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Original Article

Virtual reality combined with biofeedback improves sleep quality and daytime function in patients with chronic insomnia

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Abstract: Objective: To explore the effect of virtual reality (VR) combined with biofeedback on sleep quality and daytime function in patients with chronic insomnia. Methods: Data from 83 patients with chronic insomnia admitted to the sleep department of our hospital were retrospectively analyzed and divided into a control group and an observation group according to the treatment method. The control group received routine biofeedback therapy, and the observation group received VR combined with biofeedback therapy. The Pittsburgh Sleep Quality Index (PSQI), Insomnia Severity Index (ISI), Multidimensional Fatigue Scale (MFI) and Daytime Function Scale (DFSS) were compared between the two groups before and after treatment, and the changes of sleep latency, total sleep time and awakening times in polysomnography (PSG) were analyzed. Results: After the above treatment, the PSQI and ISI scores of the observation group were significantly lower than those of the control group ($P < 0.01$). PSG showed that the sleep latency was shortened, the total sleep time was prolonged, and the number of awakenings was reduced ($P < 0.05$). In addition, in terms of daytime function, the MFI fatigue score of the observation group decreased and the DFSS score increased, and the differences were statistically significant ($P < 0.05$). Finally, after 1 month of follow-up, there was no significant difference in the incidence of adverse reactions between the two groups ($P=0.071$). Conclusion: VR combined with biofeedback can effectively improve the sleep quality of patients with chronic insomnia, reduce nocturnal awakening, and improve daytime functional status, without increasing the incidence of adverse reactions.

Keywords: Virtual reality, biofeedback, chronic insomnia, sleep quality, daytime function

Introduction

Chronic insomnia is a common sleep disorder seen in clinical practice, characterized by prolonged difficulty in falling asleep, sleep maintenance disturbances, or early awakening, often accompanied by significant daytime dysfunction [1, 2]. Recent epidemiological data indicate that approximately 10%-30% of adults worldwide suffer from chronic insomnia. This condition not only significantly increases the risk of mental and psychological disorders such as anxiety and depression but is also closely associated with cardiovascular diseases, metabolic syndrome, and cognitive decline, making it a major public health concern [3, 4].

Currently, the treatment of chronic insomnia primarily relies on pharmacotherapy combined with cognitive behavioral therapy. Although these approaches have achieved certain clinical efficacy, limitations such as slow onset of action, poor patient compliance, unsustainable therapeutic effects, and notable adverse reactions persist, severely affecting patients' treatment expectations and long-term outcomes [5, 6]. Therefore, exploring safe, effective, and acceptable non-pharmacological interventions is of great significance for improving clinical outcomes in patients with chronic insomnia.

In recent years, non-pharmacological techniques such as biofeedback and virtual reality have shown promising potential in the fields of mental health and rehabilitation medicine. Biofeedback can regulate autonomic nervous system function and help patients restore nor-

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mal sleep physiological rhythms. Virtual reality technology, by creating an immersive relaxation environment, alleviates emotional anxiety and promotes physical and mental relaxation, and has gradually been applied in the adjunctive treatment of chronic insomnia [7, 8]. However, most existing studies have focused on the effects of single techniques, with limited research exploring the integration of these approaches on sleep architecture and daytime function in patients with chronic insomnia [9]. To address this research gap, this study is the first to combine virtual reality technology with biofeedback in the clinical intervention of chronic insomnia, with a focus on evaluating their combined effects on sleep quality and daytime function. This study aims to provide new evidence-based support for comprehensive non-pharmacological interventions in chronic insomnia, highlighting its clear innovation and clinical translational value.

General information and methods

General information

Data from 83 patients with chronic insomnia admitted to the sleep department of our hospital from January 2021 to December 2023 were retrospectively analyzed. According to the treatment methods, they were divided into control group and experimental group. Among them, there were 39 cases in the control group (conventional biofeedback therapy) and 44 cases in the observation group (VR combined with biofeedback therapy). This study was approved by the ethics committee of the West China Hospital, Sichuan University (ethics number: KYLL-2024-12-10).

Sample size estimation: Sample size estimation was based on the primary outcome measure (sleep quality score). Referring to previous similar studies and the results of our preliminary experiment, we assumed a mean difference in the Pittsburgh Sleep Quality Index (PSQI) score of 2.5 points between the combined intervention group and the control group, with a pooled standard deviation of 3.0. A two-sided significance level of $\alpha=0.05$ and a statistical power of $1-\beta=0.80$ were set. The sample size was calculated using the formula for comparing two means: $n=(Z_{\alpha/2}+Z_{\beta})^2\times 2\sigma^2/\delta^2$.

