistically homogeneous, with no significant difference detected ($p=0.999$), indicating that gender did not confound the observed outcomes.

Data are presented as median (25th–75th percentiles) or mean ± standard deviation, depending on distribution. Test statistic refers to Wilcoxon signed-rank test (Z) for non-normally distributed data and paired-samples t-test (t) for

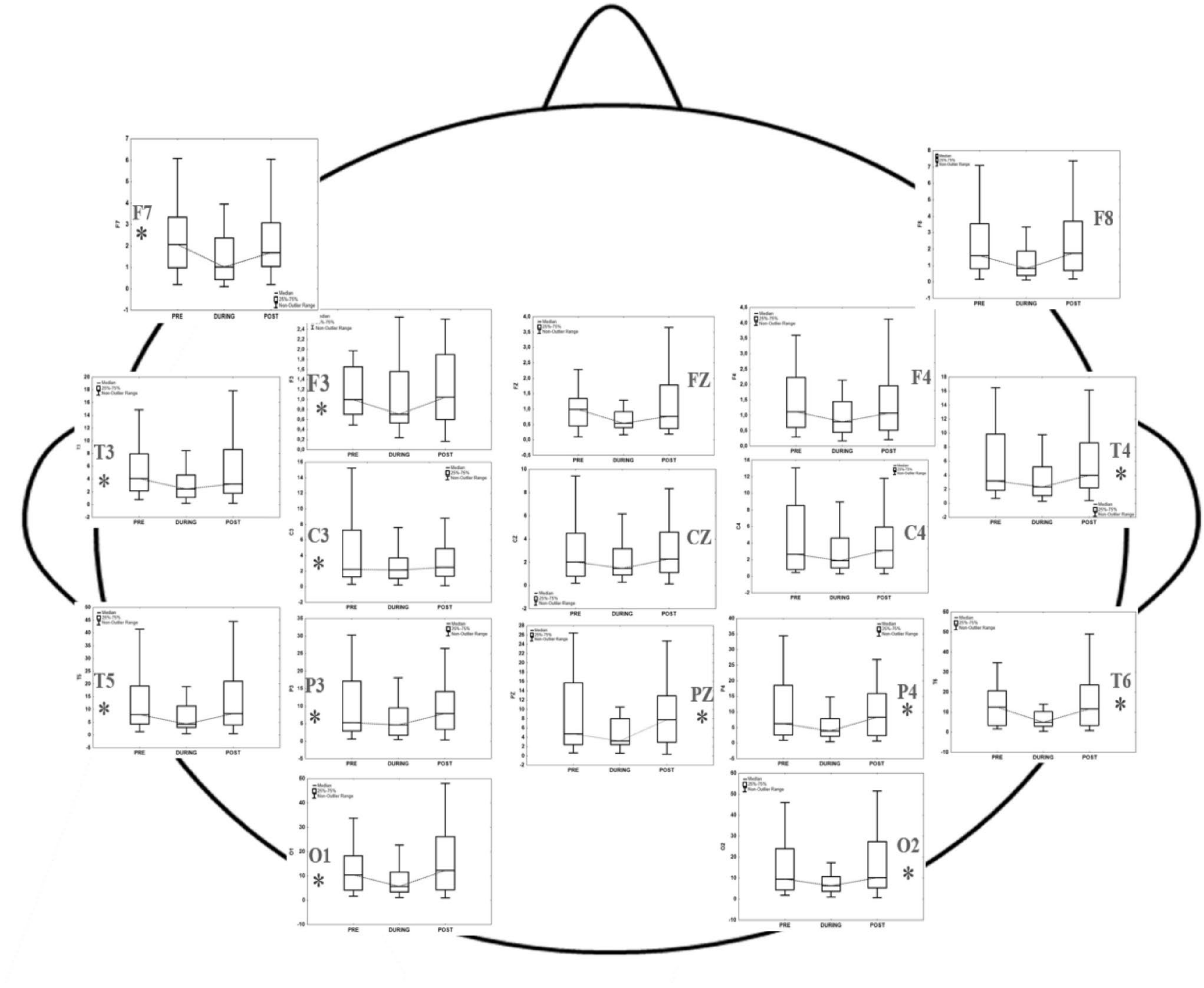

Fig. 1 Box plots for the subject group for the raw sum values before, during and after the alpha wave with statistically significant differences for F3, C3, P3, P4, O1, O2, F7, T3, T4, T5, T6, PZ and statistically no differences for CZ, C4, FZ, F4, F8 (*: statistically significant differences)

normally distributed data. Significant results are shown in bold ($p < 0.05$).

