

A brief pre-test relaxation protocol improves sleep initiation during diagnostic polysomnography: a randomized controlled study

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Abstract

Purpose

Laboratory adaptation and pre-test arousal may prolong sleep-onset latency during attended polysomnography (PSG). We evaluated whether a brief combined music and paced-breathing protocol shortens sleep-onset latency during diagnostic PSG.

Methods

In this prospective, single-centre, assessor-blinded, parallel-group randomized controlled trial, 72 adults referred for Type 1 PSG for suspected obstructive sleep apnea were randomized. Seventy participants formed the modified intention-to-treat cohort (intervention $n = 34$; control $n = 36$). The intervention comprised 30 minutes of standardized slow-tempo instrumental music, with guided paced breathing during the first 15 minutes; controls underwent an equal-duration standard preparation period. Sleep-onset latency, defined as the interval from lights-off to the first epoch scored as sleep, was the primary outcome. Respiratory, oxygenation, and sleep-architecture measures were exploratory secondary outcomes.

Results

Sleep-onset latency was shorter in the intervention group (median 8.8 min [IQR 4.5–13.4] vs 17.8 min [8.9–33.6]; $p < 0.001$; rank-biserial $r = 0.45$; Hodges–Lehmann shift – 9.5 min; fully adjusted $\beta = -10.7$ min, 95% CI – 18.2 to – 3.2). No statistically significant between-group differences were detected in AHI, REM-AHI, non-REM AHI, minimum oxygen saturation, total sleep time, sleep efficiency, REM latency, arousal index, or sleep-stage proportions. Mean oxygen saturation was 0.5 percentage points higher in the intervention group ($p = 0.014$).

Conclusion

A brief pre-test music and paced-breathing protocol shortened sleep-onset latency during diagnostic PSG. The exploratory secondary PSG findings did not show measurable suppression of respiratory burden in this sample, but the trial was not designed to establish equivalence or diagnostic neutrality.

Introduction

Obstructive sleep apnea (OSA) is among the most common chronic disorders, with close to one billion adults aged 30–69 years estimated to have at least mild disease worldwide [1]. Attended Type 1 polysomnography (PSG) remains the reference standard for diagnosis and severity assessment because it records sleep stages, respiratory events, oxygenation, body position, limb activity, and cardiac rhythm.

The unfamiliar and instrumented laboratory environment may delay sleep onset and alter sleep continuity. Studies of the first-night effect report longer sleep-onset and REM latency, lower total sleep time and sleep efficiency, reduced REM sleep, and more stage N1 on the first laboratory night than on subsequent nights [2, 3]. The present study did not include a second reference night and therefore did not directly quantify a first-night effect; nevertheless, delayed sleep initiation remains a practical problem during attended PSG.

A brief non-pharmacological preparation procedure could improve initiation of the recording without drug-related risk. Instrumental music has been associated with improved sleep outcomes in several populations [4, 5], while slow paced breathing can influence autonomic regulation and reduce physiological arousal [6, 7]. Both approaches are relevant to the cognitive and somatic arousal that can delay sleep onset [8, 9].

In a diagnostic study, shorter latency is useful only if the procedure does not measurably suppress the respiratory and oxygenation findings used to identify and grade OSA. However, absence of a statistically significant difference in secondary PSG outcomes is not equivalent to proof that the outcomes are identical.

We therefore tested whether a brief combined music and paced-breathing protocol shortens sleep-onset latency during diagnostic PSG. Sleep-onset latency was the pre-specified primary outcome; respiratory, oxygenation, and sleep-architecture measures were evaluated as exploratory secondary outcomes.

Methods

Design and setting

This was a prospective, single-centre, assessor-blinded, parallel-group randomized controlled trial conducted in the sleep laboratory of Ankara Bilkent City Hospital. The study was approved by the institutional ethics committee (TABED 2-26-1877, January 2026) and conducted in accordance with the Declaration of Helsinki. All participants provided written informed consent. The trial was not registered in a publicly accessible clinical trial registry; the prospectively approved protocol and institutional study record are retained by the hospital ethics committee. Participant flow is shown in Fig. 1.