The calculation yielded a required sample size of approximately 36 patients per group. Considering a 10% follow-up dropout rate, the final target was set at no less than 40 patients per group. In this study, 44 patients were ultimately included in the observation group and 39 in the control group, which generally met the sample size requirements.

Inclusion criteria

Aged 18-60 years; Chronic insomnia diagnosis in line with the relevant guidelines and standards [10]; Pittsburgh sleep quality index (PSQI) total score ≥ 8 points before treatment; Conscious with normal audiovisual and comprehension abilities; First-time treatment for chronic insomnia; Complete clinical data; Patients were right-handed; Years of education > 9 years.

Exclusion criteria

People with severe mental illness (such as major depression, schizophrenia) or nervous system diseases; Patients with other primary sleep disorders such as sleep apnea syndrome and periodic limb movement disorders; Patients who have been treated with sedative-hypnotic drugs or psychotropic drugs in the past two months; Patients with previous visual or auditory disorders, motion sickness, or other conditions that preclude adaptation to VR equipment; Pregnant or lactating women; Incomplete information.

Data collection

Data on general characteristics, clinical indicators, and scale scores were collected from the electronic medical record system and through standardized scale assessments. This study used a self-developed cognitive training system consisting of three modules (working memory training, biofeedback, and scene modeling) and 22 immersive scenes (e.g., bamboo forest, grassland, garden, beach, and courtyard). Patients were instructed to relax and lie on a training bed while wearing the device and holding a handheld controller, with a therapist standing beside them to guide the session. Each session began with a 10-minute scene selection and communication period, during which the therapist discussed the patient's views on sleep and treatment, explored poten-

tial causes of insomnia, and recorded feedback; subsequently, the selected scene was played, and the therapist guided the patient to correct maladaptive cognitions, encouraged a positive perspective on sleep, and reinforced self-positive suggestions. Patients were also instructed to maintain a daily sleep diary, record sleep and wake times, sleep environment, and bedtime habits, and were advised to create a quiet and comfortable sleep environment while avoiding non-sleep-related activities or electronic device use before bedtime. Biofeedback was delivered using the FreeMind-G biofeedback instrument (Nanjing Weisi Medical Technology Co., Ltd., China) in a dedicated biofeedback therapy room by trained therapists. Interventions were conducted once daily, with each session lasting 30 minutes (including 10 minutes of scene selection and communication and 20 minutes of active intervention combining VR scene exposure with biofeedback), over a treatment course of 2 weeks (14 consecutive days). To ensure comparability, the total intervention time per session was identical between groups: the observation group received 30 minutes of combined VR and biofeedback therapy per session, while the control group received 30 minutes of biofeedback therapy alone per session, with all other procedures and schedules consistent across groups.

Observation indicators

Main observation indexes: The Pittsburgh Sleep Quality Index (PSQI), Insomnia Severity Index (ISI), Multidimensional Fatigue Scale (MFI) and Daytime Function Scale (DFSS) were used to evaluate the sleep status of the two groups before and after treatment [11, 12]. The polysomnography (PSG) monitoring method was used to evaluate the sleep quality of patients, and the American Neurotech polysomnography monitoring system (model: NT-2000) was used for overnight monitoring. Caffeine-containing beverages were prohibited 24 hours before monitoring. EEG, EOG, EMG, ECG, and respiratory sensors in a standard sleep laboratory environment, and recorded sleep parameters throughout the night, including sleep latency, total sleep time, and number of awakenings. Data were analyzed using the supporting software with automatic identification combined with manual review. At the same time, heart rate variability-related indicators were collected.

Secondary observation indicators: Assessment of Anxiety and Depression: Anxiety symptoms were assessed using the Hamilton Anxiety Rating Scale (HAMA), a 14-item scale rated on a 0-4 scale (total score 0-56), with higher scores indicating greater anxiety severity. Depressive symptoms were evaluated using the 17-item Hamilton Depression Rating Scale (HAMD), rated on 0-2 or 0-4 scales (total score 0-54), with higher scores indicating more severe depression. All assessments were conducted by trained psychiatrists in a quiet, independent room at baseline and within 24 hours after the intervention. Assessors were blinded to group allocation to ensure objectivity.