This study examined the neurophysiological and clinical effects of audio-visual stimulation (AVS)—specifically, binaural beats and kaleidoscopic mandala videos featuring green hues—on anxiety, as compared to a white noise control. EEG patterns and State-Trait Anxiety Inventory (STAI) scores were analyzed across pre-, during-, and post-stimulation phases for both subject and control groups.

Neurophysiological Findings

At baseline, no significant differences in alpha or beta wave power were found between groups. The control group exhibited consistent EEG activity across all phases, indicating minimal effect from white noise stimulation. In contrast, the subject group showed a significant reduction in both alpha and beta power during AVS, with values returning toward baseline post-stimulation. This transient decrease suggests a phase-specific neurophysiological response to the AVS intervention.

These results are consistent with prior literature indicating that reductions in alpha and beta power reflect changes in cortical activation, potentially linked to shifts in attention, cognitive load, and emotional state (Griffiths et al., 2019; Van Ede et al., 2011).

Clinical Outcomes

Clinically, STAI scores in the subject group decreased significantly following AVS, whereas the control group showed no change. This suggests a meaningful reduction in subjective anxiety levels attributable to the AVS intervention. The

Table 6 Descriptive statistics of the subject group in terms of pre-during-post area raw sum measurements by beta wave and comparison results

Measurement	Pre	During	Post	Test statistic	p
F3	3.41 (1.75–6.04)^{a}	1.50 (1.30–2.29)^{b}	3.02 (1.46–5.64)^{a}	10.963	**0.004**
F4	2.08 (1.34–4.13)^{a}	1.17 (0.78–3)^{b}	2.04 (0.91–4.41)^{ab}	6.000	**0.049**
C3	2.41 (1.83–4.44)	2.17 (1.44–3.03)	2.61 (1.57–4.32)	4.963	0.084
C4	2.92 (1.51–5.16)	2.27 (1.53–3.03)	2.31 (1.44–4.69)	3.852	0.146
P3	5.44 (2.45–6.83)^{a}	3.40 (2.02–5.86)^{b}	4.59 (2.46–6.70)^{ab}	9.185	**0.010**
P4	4.09 (2.67–4.79)^{a}	2.95 (1.94–4.79)^{b}	3.73 (2.56–7.52)^{a}	10.963	**0.004**
O1	6.46 (3.58–10.77)	5.68 (2.94–7.74)	5.94 (3.88–11.38)	5.852	0.054
O2	5.86 (3.86–12.16)^{a}	5.16 (3.06–8)^{b}	6.33 (3.74–10.39)^{a}	6.889	**0.032**
F7	2.94 (1.13–4.39)^{a}	1.37 (0.68–2.34)^{b}	2.34 (1.29–3.72)^{a}	6.889	**0.032**
F8	1.86 (1.27–3.96)^{a}	1.34 (0.70–2.95)^{b}	2.43 (1.39–3.65)^{a}	10.889	**0.004**
T3	5.26 (3.51–10.05)^{a}	3.11 (1.86–6.86)^{b}	5.40 (2.90–9.35)^{a}	14.296	**$<$0.001**
T4	4.79 (2.67–10.12)^{a}	4.08 (2.02–7.31)^{b}	4.53 (3.31–8.11)^{ab}	6.000	**0.049**
T5	7.32 (3.97–12.06)^{a}	4.44 (3.44–7.61)^{b}	6.54 (4.15–11.14)^{a}	9.556	**0.008**
T6	6.95 (4.08–10.47)^{a}	5.57 (3.54–7.21)^{b}	6.15 (4.46–11.09)^{a}	6.741	**0.034**
FZ	1.17 (0.60–1.80)	0.79 (0.59–1.25)	0.88 (0.52–1.61)	4.222	0.121
CZ	1.94 (1.36–3.96)	1.88 (1.07–2.40)	1.82 (1.29–3.38)	0.296	0.862
PZ	3.64 (1.94–6.15)^{a}	2.87 (1.76–4.07)^{b}	3.42 (2.10–6.03)^{a}	10.963	**0.004**