Participants

Adults aged 18–65 years referred for Type 1 PSG for witnessed apnea, snoring, or excessive daytime sleepiness were eligible. Exclusion criteria were diabetes mellitus, acute coronary syndrome, chronic obstructive pulmonary disease, autoimmune or rheumatologic disease, chronic infection, malignancy, antidepressant or other medication use known to alter sleep architecture, and morbid obesity (body-mass index [BMI] ≥ 40 kg/m²). Of 72 randomized participants, two allocated to the intervention group were subsequently found to be taking antidepressants. PSG outcome data were unavailable for these

participants, and the exclusion decision was made before any PSG outcomes were reviewed. The modified intention-to-treat cohort therefore comprised 70 participants (control n = 36; intervention n = 34).

Sample size

An a priori sample-size calculation for the primary outcome was documented in the approved study protocol. The calculation indicated that 36 participants per group were required at a two-sided α of 0.05. Accordingly, 72 participants were randomized.

Randomization and blinding

Participants were allocated in a 1:1 ratio using sealed envelopes prepared by an independent individual who was not involved in enrolment, intervention delivery, or outcome assessment. The recording technician opened the envelope immediately before the pre-recording preparation period. PSG recordings were staged and scored by an otolaryngologist who was unaware of group assignment. Participants and the attending technician could not be blinded because the intervention was experiential.

Intervention

During the 30-minute pre-recording period, participants in the intervention group listened to a standardized slow-tempo instrumental track at approximately 60 beats per minute. The track was tuned to 432 Hz to maintain a uniform auditory stimulus; tuning frequency was not evaluated as an independent intervention component. During the first 15 minutes, participants additionally performed guided paced box breathing using a standardized instructional video (4 s inhalation, 4 s hold, 4 s exhalation, 4 s hold). Control participants underwent an equal-duration standard preparation period without music or guided breathing. Room lighting and temperature were standardized in both groups. Electrodes were then applied and the PSG recording commenced.

Polysomnography

Recordings were acquired with an Alice 6 system (Philips Respironics, the Netherlands) under continuous technician supervision and were scored manually according to the American Academy of Sleep Medicine Manual for the Scoring of Sleep and Associated Events, Version 3 [10]. Apnea was defined as a $\geq 90\%$ reduction in oronasal airflow lasting ≥ 10 s; hypopnea as a $\geq 30\%$ reduction in airflow lasting ≥ 10 s accompanied by a $\geq 3\%$ oxygen desaturation or an arousal.

Outcomes

The pre-specified primary outcome was sleep-onset latency, defined as the interval from lights-off to the first epoch scored as sleep. Exploratory secondary PSG outcomes included the apnea–hypopnea index (AHI), REM-AHI, non-REM AHI, minimum and mean oxygen saturation (SpO₂), total sleep time, sleep efficiency, REM latency, the proportions of stages N1, N2, N3 and REM, and the arousal index.

Data handling

Analyses used the modified intention-to-treat cohort of 70 participants. REM-AHI, non-REM AHI, and minimum SpO₂ were unavailable for six participants and were analysed using available cases (intervention 33/34; control 31/36). No missing values were imputed.

Statistics

Continuous variables were inspected for distributional shape. Because several PSG variables were skewed, between-group comparisons used the Mann–Whitney U test and are reported as medians with interquartile ranges. Rank-biserial correlation (r) was used as the effect size, and the Hodges–Lehmann estimator was reported for the primary outcome. The robustness of the primary effect to the baseline BMI imbalance was assessed with sensitivity analysis-of-covariance (ANCOVA) models (crude; adjusted for time in bed and mean SpO₂; and fully adjusted for age, BMI, and AHI). Categorical variables were compared with the chi-square test or Fisher’s exact test, as appropriate. The primary outcome was tested at a two-sided α of 0.05. Secondary PSG outcomes were exploratory; no adjustment for multiple comparisons was applied. Because the trial was powered for sleep-onset latency rather than for equivalence or non-inferiority of respiratory, oxygenation, or sleep-architecture measures, non-significant secondary results were not interpreted as proof of diagnostic neutrality. Analyses were performed using Python (statsmodels).