Statistical analysis

SPSS 23.0 statistical software was used for data analysis. Measurement data conforming to normal distribution were expressed as mean ± standard deviation, and count data were expressed as number of cases (percentage). For between-group comparisons, independent sample t-test was used for measurement data and the chi-square test was used for count data. For within-group comparisons at different time points (pre- and post-treatment), paired t-test was employed. A P-value < 0.05 was considered statistically significant. All statistical analyses were reviewed by a professional statistician to ensure methodological appropriateness.

Results

Comparison of baseline data between the two groups of patients

A total of 83 patients with chronic insomnia were included in this study, including 39 in the control group (conventional biofeedback therapy) and 44 in the observation group (VR combined with biofeedback therapy). There was no significant difference in baseline data such as age, sex, height, weight, education level, course of disease, smoking history, drinking history, hypertension history, diabetes history, coronary heart disease and family history of sleep disorders between the two groups (all P > 0.05), indicating that the two groups were comparable (**Table 1**).

Table 1. Comparison of baseline characteristics between the two groups of patients

Item	Control Group (n=39)	Observation Group (n=44)	Statistical Value	*P* Value
Age (years, $\bar{x}\pm s$)	48.6 ± 9.3	50.2 ± 8.7	-0.825	0.415
Gender (Male/Female, n)	20/19	23/21	0.011	0.920
Height (cm, $\bar{x}\pm s$)	165.41 ± 7.25	166.12 ± 6.80	-0.464	0.647
Weight (kg, $\bar{x}\pm s$)	68.50 ± 10.22	70.15 ± 9.86	-0.733	0.468
Education Level (n)			1.345	0.512
High School or Below	16	20		
College/Bachelor's Degree	18	19		
Master's Degree or Above	5	5		
Disease Duration (months, M (P_{25}, P_{75}))	24 (12, 48)	30 (15, 50)	-0.918	0.363
Smoking History (Yes/No, n)	12/27	15/29	0.112	0.740
Drinking History (Yes/No, n)	10/29	13/31	0.190	0.663
History of Hypertension (Yes/No, n)	11/28	14/30	0.157	0.698
History of Diabetes (Yes/No, n)	6/33	8/36	0.124	0.729
Coronary Heart Disease (Yes/No, n)	4/35	5/39	0.022	0.887
Family History of Sleep Disorders (Yes/No, n)	7/32	9/35	0.071	0.791

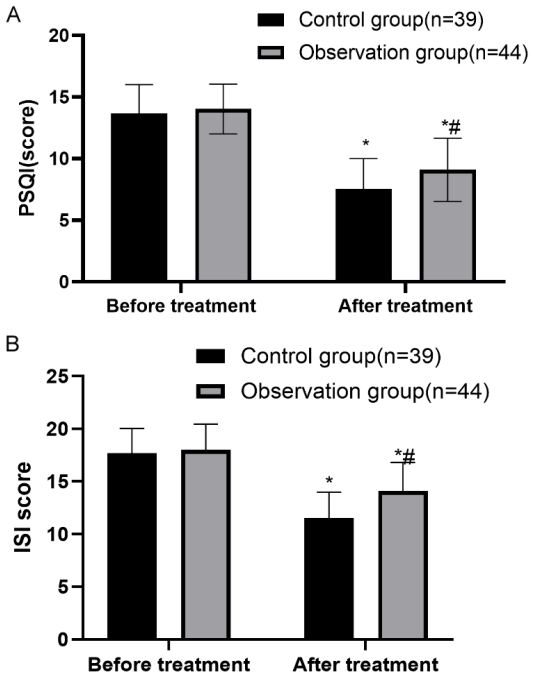

Figure 1. PSQI and ISI scores of the two groups before and after treatment. A. PSQI scores of the two groups before and after treatment; B. ISI scores of the two groups before and after treatment. *: Before treatment compared with after treatment, P < 0.05, #: After treatment, the observation group was compared with the control group, P < 0.05. PSQI, Pittsburgh Sleep Quality Index; ISI, Insomnia Severity Index.

Comparison of PSQI and ISI scores between the two groups before and after treatment

Before treatment, there was no significant difference in PSQI and ISI scores between the two groups (P > 0.05). After treatment, the PSQI score in the control group decreased from 15.32 ± 2.45 to 9.86 ± 2.12 (P < 0.001), and in the observation group decreased from 15.08 ± 2.38 to 6.74 ± 1.95 (P < 0.001). The ISI score in the control group decreased from 18.45 ± 3.21 to 12.38 ± 2.86 (P < 0.001), and in the observation group decreased from 18.12 ± 3.15 to 8.52 ± 2.43 (P < 0.001). The experimental group showed significantly greater reductions in both PSQI and ISI scores compared with the control group (P=0.002 for both). See **Figure 1**.

