RESEARCH PAPER

Effects of Theta Binaural Beats on Sleep Quality in Patients with Subacute Ischemic Stroke and Insomnia: A Pilot Study

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ABSTRACT

Background and Objective: Sleep disturbance, particularly insomnia, is common after stroke and may adversely affect recovery and quality of life. Theta binaural beats may provide a non-pharmacological approach to modulate sleep-related neural activity, but evidence in patients with subacute ischemic stroke remains limited. This pilot study evaluated the effects of theta binaural beats on subjective sleep quality and quantitative electroencephalographic characteristics in patients with subacute ischemic stroke and insomnia.

Methods: This interventional pretest–posttest pilot study included 36 patients with subacute ischemic stroke, comprising 18 participants with insomnia and 18 without insomnia. The insomnia group received theta binaural beats for 30 minutes daily for 30 consecutive days, while the non-insomnia group received noise stimulation. Sleep quality was assessed using the Pittsburgh Sleep Quality Index before and after the intervention. Electrophysiological outcomes included absolute theta power, relative theta power, and Z-score–normalized relative theta power. Within-group changes were analyzed using the Wilcoxon signed-rank test, and between-group differences in change were assessed using the Mann–Whitney U test.

Results: In the insomnia group, sleep quality improved substantially, with the median Pittsburgh Sleep Quality Index score decreasing from 16.5 to 4.5 ($p<0.001$), and the change differed significantly from that in the non-insomnia group ($p<0.001$). Absolute theta power decreased significantly in bilateral frontopolar and right frontal regions after false discovery rate correction. However, between-group differences in absolute theta power change were not significant. No significant changes were observed in relative theta power or Z-score–normalized relative theta power.

Conclusion: Theta binaural beats were associated with substantial improvement in subjective sleep quality and exploratory regional changes in absolute theta activity in patients with subacute ischemic stroke and insomnia. These findings are preliminary and warrant confirmation in larger randomized controlled studies.

Keywords: *Electroencephalography; Insomnia; Ischemic Stroke; Sleep Initiation and Maintenance Disorders; Theta Rhythm*

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INTRODUCTION

Stroke remains a leading cause of death and disability worldwide, with ischemic stroke accounting for a substantial proportion of the global cerebrovascular disease burden and continuing to impose major health and socioeconomic consequences [1,2]. Beyond motor and cognitive impairment, stroke survivors frequently experience sleep disturbances that may interfere with rehabilitation, functional recovery, and quality of life [3–5]. Poor sleep quality affects more than half of stroke survivors, highlighting sleep disturbance as an important but potentially modifiable component of post-stroke care [6–8]. Insomnia is particularly relevant, with a recent meta-analysis estimating that nearly half of patients with stroke develop insomnia during follow-up, including those with ischemic stroke or transient ischemic attack [9].

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The clinical importance of post-stroke insomnia extends beyond nocturnal symptoms, as persistent sleep disturbance may contribute to fatigue, impaired daytime functioning, poorer recovery, and reduced quality of life [10]. Although pharmacological and behavioral approaches are available, the management of post-stroke sleep disturbance remains challenging, creating interest in non-pharmacological interventions that can be integrated into neurological rehabilitation [11]. Binaural beats represent a potential auditory neuromodulatory approach in which two slightly different frequencies are presented separately to each ear, producing a perceived beat at the frequency difference between the tones [12]. Their potential effects on sleep and brain oscillatory activity have attracted increasing interest, although current evidence remains heterogeneous because of differences in stimulation protocols, study designs, EEG methodology, and outcome measures [13].

Theta-frequency stimulation is of particular interest because theta oscillations are involved in sleep-related neural processes and may represent a target for auditory modulation [14,15]. Experimental evidence from individuals with primary insomnia indicates that theta binaural beats can alter absolute theta activity, providing preliminary support for their potential neurophysiological effects [16]. However, evidence regarding theta binaural beats in patients with neurological disorders, particularly those with subacute ischemic stroke and comorbid insomnia, remains limited. Therefore, this pilot study aimed to evaluate the effects of theta binaural beats on subjective sleep quality and quantitative electroencephalographic characteristics in patients with subacute ischemic stroke and insomnia, focusing on absolute theta power, relative theta power, and Z-score–normalized relative theta power.