Results

Participant flow

Seventy-two participants were randomized. Two participants allocated to the intervention group were excluded after antidepressant use was identified and because PSG outcome data were unavailable, leaving 70 participants in the modified intention-to-treat cohort (control n = 36; intervention n = 34; Fig. 1). No analysed participant withdrew, and no analysed recording failed technically. REM-AHI, non-REM AHI, and minimum SpO₂ were available for 64 participants.

Baseline characteristics

The groups had similar age and sex distributions. BMI was higher in the intervention group (31.2 ± 4.3 vs 28.7 ± 4.5 kg/m²; p = 0.012). Participant characteristics are summarized in Table 1.

Primary outcome: sleep-onset latency

Sleep-onset latency was shorter in the intervention group: median 8.8 min (IQR 4.5–13.4) versus 17.8 min (8.9–33.6) in the control group (Mann–Whitney U $p < 0.001$; rank-biserial $r = 0.45$). The Hodges–Lehmann estimate of the between-group shift was -9.5 min (Fig. 2).

Because BMI was imbalanced at baseline, the robustness of the primary effect was examined in sensitivity ANCOVA models (Table 3). The crude group effect was -12.7 min (95% CI -19.1 to -6.2 ; $p < 0.001$); after adjustment for time in bed and mean SpO_2 it was -12.0 min (95% CI -18.8 to -5.1 ; $p < 0.001$); and in the fully adjusted model additionally including age, BMI, and AHI it remained -10.7 min (95% CI -18.2 to -3.2 ; $p = 0.006$; $R^2 = 0.21$). The stability of the estimate across models indicates that the latency reduction was not explained by the baseline imbalance.

Exploratory respiratory outcomes

AHI was 27.5 (IQR 8.3–36.5) events/h in the intervention group and 17.4 (7.8–41.7) events/h in the control group ($p = 0.58$; $r = 0.07$). REM-AHI (26.1 [8.9–65.7] vs 18.0 [8.6–48.8] events/h; $p = 0.95$; $r = 0.01$) and non-REM AHI (20.8 [8.4–31.6] vs 13.9 [6.6–35.7] events/h; $p = 0.66$; $r = 0.05$) also did not differ significantly between groups (Table 2).

Exploratory oxygenation outcomes

Minimum SpO_2 was 84.0% (IQR 73.0–87.0) in the intervention group and 82.0% (78.5–84.0) in the control group ($p = 0.79$; $r = 0.03$). Mean SpO_2 was 93.5% (92.0–95.0) and 93.0% (91.0–93.0), respectively, a difference of 0.5 percentage points ($p = 0.014$; $r = 0.29$).

Exploratory sleep-architecture outcomes

Total sleep time, sleep efficiency, N1, N2, N3 and REM proportions, arousal index, and REM latency did not differ significantly between groups (all $p > 0.1$; Table 2).

Discussion

In adults undergoing diagnostic Type 1 PSG for suspected OSA, a brief combined music and paced-breathing protocol shortened sleep-onset latency by approximately nine minutes. This objective effect was observed after an equal-duration preparation period in both groups and with PSG scoring blinded to allocation.

The finding is consistent with evidence that music-based interventions and slow breathing can reduce arousal and improve sleep-related outcomes [4–9, 14]. The standardized track was tuned to 432 Hz. Previous pilot and clinical studies have examined this tuning frequency in relation to anxiety and physiological responses [15, 16], but the present trial did not compare tuning frequencies and cannot support a frequency-specific effect. Because music and paced breathing were delivered as a bundle, their independent contributions also cannot be separated.