Fatigue and daytime function improvement in the two groups of patients

Before treatment, there was no significant difference in the scores of multidimensional fatigue scale (MFI) and daytime function scale (DFSS) between the two groups (all P > 0.05), suggesting that the baseline levels were comparable. After treatment, the MFI score in the control group decreased from 68.50 ± 8.33 to 62.11 ± 7.93 (P < 0.05), and in the observation

Table 2. Comparison of MFI and DFSS scores between the two groups before and after treatment

Measurement Indicator	Time Point	Control Group (n=39)	Observation Group (n=44)
MFI Score	Pre-treatment	68.50 ± 8.33	69.22 ± 7.83
	Post-treatment	62.11 ± 7.93*	56.34 ± 6.52*,#
DFSS Score	Pre-treatment	45.20 ± 6.83	44.82 ± 6.53
	Post-treatment	51.62 ± 7.21*	57.44 ± 6.92*,#

Note: *indicates that compared with the group before treatment, P < 0.05; #means compared with the control group after treatment, P < 0.05. MFI, Multidimensional Fatigue Inventory Score; DFSS, Daytime Function Scale.

group decreased from 69.22 ± 7.83 to 56.34 ± 6.52 (P < 0.05). The DFSS score in the control group increased from 45.20 ± 6.83 to 51.62 ± 7.21 (P < 0.05), and in the observation group increased from 44.82 ± 6.53 to 57.44 ± 6.92 (P < 0.05). The experimental group demonstrated significantly greater improvements in both fatigue reduction (MFI) and daytime function enhancement (DFSS) compared with the control group (all P < 0.05) (**Table 2**).

Comparison of autonomic nerve function between the two groups before and after treatment

Before treatment, there were no significant differences in heart rate (HR), heart rate variability time domain indexes (SDNN, RMSSD, PNN50) and frequency domain indexes (LF, HF, LF/HF) between the two groups (all P > 0.05). After treatment, HR in the control group decreased from 78.22 ± 6.50 bpm to 74.12 ± 5.82 bpm (P < 0.05), and in the observation group decreased from 77.81 ± 6.92 bpm to 70.32 ± 5.23 bpm (P < 0.05). SDNN in the control group increased from 32.52 ± 8.24 ms to 36.83 ± 9.12 ms (P < 0.05), and in the observation group increased from 33.13 ± 7.90 ms to 42.54 ± 9.62 ms (P < 0.05). RMSSD in the control group increased from 24.62 ± 7.30 ms to 28.91 ± 8.42 ms (P < 0.05), and in the observation group increased from 25.23 ± 7.11 ms to 35.23 ± 9.03 ms (P < 0.05). PNN50 in the control group increased from 8.54 ± 3.23% to 12.11 ± 4.02% (P < 0.05), and in the observation group increased from 8.92 ± 3.42% to 16.83 ± 4.51% (P < 0.05). LF in the control group increased from 342.45 ± 108.24 ms^2 to 398.56 ± 120.54 ms^2 (P < 0.05), and in the observation group increased from 351.53 ± 112.32 ms^2 to 465.32 ± 128.68 ms^2 (P < 0.05). HF in the control group increased from 256.45 ± 89.90 ms^2 to 312.21 ± 101.45 ms^2 (P < 0.05), and in the observation group increased from 263.45 ± 92.43 ms^2 to 385.12 ± 115.23 ms^2 (P < 0.05). LF/HF ratio in the control group decreased from 1.34 ± 0.42 to 1.28 ± 0.38 (P < 0.05), and in the observation group decreased from 1.33 ± 0.39 to 1.21 ± 0.35 (P < 0.05). The observation group showed significantly greater improvements in all heart rate variability indicators compared with the control group (all P < 0.05) (**Table 3**).