MATERIALS AND METHODS

Study Design and Setting

This study employed an interventional pretest–posttest design involving two clinical groups to evaluate changes in sleep quality and quantitative electroencephalographic (qEEG) spectral characteristics following auditory stimulation in patients with subacute ischemic stroke. Participants were classified at baseline according to their sleep status into an insomnia group and a non-insomnia group based on the Pittsburgh Sleep Quality Index (PSQI). Participants in the insomnia group received theta binaural beats auditory stimulation, whereas those in the non-insomnia group received noise stimulation as a clinical comparison condition. Assessments were performed before and after the 30-day stimulation period to evaluate changes in subjective sleep quality and electrophysiological brain activity.

The study was conducted at Dr. Wahidin Sudirohusodo General Hospital, Hasanuddin University Hospital, and affiliated teaching hospitals in Makassar, Indonesia. Recruitment and data collection commenced in February 2026 and continued until the required sample size was achieved.

Participants and Sampling

The study population consisted of patients diagnosed with subacute ischemic stroke who were treated at the participating hospitals during the study period. Participants were recruited using consecutive sampling, whereby eligible patients presenting to the participating hospitals were screened sequentially and enrolled until the predetermined sample size was reached.

Eligible participants were adults with subacute ischemic stroke who could be classified according to their baseline sleep status and were able to complete the study procedures. All participants were required to be cooperative and willing to participate after receiving a detailed explanation of the study procedures and providing written informed consent. Patients were excluded if they had a history of epilepsy, head trauma, other neurological disorders that could affect EEG activity, hearing impairment, anxiety disorders or other psychiatric disorders, or were receiving psychoactive or sedative medications. Participants were withdrawn from the study if they withdrew consent, failed to complete the study procedures, initiated use of an exclusion medication during the study, or died during the observation period.

Sample Size

The minimum sample size was calculated using a two-independent-group comparison formula based on the expected difference in mean outcome between the intervention and comparison conditions. With a two-sided significance level of 5% ($\alpha = 0.05$), 90% statistical power ($\beta = 0.10$), and the expected standard deviation and mean difference derived from the reference data, the calculation yielded a minimum requirement of 18 participants per group. Thus, the total minimum sample size was 36 participants.

Participants were subsequently classified into 18 participants with insomnia and 18 participants without insomnia according to their baseline sleep assessment. The insomnia group received theta binaural beats stimulation, while the non-insomnia group received noise stimulation. Because the study was designed as a pilot clinical investigation, the non-insomnia group served primarily as a clinical comparison group to explore whether electrophysiological and sleep-related changes differed according to baseline insomnia status.

Clinical Assessment and Baseline Procedures

After eligibility screening, demographic and clinical characteristics were recorded, including age, sex, educational level, contact information, and relevant clinical information. A clinical history and general and neurological examinations were subsequently performed to confirm eligibility and characterize the participants.

Sleep status was assessed before the intervention using the Indonesian-validated version of the PSQI. The questionnaire evaluates subjective sleep quality across

seven components, including sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. The total score ranges from 0 to 21, with higher scores indicating poorer sleep quality. A total score of ≤5 was categorized as good sleep quality, whereas a score >5 indicated poor sleep quality. The baseline assessment was used to classify participants into the insomnia and non-insomnia groups.

Auditory Stimulation Protocol

The auditory stimulation protocol was administered in a quiet and comfortable environment. Participants were seated in a comfortable position and instructed to close their eyes, relax, and breathe regularly to minimize external sensory stimulation during the procedure. Theta binaural beats were delivered using the Brain Wave auditory stimulation application through stereo headphones connected to a computer or compatible playback device. The stimulation consisted of two slightly different auditory frequencies delivered separately to the right and left ears, generating a perceived binaural beat within the theta-frequency range.

Participants in the insomnia group received theta binaural beats for 30 minutes per session, approximately 30 minutes before their usual bedtime. The stimulation was administered once daily for 30 consecutive days. The sound intensity was adjusted to a comfortable level for each participant while maintaining consistent delivery through stereo headphones. Participants in the non-insomnia group underwent the same procedural conditions and duration of auditory exposure but received noise stimulation as the comparison condition.

qEEG Acquisition and Processing

qEEG recordings were obtained at baseline before the initiation of auditory stimulation and repeated after completion of the 30-day intervention period. Before each recording, the EEG system underwent a 30-second calibration procedure. Recordings were performed under standardized conditions in a quiet environment, with participants seated comfortably and instructed to remain relaxed with their eyes closed.