Laboratory adaptation and pre-test arousal may delay sleep initiation [2, 3, 11–13]. A standardized, drug-free protocol that can be delivered during routine preparation may therefore be useful when patients have difficulty settling in the sleep laboratory. Because it requires only a recorded audio track and a brief guided-breathing video, the protocol adds negligible cost, uses no pharmacological agent and no specialist training, and can be incorporated into routine preparation without disrupting laboratory workflow. The present study did not measure technician workload, patient comfort, technically inadequate studies, or repeat testing; it supports improved initiation of PSG rather than broader claims of workflow efficiency.

The exploratory respiratory findings did not show statistically significant reductions in AHI, REM-AHI, non-REM AHI, or minimum SpO₂. This provides no measurable signal of respiratory suppression in this sample. However, the study was not powered around pre-specified equivalence margins, and these non-significant results cannot establish that the protocol is diagnostically neutral. The small difference in mean SpO₂ should also be interpreted as an exploratory finding whose clinical relevance was not evaluated.

Sleep-stage proportions, REM latency, total sleep time, sleep efficiency, and arousal index were also similar between groups. These findings are reassuring because a marked alteration in REM expression or sleep-stage distribution could affect measured OSA severity. Nevertheless, the same statistical limitation applies: similar group summaries do not constitute formal equivalence.

The study has several strengths, including prospective randomization, an equal-duration control condition, an objective primary outcome, blinded PSG scoring, and a simple reproducible intervention. These features support the internal validity of the primary sleep-onset finding.

Limitations

First, this was a single-centre, single-night study. Without a second reference night, it cannot quantify a first-night effect or determine whether the intervention changes night-to-night variability. Second, participants and technicians were not blinded, and no sham or attention-control condition was used; expectation and non-specific attention effects may therefore have contributed to the primary outcome. Third, the bundled intervention does not separate music, paced breathing, video guidance, or tuning frequency. Fourth, the study was powered for sleep-onset latency, not for equivalence of secondary PSG outcomes. Fifth, autonomic, biochemical, anxiety, comfort, and satisfaction measures were not collected, so the proposed mechanism remains inferential. Sixth, two participants were excluded after randomization because antidepressant use was identified and PSG outcome data were unavailable; the analysis was therefore modified intention-to-treat. REM-AHI, non-REM AHI, and minimum SpO₂ were available for 64 participants. Finally, the trial was not registered in a publicly accessible registry, although the approved protocol and institutional study record are retained by the hospital ethics committee.

Future directions

Future multicentre studies should include an attention-control condition, factorial allocation to separate music from breathing, and autonomic or patient-reported measures. Equivalence or non-inferiority margins should be pre-specified for diagnostically important PSG indices. Studies should also determine whether shorter sleep-onset latency improves patient experience or reduces technically inadequate and repeated recordings.

Conclusion

A brief pre-test protocol combining standardized instrumental music and guided paced breathing shortened sleep-onset latency during diagnostic PSG. Exploratory secondary findings did not show measurable respiratory suppression in this sample, but larger equivalence-oriented studies are required before diagnostic neutrality can be concluded.

Declarations

Author Contribution

Şeyda Akbal Çufalı and Ece Şeyma Tüzel contributed to the study methodology and protocol development. Ömer Faruk Çufalı was responsible for participant recruitment, data collection, and clinical follow-up. All authors contributed to the interpretation of the findings, manuscript preparation, and critical revision of the manuscript. All authors read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

Data Availability

This study generated clinical and polysomnographic research data. The deidentified data that support the findings of this study are available from the corresponding author upon reasonable request, subject to institutional and ethical restrictions. The analysis code is available from the corresponding author upon reasonable request.