Sleep structure of the two groups before and after treatment

Before treatment, there was no significant difference in sleep structure parameters between the two groups (all P > 0.05). After treatment, total sleep time in the control group increased from 352.00 ± 38.00 min to 382.00 ± 41.00 min (P < 0.05), and in the observation group increased from 348.00 ± 42.00 min to 412.00 ± 45.00 min (P < 0.05). N1 duration in the control group decreased from 88.00 ± 21.00 min to 72.00 ± 18.00 min (P < 0.05), and in the observation group decreased from 92.00 ± 24.00 min to 58.00 ± 16.00 min (P < 0.05). N3 duration in the control group increased from 114.00 ± 22.00 min to 134.00 ± 26.00 min (P < 0.05), and in the observation group increased from 108.00 ± 24.00 min to 156.00 ± 28.00 min (P < 0.05). REM duration in the control group increased from 68.00 ± 16.00 min to 75.00 ± 17.00 min (P < 0.05), and in the observation group increased from 65.00 ± 18.00 min to 86.00 ± 20.00 min (P < 0.05). Sleep efficiency in the control group increased from 78.20 ± 6.50% to 82.50 ± 7.10% (P < 0.05), and in the observation group increased from 77.80 ± 6.90% to 87.30 ± 7.80% (P < 0.05). Arousal Index in the control group decreased from 8.50 ± 3.20 events/hour to 6.20 ± 2.80 events/hour (P < 0.05), and in the observation group decreased from 8.90 ± 3.40

Table 3. Comparison of Heart Rate Variability (HRV) indices between the two groups before and after treatment

Index	Time Point	Control Group (n=39)	Observation Group (n=44)
HR (bpm)	Pre-treatment	78.22 ± 6.50	77.81 ± 6.92
	Post-treatment	74.12 ± 5.82*	70.32 ± 5.23*,#
SDNN (ms)	Pre-treatment	32.52 ± 8.24	33.13 ± 7.90
	Post-treatment	36.83 ± 9.12*	42.54 ± 9.62*,#
RMSSD (ms)	Pre-treatment	24.62 ± 7.30	25.23 ± 7.11
	Post-treatment	28.91 ± 8.42*	35.23 ± 9.03*,#
PNN50 (%)	Pre-treatment	8.54 ± 3.23	8.92 ± 3.42
	Post-treatment	12.11 ± 4.02*	16.83 ± 4.51*,#
LF (ms^2)	Pre-treatment	342.45 ± 108.24	351.53 ± 112.32
	Post-treatment	398.56 ± 120.54*	465.32 ± 128.68*,#
HF (ms^2)	Pre-treatment	256.45 ± 89.90	263.45 ± 92.43
	Post-treatment	312.21 ± 101.45*	385.12 ± 115.23*,#
LF/HF Ratio	Pre-treatment	1.34 ± 0.42	1.33 ± 0.39
	Post-treatment	1.28 ± 0.38*	1.21 ± 0.35*,#

Note: * indicates that compared with the group before treatment, $P < 0.05$; # means compared with the control group after treatment, $P < 0.05$. HR, Heart Rate; SDNN, Standard Deviation of all Normal-to-Normal Intervals; RMSSD, Root Mean Square of Successive Differences between Normal Heartbeats; PNN50, Percentage of Successive Normal-to-Normal Intervals exceeding 50 ms; LF, Low Frequency Power; HF, High Frequency Power; LF/HF Ratio, Ratio of Low Frequency Power to High Frequency Power.

Table 4. Comparison of sleep architecture parameters between the two groups before and after treatment

Parameter	Time Point	Control Group (n=39)	Observation Group (n=44)
Total Sleep Time (min)	Pre-treatment	352.00 ± 38.00	348.00 ± 42.00
	Post-treatment	382.00 ± 41.00*	412.00 ± 45.00*,#
N1 Duration (min)	Pre-treatment	88.00 ± 21.00	92.00 ± 24.00
	Post-treatment	72.00 ± 18.00*	58.00 ± 16.00*,#
N2 Duration (min)	Pre-treatment	150.00 ± 28.00	148.00 ± 30.00
	Post-treatment	176.00 ± 32.00*	198.00 ± 34.00*,#
N3 Duration (min)	Pre-treatment	114.00 ± 22.00	108.00 ± 24.00
	Post-treatment	134.00 ± 26.00*	156.00 ± 28.00*,#
REM Duration (min)	Pre-treatment	68.00 ± 16.00	65.00 ± 18.00
	Post-treatment	75.00 ± 17.00*	86.00 ± 20.00*,#
Sleep Efficiency (%)	Pre-treatment	78.20 ± 6.50	77.80 ± 6.90
	Post-treatment	82.50 ± 7.10*	87.30 ± 7.80*,#
Sleep Onset Latency (min)	Pre-treatment	32.50 ± 8.20	33.10 ± 7.90
	Post-treatment	26.80 ± 7.40*	21.50 ± 6.80*,#
Arousal Index (events/hour)	Pre-treatment	8.50 ± 3.20	8.90 ± 3.40
	Post-treatment	6.20 ± 2.80*	4.10 ± 2.30*,#

Note: *indicates that compared with the group before treatment, $P < 0.05$; #means compared with the control group after treatment, $P < 0.05$.

events/hour to 4.10 ± 2.30 events/hour ($P < 0.05$). The observation group demonstrated significantly greater improvements in all sleep structure parameters compared with the control group (all $P < 0.05$) (**Table 4**).