EEG signals were acquired using the Cadwell Easy III system and subsequently processed using NeuroGuide Deluxe software. Spectral analysis was performed using Fast Fourier Transform (FFT) to transform the EEG signal from the time domain into the frequency domain. Theta activity was evaluated within the 4–8 Hz frequency range. Absolute theta power represented the spectral power within the theta frequency range, whereas relative theta power represented theta power relative to the total spectral power within the analyzed frequency spectrum.

The resulting spectral measures were further standardized against the normative reference database available within the qEEG analysis system to obtain Z-scores. A Z-score of zero represented the normative mean, whereas positive and negative values indicated spectral power above and below the normative reference mean, respectively. The principal electrophysiological outcomes were FFT absolute theta power, FFT relative theta power, and Z-score–normalized FFT relative theta power across the analyzed brain regions and hemispheres.

Outcome Measures

The primary clinical outcome was change in subjective sleep quality, assessed using the total PSQI score before and after the stimulation period. The primary electrophysiological outcomes were changes in FFT absolute theta power, FFT relative theta power, and Z-score–normalized FFT relative theta power between baseline and post-intervention assessments. For each outcome, the magnitude of change was calculated as the post-intervention value minus the baseline value (Δ = post-intervention − baseline). Positive or negative changes were interpreted according to the direction and physiological meaning of the respective parameter.

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics version 27. Data distribution was assessed using the Shapiro–Wilk test. Continuous variables that did not meet the assumption of normality were summarized using the median and interquartile range (25th–75th percentiles), whereas categorical variables were presented as frequencies and percentages.

Within-group changes between baseline and post-intervention assessments were evaluated using the Wilcoxon signed-rank test. The magnitude of change for each outcome was calculated as the difference between post-intervention and baseline values. Differences in these changes between the insomnia and non-insomnia groups were assessed using the Mann–Whitney U test. Statistical significance was defined as a two-sided p value <0.05.

Within-group and between-group findings were interpreted separately. A statistically significant change within one group was not considered evidence of a differential treatment effect unless the corresponding between-group comparison of change also demonstrated statistical significance.

Because FFT absolute theta power involved multiple regional comparisons, the Benjamini–Hochberg procedure was applied to control the false discovery rate (FDR) at 5%. Multiple-comparison correction was performed for 16 within-group regional comparisons, comprising eight region–hemisphere combinations in the insomnia group and eight corresponding combinations in the non-insomnia group. Corrected results were reported as FDR-adjusted p values (p-FDR), with p-FDR <0.05 considered statistically significant.

Ethical Considerations

The study was conducted in accordance with applicable ethical principles for research involving human participants. Ethical approval was obtained from the Health Research Ethics Committee of the Faculty of Medicine, Hasanuddin University, under approval number 947/UN4.6.4.5.31/PP36/2026. Written informed consent was obtained from all participants before enrollment.

Participants were informed regarding the study objectives, procedures, potential benefits, and possible risks associated with participation, and were free to withdraw from the study at any time without consequences for their clinical care.

RESULTS

Participant Characteristics

The study was conducted at Dr. Wahidin Sudirohusodo General Hospital, Hasanuddin University Hospital, and affiliated teaching hospitals in Makassar, Indonesia, from February to July 2026. A total of 36 patients with subacute ischemic stroke were included, comprising 18 participants with insomnia and 18 without insomnia. The baseline characteristics of the participants are presented in Table 1. The mean age was comparable between the two groups, with participants generally in the middle-to-late adult age range. Both groups were predominantly male. The timing of stroke onset showed a wider distribution in the non-insomnia group, with a later median onset than that observed in the insomnia group. Overall, the baseline characteristics indicate that the study population represented patients in the subacute phase of ischemic stroke, although differences in the timing of assessment should be considered when interpreting subsequent clinical and electrophysiological changes.

Change in Sleep Quality

Sleep quality was assessed using the PSQI before and after the 30-day auditory stimulation period. As the change scores were not uniformly normally distributed, continuous data are presented as median and interquartile range (IQR). Changes were calculated as post-intervention minus baseline values, with negative values indicating improvement in sleep quality.