Data are presented as median (25th–75th percentiles). Test statistic refers to the Friedman test (χ^2), which was used due to non-normal distribution of variables. Significant results are shown in bold ($p<0.05$). Upper letters that are similar represent the similarity between groups, that are different represent significant differences between groups.

homogeneous gender distribution ($p=0.999$) rules out gender-related confounding in these outcomes.

Theoretical and Therapeutic Context

Anxiety is a multifaceted mental health condition influenced by genetic, environmental, and neurobiological factors. In addition to biological mechanisms, anxiety is closely linked with maladaptive cognitive processes such as inflexible beliefs and anticipatory worry (Butler & Mathews, 1983; Charpentier et al., 2017). Effective treatment, therefore, often requires addressing both biological and psychological domains (Curtiss et al., 2021).

While pharmacological approaches aim to normalize neurotransmitter function, increasing attention is being directed toward non-pharmacological alternatives—including mindfulness, physical activity, virtual reality, and neuromodulation (Antos et al., 2024). Among these, binaural beats have gained traction for their anxiolytic potential. A review of twelve studies identified binaural beats as more effective than both music and noise-canceling headphones for reducing anxiety (Baseanu et al., 2024). Further, Yusim and Grigaitis (2020) found enhanced STAI score reductions when binaural rhythms were combined with psychotherapy.

Alpha and Beta Waves in Anxiety Research

Although EEG is not traditionally used for mood assessment, multiple studies have identified associations between alpha and beta rhythms and anxiety states. Increased alpha activity has been linked to reduced anxiety in some studies (e.g., after biofeedback or tACS interventions), while others have reported heightened alpha in anxious individuals, particularly in frontal and parietal regions (Bhat, 2010; Clancy et al., 2018; Cho et al., 2011; Knyazev et al., 2004, Shehani et al., 2024). These conflicting findings suggest that the relationship between alpha activity and anxiety may depend on individual neurophysiology, anxiety subtype, and cortical region.

Beta waves (13–30 Hz), associated with cognitive effort and arousal, have also been implicated in anxiety. Elevated beta activity has been observed in individuals with anxiety disorders and is considered a potential biomarker (Huebl et al., 2016; Ribas et al., 2018). Several studies have demonstrated that effective anxiety interventions—such as virtual reality and EEG neurofeedback—can modulate beta activity, often showing a shift from high to low beta power corresponding with reduced anxiety (Díaz et al., 2019; Tarrant et al., 2018).