Comparison of cognitive scale scores between the two groups before and after treatment

Before treatment, there was no significant difference in the total score of MMSE and the

Table 5. Comparison of cognitive function indicators between the two groups before and after treatment

Indicator	Time Point	Control Group (n=39)	Observation Group (n=44)
Total MMSE Score (points)	Pre-treatment	26.32 ± 2.45	26.18 ± 2.37
	Post-treatment	27.15 ± 2.28*	28.76 ± 2.14*,#
Memory Function (AVLT, points)	Pre-treatment	38.65 ± 5.23	38.92 ± 5.41
	Post-treatment	40.82 ± 5.67*	44.73 ± 5.85*,#
Working Memory (DSF, points)	Pre-treatment	8.12 ± 1.45	8.06 ± 1.38
	Post-treatment	8.45 ± 1.37*	9.28 ± 1.42*,#
Working Memory (DSB, points)	Pre-treatment	6.23 ± 1.28	6.17 ± 1.31
	Post-treatment	6.58 ± 1.22*	7.45 ± 1.27*,#
Orientation (Benton, points)	Pre-treatment	23.45 ± 3.12	23.68 ± 3.08
	Post-treatment	24.78 ± 2.95*	26.92 ± 2.87*,#
Attention (SDMT, points)	Pre-treatment	42.36 ± 5.67	42.85 ± 5.73
	Post-treatment	45.12 ± 5.48*	49.76 ± 5.52*,#

Note: *indicates that compared with the group before treatment, $P < 0.05$; #means compared with the control group after treatment, $P < 0.05$. MMSE, Mini-Mental State Examination; AVLT, Auditory Verbal Learning Test; DSF, Digit Span Forward; DSB, Digit Span Backward; SDMT, Symbol Digit Modalities Test.

scores of cognitive dimensions between the two groups (all $P > 0.05$). After treatment, the MMSE total score in the control group increased from 26.32 ± 2.45 to 27.15 ± 2.28 ($P < 0.05$), and in the observation group increased from 26.18 ± 2.37 to 28.76 ± 2.14 ($P < 0.05$). Memory function (AVLT) in the control group increased from 38.65 ± 5.23 to 40.82 ± 5.67 ($P < 0.05$), and in the observation group increased from 38.92 ± 5.41 to 44.73 ± 5.85 ($P < 0.05$). Working memory forward (DSF) in the control group increased from 8.12 ± 1.45 to 8.45 ± 1.37 ($P < 0.05$), and in the observation group increased from 8.06 ± 1.38 to 9.28 ± 1.42 ($P < 0.05$). Working memory backward (DSB) in the control group increased from 6.23 ± 1.28 to 6.58 ± 1.22 ($P < 0.05$), and in the observation group increased from 6.17 ± 1.31 to 7.45 ± 1.27 ($P < 0.05$). Orientation (Benton) in the control group increased from 23.45 ± 3.12 to 24.78 ± 2.95 ($P < 0.05$), and in the observation group increased from 23.68 ± 3.08 to 26.92 ± 2.87 ($P < 0.05$). Attention (SDMT) in the control group increased from 42.36 ± 5.67 to 45.12 ± 5.48 ($P < 0.05$), and in the observation group increased from 42.85 ± 5.73 to 49.76 ± 5.52 ($P < 0.05$). The observation group showed significantly greater improvements in overall cognitive function (MMSE total score) and all cognitive dimensions compared with the control group (all $P < 0.05$) (**Table 5**).

Comparison of emotional related scale scores between the two groups before and after treatment

Before treatment, there was no significant difference in HAMA anxiety score and HAMD depression score between the two groups (all $P > 0.05$). After treatment, the HAMA score in the control group decreased from 18.65 ± 4.32 to 16.23 ± 3.98 ($P < 0.05$), and in the observation group decreased from 18.92 ± 4.15 to 12.45 ± 3.62 ($P < 0.05$). The HAMD score in the control group decreased from 20.14 ± 5.12 to 17.85 ± 4.76 ($P < 0.05$), and in the observation group decreased from 20.37 ± 5.03 to 13.28 ± 4.21 ($P < 0.05$). The observation group showed significantly greater improvements in both anxiety (HAMA) and depression (HAMD) symptoms compared with the control group (all $P < 0.05$) (**Table 6**).