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Tables

Table 1
Participant characteristics

Variable	Control (n = 36)	Intervention (n = 34)	p	Test
Age, years (mean ± SD)	42.0 ± 10.8	42.9 ± 11.5	0.69	MWU
Female, n (%)	11 (31)	14 (41)	0.50	χ ²
BMI, kg/m ² (mean ± SD)	28.7 ± 4.5	31.2 ± 4.3	0.012	MWU
Time in bed, min	440.8 (426.3–453.6)	428.6 (423.7–437.2)	0.078	MWU
Mean SpO ₂ , %	93.0 (91.0–93.0)	93.5 (92.0–95.0)	0.014	MWU

Values are mean ± SD unless otherwise stated. BMI, body-mass index.

Table 2
Primary and exploratory secondary PSG outcomes by group

Parameter	Control	Intervention	p	r
Sleep-onset latency, min	17.8 (8.9–33.6)	8.8 (4.5–13.4)	< 0.001	0.45
AHI, events/h	17.4 (7.8–41.7)	27.5 (8.3–36.5)	0.58	0.07
REM-AHI, events/h †	18.0 (8.6–48.8)	26.1 (8.9–65.7)	0.95	0.01
Non-REM AHI, events/h †	13.9 (6.6–35.7)	20.8 (8.4–31.6)	0.66	0.05
Minimum SpO ₂ , % †	82.0 (78.5–84.0)	84.0 (73.0–87.0)	0.79	0.03
Mean SpO ₂ , %	93.0 (91.0–93.0)	93.5 (92.0–95.0)	0.014	0.29
Total sleep time, min	373.5 (329.2–407.0)	371.2 (345.0–400.5)	0.51	0.08
Sleep efficiency, %	83.5 (74.3–89.7)	86.8 (80.9–90.5)	0.12	0.18
N1, %	3.5 (2.4–5.2)	3.6 (2.9–4.5)	0.96	0.01
N2, %	59.7 (57.0–67.6)	63.3 (55.9–66.6)	0.46	0.09
N3, %	19.4 (14.3–24.3)	20.2 (14.2–25.1)	0.81	0.03
REM, %	14.7 (9.5–19.4)	14.9 (9.6–17.6)	0.65	0.05
Arousal index	1.9 (0.6–3.6)	1.7 (0.9–4.0)	0.81	0.03
REM latency, min	120.0 (82.2–167.8)	119.8 (82.8–189.4)	0.91	0.01

Values are median (IQR). *r*, rank-biserial effect size. † Available for *n* = 64 (intervention 33, control 31). AHI, apnea–hypopnea index; SpO₂, peripheral oxygen saturation.

Table 3
Sensitivity ANCOVA models for sleep-onset latency

Model	Group β (min)	95% CI	p	R ²
Crude	–12.7	–19.1 to – 6.2	< 0.001	0.18
Adjusted (time in bed + mean SpO ₂)	–12.0	–18.8 to – 5.1	< 0.001	0.19
Fully adjusted (+ age, BMI, AHI)	–10.7	–18.2 to – 3.2	0.006	0.21

Dependent variable: sleep-onset latency (min); group coded control = 0, intervention = 1. These are sensitivity analyses, not equivalence tests.