In the insomnia group, the median PSQI score decreased substantially from 16.5 (IQR, 14.0–18.0) at baseline to 4.5 (IQR, 4.0–5.0) after the intervention, corresponding to a median change of −12.5 (IQR, −13.75 to −9.25; $p<0.001$). All participants in this group had baseline PSQI scores above the threshold for poor sleep quality, whereas their post-intervention scores were within the range corresponding to good sleep quality. In contrast, the non-insomnia group showed no meaningful change in sleep quality, with a median change of 0.0 (IQR, 0.0–1.0; $p=0.675$). The magnitude of change in PSQI differed significantly between groups ($p<0.001$), indicating a substantially greater improvement in subjective sleep quality in the insomnia group. These findings are summarized in Table 2. A graphical representation of individual or paired changes in PSQI is recommended to complement the tabulated results.

Changes in FFT Absolute Theta Power

FFT absolute theta power was assessed across four cortical regions in each hemisphere. In the insomnia group, significant pre–post reductions were initially observed in the left frontopolar, left temporal, right frontopolar, and right frontal regions. After Benjamini–Hochberg correction for multiple comparisons, the reductions remained statistically significant in the bilateral frontopolar and right frontal regions, with large effect sizes. The change in left temporal activity did not remain significant after correction and was therefore considered an exploratory finding.

No significant pre–post changes were detected in any region in the non-insomnia group after correction. Although several regions demonstrated small-to-moderate effect sizes, these changes did not reach statistical significance. Importantly, comparison of the magnitude of change between groups showed no significant differences across any examined region. Thus, despite the significant within-group reductions observed in selected regions among participants with insomnia, the corresponding changes were not statistically different from those observed in the non-insomnia group. These findings indicate that the regional electrophysiological changes should be interpreted as exploratory rather than definitive evidence of a specific intervention effect.

The principal findings are presented in Table 3.

Changes in FFT Relative Theta Power

FFT relative theta power was assessed across four cortical regions in each hemisphere. No significant pre–post changes were observed in any region in either group. In the insomnia group, all within-group comparisons were non-significant, with effect sizes ranging from very small to small, indicating limited changes in the proportion of theta activity relative to total spectral power following stimulation.

Similarly, no significant changes were observed in the non-insomnia group. The largest within-group effect was observed in the left central region, although this did not reach statistical significance ($p=0.124$; $r=0.363$). Importantly, comparison of the magnitude of change between groups revealed no significant differences across any region, with all between-group effect sizes remaining small or very small.

Overall, these findings indicate that the stimulation period was not associated with a consistent change in relative theta power, either within groups or in the magnitude of change between the insomnia and non-insomnia groups. This pattern contrasts with the regional changes observed for absolute theta power and suggests that the observed electrophysiological changes were not accompanied by a measurable shift in the proportion of theta activity within the overall spectral power distribution. The complete regional findings are presented in Table 4.

Changes in Z-score FFT Relative Theta Power

Z-score FFT relative theta power was evaluated across four cortical regions in each hemisphere. No significant pre–post changes were observed in any region in the insomnia group, with within-group p values ranging from 0.459 to 0.879. The median changes were small and showed no consistent directional pattern across regions.

Similarly, no significant changes were identified in the non-insomnia group. Although the left central region showed the smallest within-group p value (p=0.059), this did not reach the prespecified threshold for statistical significance. The direction and magnitude of changes also varied across regions.

Importantly, comparison of the magnitude of change between groups showed no significant differences across any region, with between-group p values ranging from 0.142 to 1.000. Overall, neither within-group nor between-group analyses demonstrated evidence of a consistent shift in Z-score–normalized relative theta power following the study protocol. These findings indicate that the regional changes observed in absolute theta power were not accompanied by a corresponding normalization of relative theta activity toward the normative reference distribution. The complete regional findings are presented in Table 5.

DISCUSSION

The present study demonstrates a distinct improvement in subjective sleep quality among patients with subacute ischemic stroke and insomnia following theta binaural beats stimulation. This finding is clinically relevant because sleep disturbance is common after stroke and may adversely affect neurological recovery, functional rehabilitation, fatigue, and quality of life. These findings are in line with Mayer-Suess et al. (2024) [17], who emphasized the bidirectional relationship between sleep disturbances and post-stroke recovery, and with Luo et al. (2023) [6], who reported a substantial burden of poor sleep quality among stroke survivors. The marked improvement in subjective sleep quality observed in the present study therefore supports the potential clinical relevance of non-pharmacological auditory approaches for post-stroke sleep disturbance.