Comparison of the incidence of adverse reactions between the two groups

The incidence of adverse reactions was low in both groups during treatment. Among them, 11 cases (28.21%) were reported in the control group and 14 cases (31.82%) were reported in the observation group. The main manifestations were mild visual discomfort, dizziness/nausea, fatigue and transient emotional fluctuations. All symptoms were transient and did not affect the treatment process. Chi-square

Table 6. Comparison of HAMA and HAMD scores between the two groups before and after treatment (points, $\bar{x} \pm s$)

Indicator	Time Point	Control Group (n=39)	Observation Group (n=44)
HAMA	Pre-treatment	18.65 ± 4.32	18.92 ± 4.15
	Post-treatment	16.23 ± 3.98*	12.45 ± 3.62*,#
HAMD	Pre-treatment	20.14 ± 5.12	20.37 ± 5.03
	Post-treatment	17.85 ± 4.76*	13.28 ± 4.21*,#

Note: HAMA, Hamilton Anxiety Rating Scale; HAMD, Hamilton Depression Rating Scale. Data are presented as mean ± standard deviation. *indicates that compared with the group before treatment, $P < 0.05$; #means compared with the control group after treatment, $P < 0.05$.

Table 7. Comparison of treatment-related adverse events between the two groups [n (%)]

Category of Adverse Event	Control Group (n=39)	Observation Group (n=44)	χ^2 Value	*P* Value
Visual Discomfort	2 (5.13%)	3 (6.82%)	0.104	0.752
Dizziness/Nausea	3 (7.69%)	4 (9.09%)	0.051	0.820
Fatigue	4 (10.26%)	5 (11.36%)	0.026	0.875
Mood Fluctuation	1 (2.56%)	2 (4.55%)	0.214	0.647
Other Discomfort	1 (2.56%)	0 (0.00%)	1.147	0.285
Total	11 (28.21%)	14 (31.82%)	0.138	0.715

test showed no significant difference in the incidence of adverse reactions between the two groups (P=0.715) (**Table 7**).

Discussion

Chronic insomnia is one of the most common sleep disorders found in clinical practice, leading to fatigue, cognitive decline, emotional disturbances, and autonomic nervous system dysfunction, which severely affect patients' quality of life and physical and mental health [13, 14]. Moreover, its prolonged course, high recurrence rate, and suboptimal response to conventional treatments pose significant challenges to public health services [15]. Therefore, improving therapeutic outcomes for chronic insomnia is of great clinical importance.

Current treatments for chronic insomnia include pharmacotherapy, psychological intervention, and biofeedback therapy, which have achieved certain clinical efficacy [16, 17]. Biofeedback therapy, as a non-pharmacological intervention, has been widely used in the treatment of chronic insomnia. By providing real-time feedback of physiological signals such as heart rate and respiration, it helps patients independently regulate their physical and mental state, thereby reducing anxiety at sleep onset and improving sleep initiation and maintenance. Our results showed that both groups demonstrated significant improvements in sleep parameters following treatment, with greater improvements observed in the experimental group that received combined VR and biofeedback intervention. The superior effect of the combined intervention may be attributed to the distinct neurobiological mechanisms of each modality. Biofeedback therapy enhances parasympathetic tone via operant conditioning of autonomic responses, promoting a relaxation response that counteracts hyperarousal - a core feature of chronic insomnia [18]. VR technology, on the other hand, provides an immersive, multisensory environment that engages the brain's default mode network and reduces activity in the amygdala and other stress-related regions, thereby alleviating sleep-related anxiety and facilitating the transition to sleep. The synergistic effect of these two modalities likely results from their complementary actions on both the cognitive-emotional and autonomic components of hyperarousal [19, 20].

Previous studies have shown that patients with chronic insomnia often experience fatigue and daytime dysfunction, which interact to reduce daily activity and quality of life [21]. Our results demonstrated significant reductions in fatigue scores and improvements in daytime function in both groups, with the observation group showing superior outcomes. The enhanced ef-

ficacy of the combined intervention can be understood through the lens of sleep-dependent neuroplasticity. Improved sleep quality - particularly the increase in N3 (slow-wave sleep) and REM sleep observed in the observation group - facilitates glymphatic clearance of metabolic waste products such as beta-amyloid and tau proteins, which accumulate during wakefulness and contribute to neuronal fatigue. Additionally, REM sleep plays a critical role in emotional memory processing and restoration of prefrontal cortical function. The greater increase in REM duration in the observation group (from 65.0 ± 18.0 min to 86.0 ± 20.0 min) may underlie the more pronounced improvements in daytime function and emotional regulation [22-24].