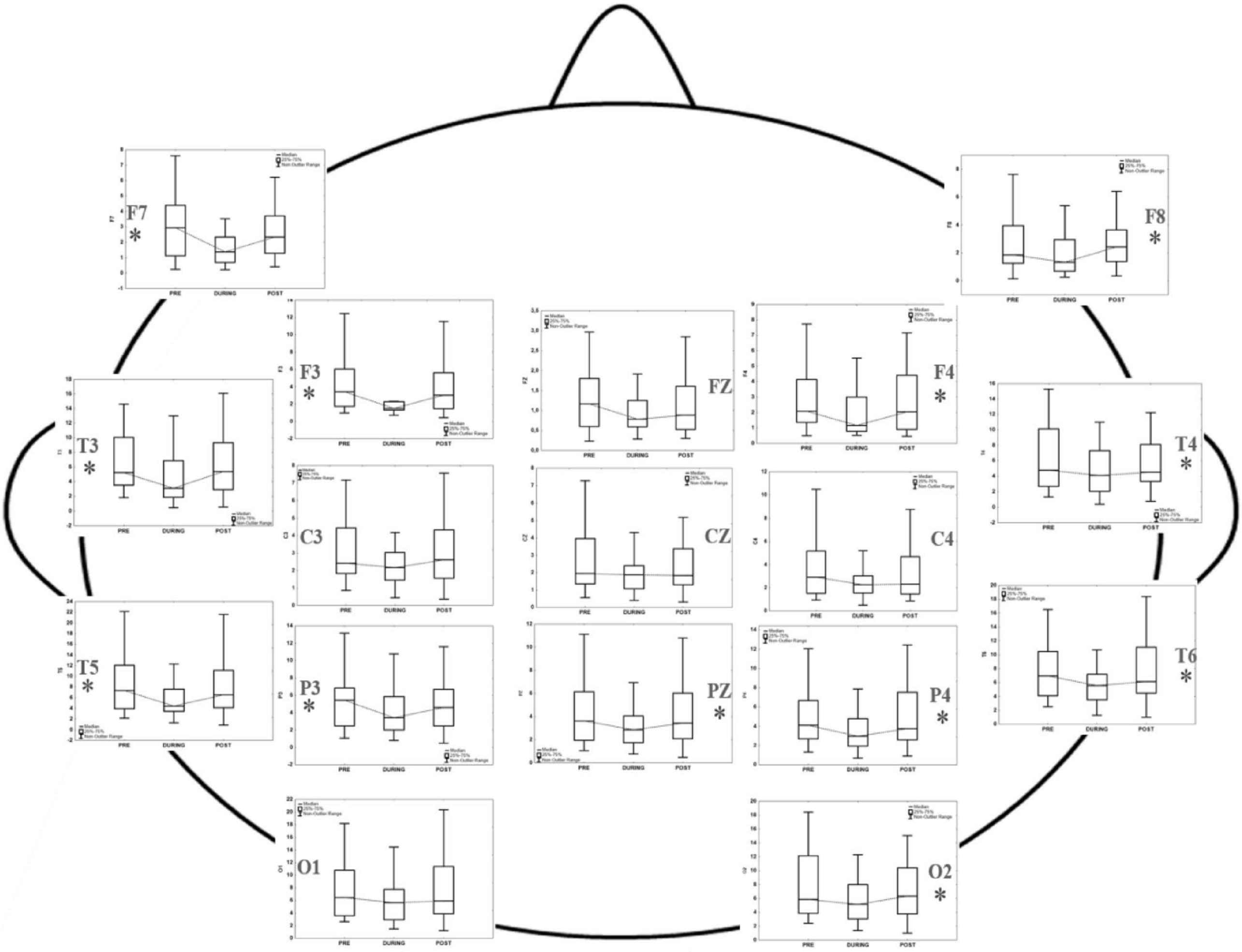

Fig. 2 Box plots for the subject group for the raw sum values before, during and after the beta wave with statistically significant differences for F3, F4, P3, P4, O2, F7, F8, T3, T4, T5, T6, PZ and statistically no differences for C3,C4, O1, FZ, CZ, (*: statistically significant differences)

Table 7 Comparison STAI results of pre-post measurements for subject and control groups

	Pre	Post	Test statistic	p
Subject Score	51 (39–65)	26 (23–33)	− 4.542	**< 0.001**
Control score	37.17 ± 11.77	42.67 ± 15.62	− 1.837	0.093

Mechanistic Insights

In this study, significant reductions in both alpha and beta power during AVS were paralleled by reductions in STAI scores, providing both neurophysiological and clinical evidence of anxiety alleviation. While the exact mechanism remains unclear, one hypothesis is that decreased alpha and beta power reflects a disruption of synchronized neuronal firing. Such desynchronization is thought to improve information processing by unmasking stimulus-specific neural activity (Griffiths et al., 2019; Pfurtscheller & Lopes Da Silva, 1999).