The observed improvement is also biologically plausible. Binaural beats are generated when slightly different frequencies are presented separately to each ear, producing a perceived beat corresponding to the frequency difference between the two tones. Theta-frequency stimulation has been proposed to influence neural oscillatory activity associated with relaxation and sleep regulation. Bavafa et al. (2023) reported that theta binaural beats could modulate absolute theta activity in individuals with primary insomnia, providing preliminary neurophysiological support for the present approach [16]. However, the clinical population in the current study differs substantially from individuals with primary insomnia because post-stroke brain activity may be influenced by lesion characteristics, neural reorganization, medications, and recovery stage. Therefore, the electrophysiological response to auditory stimulation in stroke survivors may not necessarily reproduce findings observed in otherwise neurologically healthy populations with insomnia.

The changes observed in absolute theta power in selected cortical regions further suggest that theta binaural beats may influence cortical oscillatory activity, although the response was regionally heterogeneous [16]. This partial concordance with Bavafa et al. (2023) is important because it indicates that the electrophysiological effects of theta stimulation may be detectable at the level of absolute spectral power. Nevertheless, the absence of a consistent difference in change between the insomnia and non-insomnia groups limits the interpretation of these regional findings as evidence of a specific intervention effect. A significant pre–post change within one group does not establish superiority over another condition when the corresponding between-group comparison is not significant. This distinction is particularly important in the present study because the groups were defined according to baseline sleep status rather than randomized to theta stimulation versus placebo among patients with comparable insomnia.

In contrast to absolute theta power, relative theta power and its Z-score representation did not demonstrate a consistent change following stimulation. This apparent discrepancy is physiologically plausible because absolute power reflects the magnitude of activity within a specific frequency band, whereas relative power represents its proportion of total spectral activity. Thus, an increase or decrease in absolute theta power may occur without a corresponding change in relative theta power if other frequency bands change concurrently. The lack of a consistent shift in the normalized measure also suggests that the observed electrophysiological changes did not represent a generalized movement toward or away from the normative reference distribution.

These findings are consistent with the broader evidence indicating that the neurophysiological effects of binaural beats remain heterogeneous. Ingendoh et al. (2023) reported substantial variability across studies in stimulation frequency, duration, experimental protocols, EEG methodology, and observed oscillatory responses [13]. Such heterogeneity may be particularly relevant in stroke populations, where lesion location, cortical reorganization, baseline EEG characteristics, and clinical status can substantially influence spectral activity. Accordingly, the present findings suggest that sleep improvement and electrophysiological modulation may represent partially independent dimensions of response rather than necessarily occurring in parallel.

The discrepancy between the prominent improvement in subjective sleep quality and the less consistent qEEG changes is noteworthy. Sleep quality is a multidimensional clinical construct influenced not only by cortical oscillatory activity but also by psychological state, environmental factors, sleep behavior, medications, and the broader post-stroke recovery process. Consequently, improvement in subjective sleep quality does not necessarily require a uniform change in a single EEG frequency band. The present findings therefore support the use of both clinical and electrophysiological outcomes when evaluating auditory neuromodulation in post-stroke insomnia.

Several limitations should be considered when interpreting these findings. First, the pilot nature and limited sample size reduce statistical power and limit generalizability. Second, allocation according to baseline insomnia status rather than randomization between active and sham stimulation makes it difficult to distinguish the specific effect of theta binaural beats from natural recovery, expectancy effects, regression to the mean, or other nonspecific effects. The substantially different baseline sleep status between groups also introduces the possibility of a floor effect in the comparison group. Third, stroke-related heterogeneity and individual variability in EEG activity may have contributed to the regional inconsistency of the electrophysiological findings. Future studies should therefore employ a larger randomized controlled design in which patients with post-stroke insomnia are allocated to theta binaural beats or a well-matched sham condition, with standardized stimulation parameters and longitudinal assessment of both sleep and qEEG outcomes.

Overall, the findings provide preliminary evidence that theta binaural beats may be associated with clinically meaningful improvement in subjective sleep quality among patients with subacute ischemic stroke and insomnia, accompanied by exploratory regional changes in absolute theta activity. However, the absence of consistent between-group changes in relative theta power and Z-score measures indicates that the neurophysiological mechanism remains uncertain. These results should therefore be regarded as hypothesis-generating and provide a rationale for adequately powered randomized studies integrating clinical sleep outcomes with objective electrophysiological measures.

LIMITATIONS

This study has several limitations that should be considered when interpreting its findings. First, the pilot design and relatively small sample size limited statistical power and generalizability, particularly for detecting modest electrophysiological effects. Second, the groups differed substantially in baseline sleep quality, creating potential floor effects in the non-insomnia group and regression toward the mean in participants with poorer baseline sleep, thereby limiting direct interpretation of between-group differences.