Autonomic nervous system dysfunction is a common comorbidity in chronic insomnia and a key factor in its persistence. Heart rate variability (HRV) is a sensitive indicator of autonomic function [25]. Our results showed that both interventions significantly reduced heart rate and improved HRV parameters, with the observation group demonstrating superior autonomic regulation. The underlying mechanisms involve central autonomic network modulation. Biofeedback therapy operates via the insula and anterior cingulate cortex, regions involved in interoceptive awareness and autonomic control, promoting vagal tone through real-time feedback learning [26]. VR technology adds value by activating the ventromedial prefrontal cortex and attenuating amygdala reactivity, thereby reducing sympathetic outflow. The combined approach may synergistically engage both top-down (cognitive) and bottom-up (sensory) pathways to restore the sympathetic-vagal balance, which is typically shifted toward sympathetic dominance in chronic insomnia [27, 28].

Abnormal sleep architecture is a core pathological feature of chronic insomnia, characterized by reduced total sleep time, decreased stable sleep (N2/N3), increased unstable sleep (N1), abnormal REM sleep proportion, reduced sleep efficiency, and increased nocturnal awakenings [29]. Our results showed that both interventions significantly improved sleep architecture parameters, with the observation group showing greater improvements in total sleep time, N2 and N3 duration, REM duration, sleep efficiency, and arousal index. The neurobiological basis for these improvements likely involves the restoration of thalamocortical and hippocampal-neocortical networks. Slow-wave sleep (N3) is generated by synchronized thalamocortical oscillations, which are disrupted in chronic insomnia due to hyperarousal. Biofeedback therapy may facilitate the reinstatement of these oscillations by reducing sympathetic tone, while VR technology may enhance the consolidation of sleep architecture through its effects on the hypothalamic-pituitary-adrenal (HPA) axis. Specifically, VR-induced relaxation reduces cortisol secretion, a key inhibitor of slow-wave sleep, thereby allowing for deeper and more stable sleep [30-32].

Chronic insomnia is associated with reduced cerebral blood flow and oxygen supply, leading to cognitive impairments in memory, working memory, orientation, and attention [33]. Our results showed significant improvements in MMSE total and subdomain scores in both groups, with greater improvements in the observation group. The cognitive benefits of the combined intervention may be mediated by neuroplastic changes in the hippocampus and prefrontal cortex. Sleep, particularly N3 and REM sleep, is essential for synaptic homeostasis and memory consolidation. The experimental group's greater increase in N3 and REM duration likely enhanced hippocampal-neocortical dialogue, facilitating the transfer of information from short-term to long-term memory. Furthermore, VR-based cognitive training may directly stimulate neuroplasticity by engaging attentional networks and promoting dopamine release in prefrontal regions, thereby complementing the restorative effects of improved sleep [34].

Intervention safety is a critical consideration in clinical practice. Our results showed low and comparable incidence of adverse reactions between groups, indicating that the addition of VR technology does not increase safety risks, consistent with previous reports [35].

This study has several limitations. First, as a single-center retrospective study, selection and information biases may be present. Second, the sample size is relatively small, and stratified analyses based on disease duration or age were not performed, which may affect the precision of the findings. Third, long-term follow-up

was not conducted, limiting our ability to assess the durability of treatment effects and recurrence rates. Future prospective, multicenter studies with larger sample sizes and extended follow-up periods are needed to validate these findings and explore the long-term trajectory of sleep quality, cognitive function, and emotional outcomes.

In conclusion, both VR combined with biofeedback and conventional biofeedback alone effectively improve sleep quality, sleep architecture, cognitive function, and autonomic nervous system function in patients with chronic insomnia, reducing insomnia severity, fatigue, anxiety, and depression while enhancing daytime function, with favorable safety profiles. Notably, the combined VR and biofeedback intervention demonstrated superior efficacy, offering a more comprehensive and effective approach to managing chronic insomnia. The enhanced effects are likely mediated by synergistic modulation of the autonomic nervous system, restoration of sleep-dependent neuroplasticity, and reduction of HPA axis hyperactivity.

Disclosure of conflict of interest

None.

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