Supporting this, Van Ede et al. (2011) found that attentional engagement with upcoming stimuli led to decreased alpha and beta power in sensorimotor areas, attributed to a breakdown in neural synchrony. Similarly, during visual and auditory processing tasks, power reductions in both bands were linked to enhanced cognitive throughput and attentional modulation (Griffiths et al., 2019).

These observations suggest that the EEG power decreases observed during AVS may reflect an active neurocognitive reconfiguration, where reduced synchronization enables more flexible and adaptive neural responses—possibly reducing maladaptive patterns associated with anxiety.

Limitations and Future Perspectives

This study was conducted with a relatively small cohort of 39 participants. While the results were statistically significant, a larger sample size would enhance the generalizability and robustness of the findings. Additionally, this

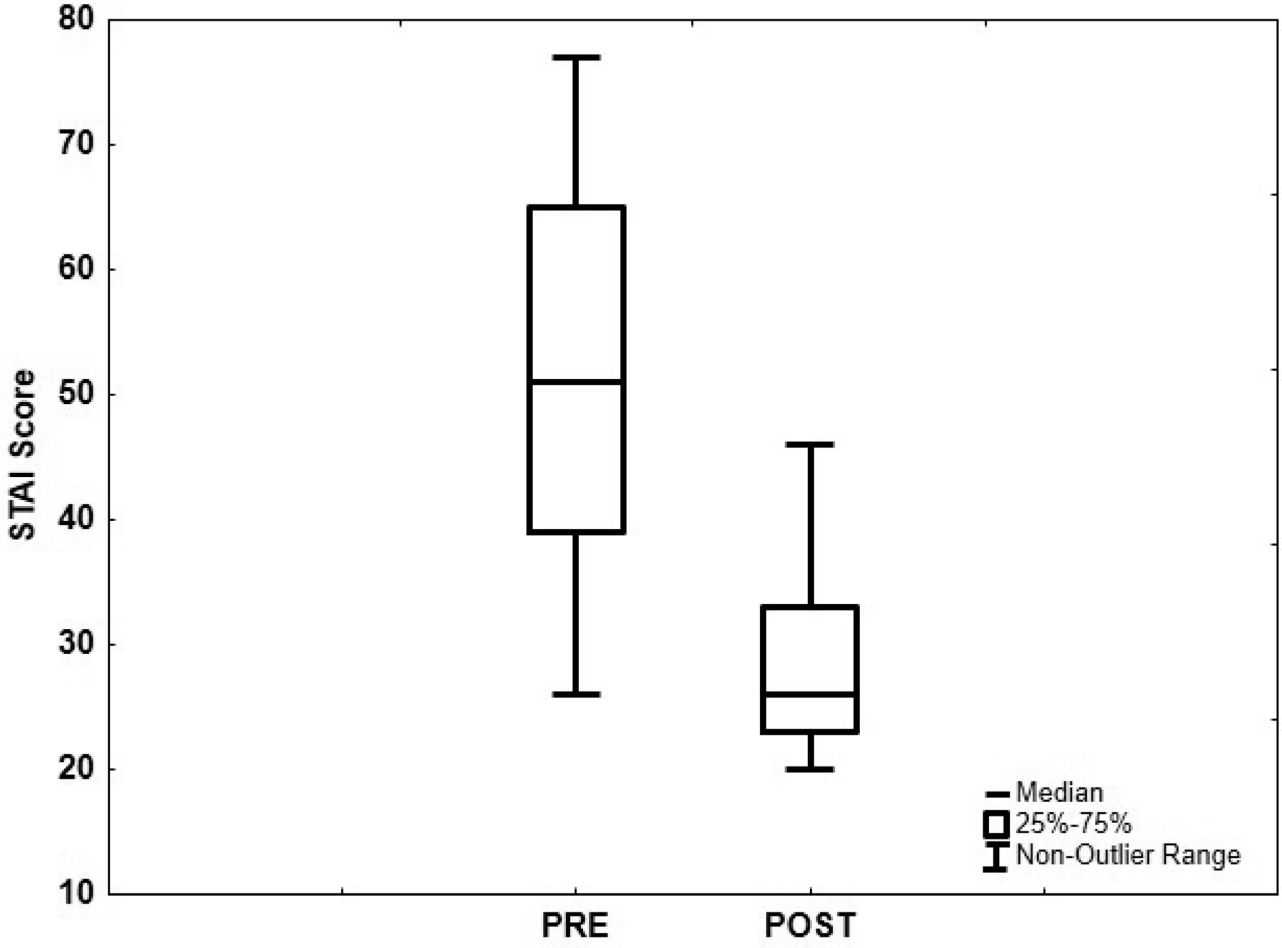

Fig. 3 Box plot of pre and post-scores of the subject group

study is the gender imbalance, with more male participants included due to inclusion–exclusion criteria. Although similar female-to-male ratios were maintained across groups, the overall distribution remained uneven. However, the repeated-measures design minimized the potential influence of gender on the results. Increasing the sample size in future studies may enable a more comprehensive examination of potential gender differences.