Third, sleep quality was assessed using a subjective questionnaire and therefore may not fully capture objective sleep architecture or physiological parameters that could be obtained through polysomnography or other objective sleep measures. Fourth, the regional qEEG analysis involved multiple comparisons. Although the Benjamini–Hochberg procedure was applied to control the false discovery rate for the prespecified absolute theta power comparisons, other regional analyses remained exploratory and should be interpreted cautiously.

Fifth, the non-parametric analyses used because of the distribution and sample characteristics provided appropriate statistical inference but remained limited in their ability to detect small effects. Sixth, potentially important clinical and neurophysiological confounders, including lesion location and extent, stroke severity, medications, comorbidities, psychological status, sleep environment, and individual EEG characteristics, could not be completely controlled. Finally, the relatively short observation period prevented assessment of whether improvements in sleep quality or electrophysiological activity were sustained over the longer term. These limitations support the need for larger randomized controlled studies with well-matched sham controls, objective sleep assessment, prespecified electrophysiological outcomes, and longer follow-up.

CONCLUSIONS

Theta binaural beats auditory stimulation was associated with a clinically meaningful improvement in subjective sleep quality among patients with subacute ischemic stroke and insomnia, accompanied by exploratory regional modulation of absolute theta activity, particularly in bilateral frontopolar and right frontal regions. However, these electrophysiological changes were not accompanied by a significant difference in the magnitude of absolute theta power change compared with the non-insomnia group, nor by consistent changes in relative theta power or its Z-score representation. Overall, the findings suggest that theta binaural beats may have potential as a non-pharmacological approach for improving sleep quality after subacute ischemic stroke, while the observed electrophysiological effects remain preliminary and should be interpreted as hypothesis-generating. Larger randomized controlled studies using well-matched sham controls, objective sleep measures, and standardized qEEG outcomes are warranted to confirm the clinical and neurophysiological effects of this intervention.

DECLARATIONS

Consent for Publication

Not applicable. No individual participant data or identifiable information are presented in this manuscript.

Competing Interests

The authors declare that they have no competing interests.

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Authors' Contributions

MR: Conceptualization, Methodology, Investigation, Data Curation, Formal Analysis, Writing – Original Draft, and Project Administration. **MIB:** Conceptualization, Methodology, Supervision, and Writing – Review & Editing. **JT:** Conceptualization, Methodology, Supervision, and Writing – Review & Editing. **YAF:** Investigation, Data Curation, and Writing – Review & Editing. **CR:** Investigation, Data Curation, and Writing – Review & Editing. **ADW:** Supervision, Validation, Writing – Review & Editing, and Project Administration. All authors reviewed and approved the final version of the

manuscript and agreed to be accountable for all aspects of the work.

Data Availability

The datasets generated and/or analyzed during the current study are not publicly available because they contain clinical data from human participants but may be made available from the corresponding author upon reasonable request and subject to applicable ethical and institutional requirements.

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TABLES

Table 1. Baseline Characteristics of Participants With Subacute Ischemic Stroke

Characteristic	Insomnia (n=18)	Non-insomnia (n=18)
Age, years, mean ± SD	56.17 ± 10.64	57.61 ± 8.88
Male, n (%)	13 (72.2)	11 (61.1)
Female, n (%)	5 (27.8)	7 (38.9)
Stroke onset, days, median (IQR)	32.0 (24.0–53.25)	52.5 (30.0–60.0)

Table 2. Change in PSQI Before and After Auditory Stimulation

PSQI	Insomnia (n=18)	Non-insomnia (n=18)	Between-group p-value*
Baseline	16.5 (14.0–18.0)	4.0 (3.0–5.0)	<0.001
Post-intervention	4.5 (4.0–5.0)	4.0 (3.25–5.0)	—
Change (Δ)	−12.5 (−13.75 to −9.25)	0.0 (0.0–1.0)	<0.001
Within-group p-value†	<0.001	0.675	—

*Between-group p-value based on the Mann–Whitney U test comparing Δ PSQI.

†Within-group p-value based on the Wilcoxon signed-rank test.

Data are presented as median (IQR). Δ = post-intervention − baseline.