Figures

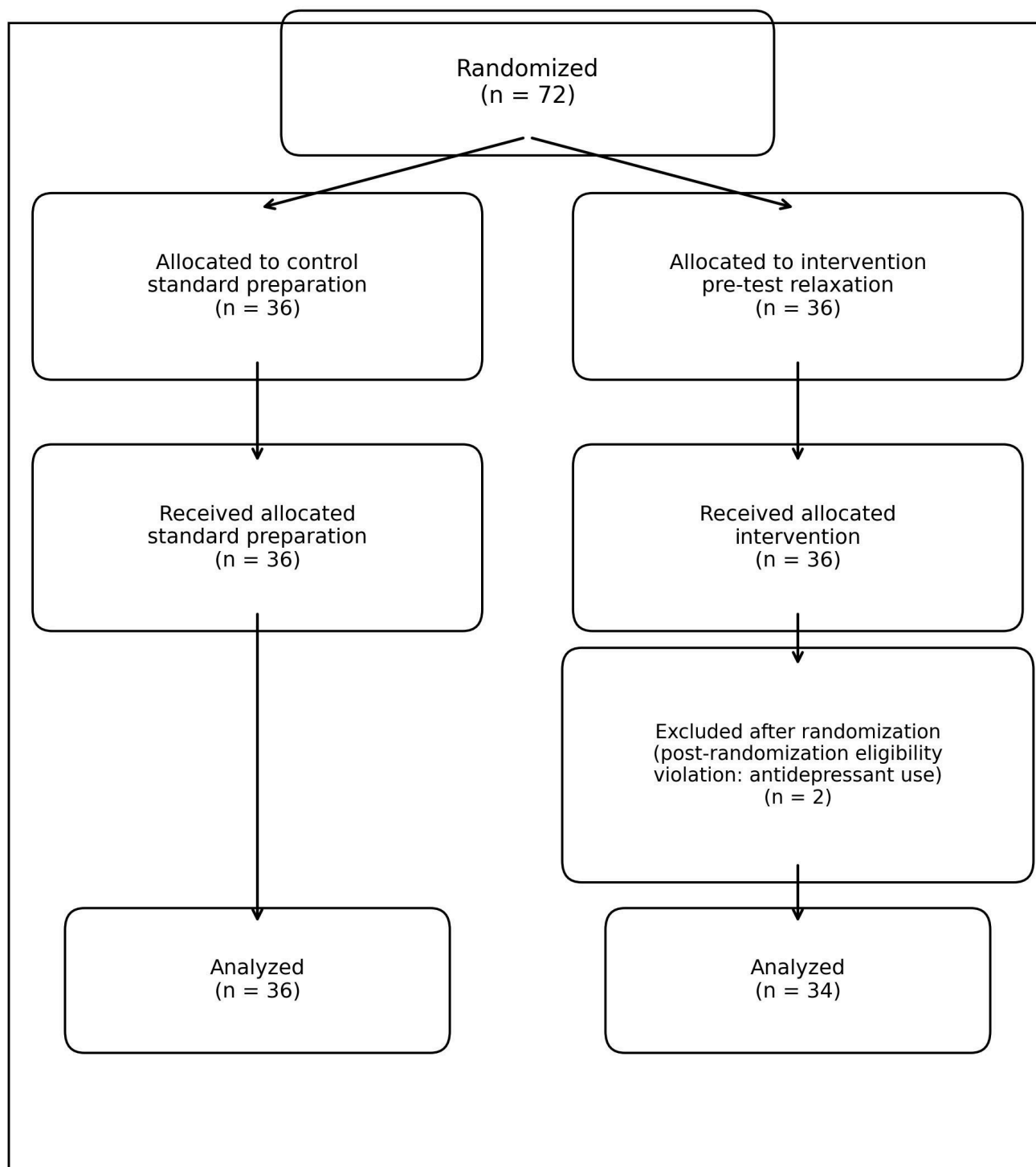


Figure 1

CONSORT participant-flow diagram. Seventy-two participants were randomized. Two participants allocated to the intervention group were subsequently found to be taking antidepressants; PSG outcome data were unavailable, and they were excluded before outcome review. The modified intention-to-treat cohort comprised 70 participants (intervention n = 34; control n = 36). REM-AHI, non-REM AHI, and minimum SpO₂ were available for 64 participants.