The study focused solely on the immediate effects of audio-visual stimulation (AVS) on anxiety levels. Future research should explore the long-term efficacy of AVS interventions and assess whether the observed benefits are sustained over time.

The AVS protocol used in this study was based on a fixed combination of binaural beats and green kaleidoscopic mandala visuals. However, the effects of varying binaural beat frequencies and visual stimuli (e.g., color, pattern, intensity) were not investigated. Future studies could employ a comparative design to identify the most effective combinations of auditory and visual stimuli for anxiety reduction. This would allow for the optimization of AVS parameters tailored to individual needs or specific anxiety profiles.

Conclusions

This study investigated the clinical and neurophysiological effects of audio-visual stimulation, using binaural beats and green mandala imagery, on anxiety. Participants exposed to this AVS protocol demonstrated significant reductions in both alpha and beta EEG power during the stimulation phase, which returned toward baseline post-stimulation. In parallel, STAI scores in the subject group decreased significantly following the intervention, while no significant changes were observed in the control group exposed to white noise.

These findings provide converging neurophysiological and clinical evidence that AVS can serve as an effective, non-pharmacological intervention for the alleviation of anxiety symptoms. The likely mechanism underlying these effects involves a disruption of synchronized neuronal firing, as reflected in the transient reduction of alpha and beta