Table 3. Regional Changes in FFT Absolute Theta Power After Stimulation

Hemisphere	Region	Insomnia: Δ power, median (IQR)	Within-group p-FDR	Effect size (r)	Non-insomnia: Δ power, median (IQR)	Between-group p	Between-group r
Left	Frontopolar	−1.54 (−7.14 to −0.28)	0.003	0.878	−0.95 (−4.05 to 1.21)	0.129	0.253
Right	Frontopolar	−1.46 (−5.21 to −0.08)	0.003	0.829	−0.90 (−4.40 to 0.67)	0.255	0.190
Right	Frontal	−0.86 (−2.98 to −0.13)	0.007	0.753	−1.19 (−3.96 to 1.82)	0.825	0.037

Values are presented as median (IQR). Δ = post-intervention − baseline. p-FDR was obtained using the Benjamini–Hochberg procedure with a false discovery rate of 5%. Between-group p values were derived using the Mann–Whitney U test comparing Δ values. Effect sizes were calculated as $|Z|/\sqrt{N}$.

Table 4. Changes in FFT Relative Theta Power Before and After Stimulation

Hemisphere	Region	Insomnia Δ median (IQR), %	p	r	Non-insomnia Δ median (IQR), %	p	r	Between-group p	r*
Left	Frontopolar	0.00 (−0.93 to 2.19)	0.632	0.113	−0.33 (−1.22 to 0.68)	0.384	0.205	0.411	0.137
Left	Frontal	0.03 (−0.45 to 0.61)	0.879	0.036	−0.27 (−2.16 to 0.46)	0.306	0.241	0.402	0.140
Left	Central	−0.28 (−0.59 to 1.28)	0.983	0.005	−0.64 (−2.96 to 0.14)	0.124	0.363	0.217	0.206
Left	Temporal	−0.13 (−0.80 to 0.65)	0.616	0.118	−0.17 (−1.32 to 0.60)	0.349	0.221	0.764	0.050
Right	Frontopolar	−0.01 (−0.69 to 4.29)	0.528	0.149	−0.15 (−1.49 to 0.62)	0.316	0.236	0.327	0.164
Right	Frontal	−0.14 (−0.66 to	0.862	0.041	0.00 (−0.85 to 0.74)	0.828	0.051	0.899	0.021

		1.12)							
Right	Central	−0.25 (−0.77 to 0.78)	0.795	0.061	−0.34 (−4.08 to 1.38)	0.360	0.216	0.477	0.119
Right	Temporal	−0.17 (−0.95 to 0.64)	0.636	0.111	0.02 (−0.84 to 0.32)	0.948	0.015	0.740	0.055

Values are presented as median (IQR). FFT relative theta power is expressed as a percentage (%). Δ = post-intervention − baseline. Within-group p values were obtained using the Wilcoxon signed-rank test. Between-group p values compare Δ using the Mann–Whitney U test. *r* and $r*$ represent within- and between-group effect sizes, respectively.

Table 5. Changes in Z-score FFT Relative Theta Power Before and After Stimulation

Hemisphere	**Region**	**Insomnia Δ, median (IQR), Z-score**	**p†**	**Non-insomnia Δ, median (IQR), Z-score**	**p†**	**Between-group p***
Left	Frontopolar	0.01 (−0.10 to 0.49)	0.601	0.06 (−0.49 to 0.39)	0.868	0.792
Left	Frontal	−0.01 (−0.26 to 0.26)	0.777	−0.11 (−0.80 to 0.28)	0.554	0.704
Left	Central	−0.10 (−0.31 to 0.18)	0.459	−0.39 (−0.92 to −0.01)	0.059	0.142
Left	Temporal	−0.03 (−0.16 to 0.40)	0.879	−0.13 (−0.77 to 0.28)	0.463	0.419
Right	Frontopolar	0.08 (−0.37 to 0.45)	0.616	−0.01 (−0.26 to 0.47)	0.756	1.000
Right	Frontal	0.04 (−0.18 to 0.33)	0.616	−0.06 (−0.45 to 0.62)	0.705	0.499
Right	Central	−0.10 (−0.18 to 0.20)	0.794	−0.15 (−0.97 to 0.17)	0.246	0.400
Right	Temporal	0.03 (−0.28 to 0.23)	0.828	0.02 (−1.25 to 0.39)	0.943	0.729

Values are presented as median (IQR). Z-score is dimensionless. Δ = post-intervention − baseline. Within-group p values were obtained using the Wilcoxon signed-rank test. Between-group p values compare Δ using the Mann–Whitney U test.