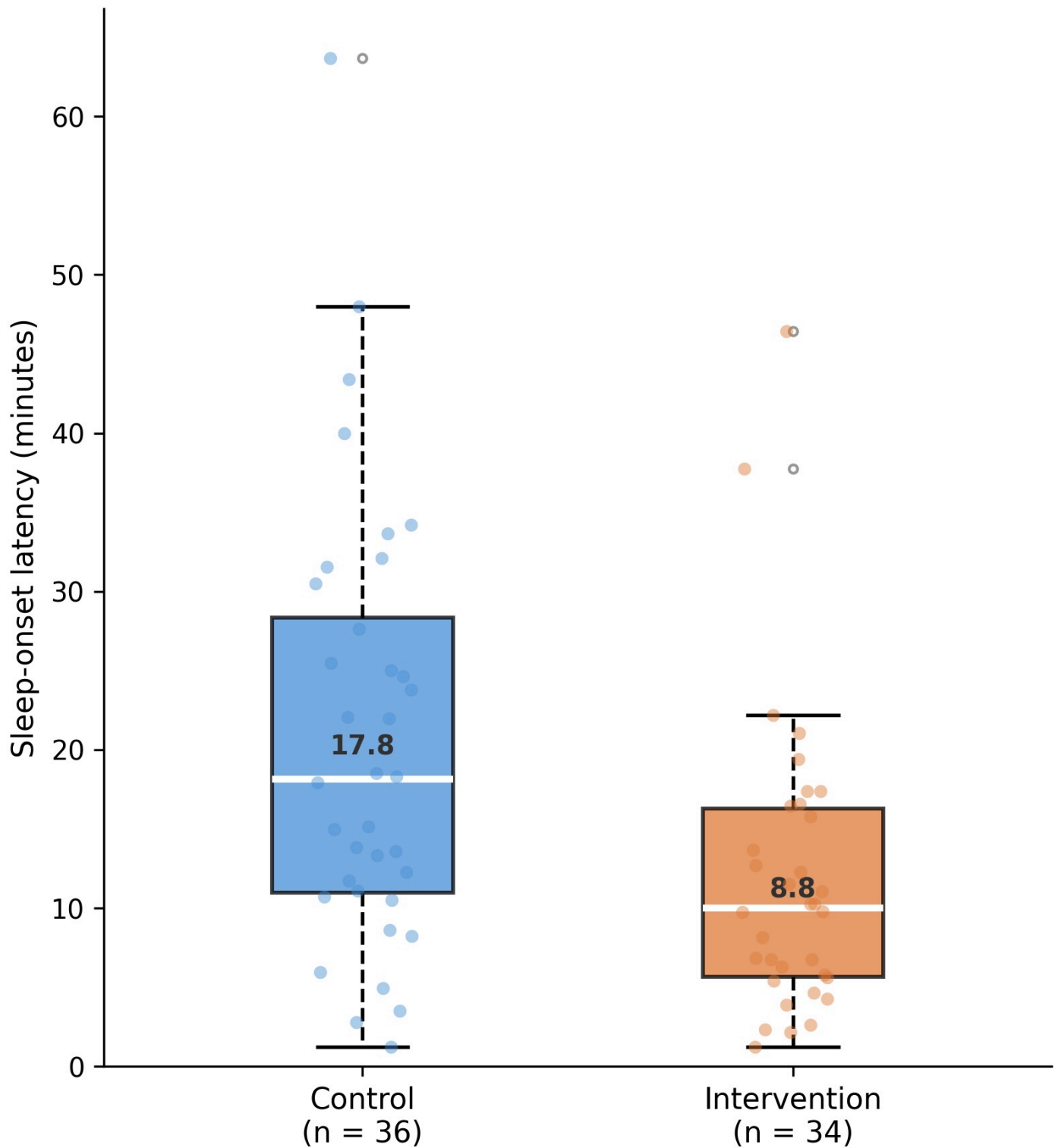


Figure 2

Sleep-onset latency by group. Box plots with individual observations show shorter latency in the intervention group (median 8.8 vs 17.8 min; Mann–Whitney U $p < 0.001$; rank-biserial $r = 0.45$).