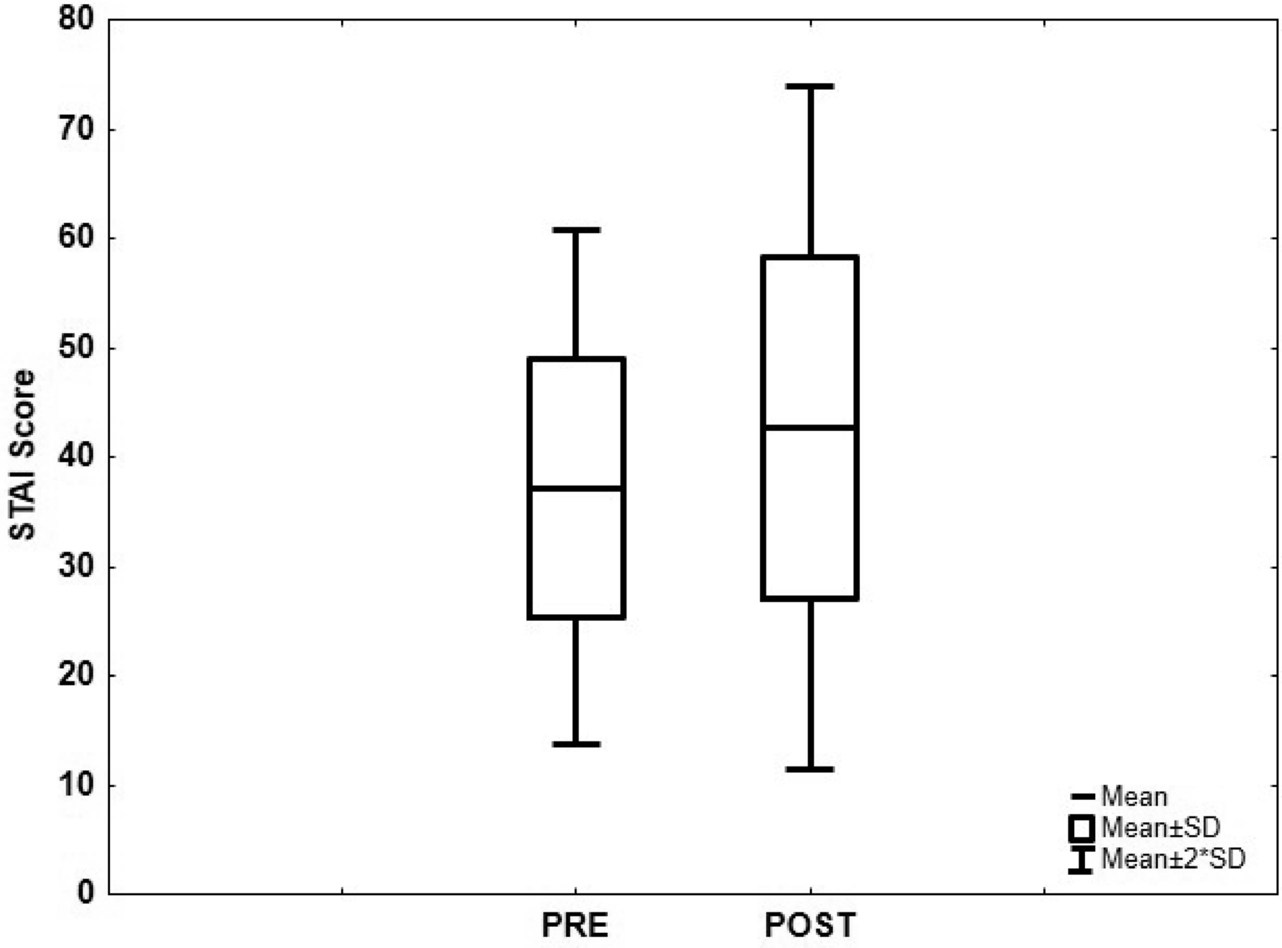

Fig. 4 Box plot of pre and post-scores of the control group

power during AVS exposure. This desynchronization may enhance cognitive flexibility and reduce maladaptive neural activity associated with anxiety.

Further research is warranted to optimize AVS protocols and to establish their long-term therapeutic potential in both clinical and non-clinical populations.

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Author's Contribution İnan ÖZDEMİR: Conceptualization, Methodology, Software, Validation, Formal analysis, Investigation, Resources, Data Curation, Writing—Original Draft, Writing—Review & Editing, Visualization Emine Elif TÜLAY: Conceptualization, Methodology, Software, Validation, Formal analysis, Investigation, Resources, Data Curation, Writing—Review & Editing Serkan AKSU: Methodology, Software, Validation, Formal analysis, Investigation, Data Curation, Writing—Original Draft, Writing—Review & Editing Fulden Candaş TÜRKİŞ: Methodology, Software, Validation, Formal analysis, Data Curation, Writing—Review & Editing Çağla Abalı ÇELEBİ: Methodology, Software, Validation, Writing—Review & Editing Semai BEK: Conceptualization, Methodology, Software, Validation, Formal analysis, Investigation, Resources, Writing—Original Draft, Writing—Review & Editing, Visualization, Supervision Gülnihal KUTLU: Conceptualization, Methodology, Software, Validation, Formal analysis, Investigation, Resources, Writing—Original Draft, Writing—Review & Editing, Supervision All authors have contributed to writing and/or editing of this report and have read and approved the final manuscript.

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Data Availability No datasets were generated or analysed during the current study.

Declarations

Conflict of interest The authors declare no competing interests.

Ethics Approval Our study was conducted prospectively and in accordance with the 2009 Declaration of Helsinki. Ethical approval of the research was received with Muğla Sıtkı Koçman University Medicine and Health Sciences Ethics Committee Decision 7/XX (Dated 23.03.2023).

Informed Consent Written informed consent was obtained from all participants.

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