



# OPEN Sleep architecture characteristics in patients with acute ischemic stroke

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To investigate the changes in sleep architecture in patients with acute ischemic stroke (AIS) accompanied by sleep-disordered breathing (SDB), providing a basis for clinical treatment strategies. 1367 patients with acute ischemic stroke within 48 h of onset who were hospitalized in the Department of Neurology of Kailuan General Hospital from November 2020 to December 2022 were selected. Among them, 963 cases were male and 404 cases were female, age: 33–92 ( $66.12 \pm 10.62$ ) years old. From the start of hospitalization, patients were monitored for 5 consecutive days using the intelligent mattress type sleep monitoring platform system (IMTSMPS). From day 1 to day 5, there was a difference in non-rapid eye movement 3 (NREM3) sleep (min) day two, and day five compared to day one in the ACl without SDB group ( $p = 0.019$ ), and rapid eye movement (REM) sleep (min) day three compared to day five in the ACl with SDB group ( $p = 0.001$ ). TST (min), SL (min), WASO (min), TOB (min), NOA (min), HRV1, fluctuated between 5 d in mild SDB group ( $p < 0.05$ ). SL (min), TOB (min), NOA (min), and HAV1(%) in the moderate SDB group, with fluctuations between 5 d ( $P < 0.05$ ). TST (min), SL (min), REML(min), NREM3 (min), REM (min), proportion of REM sleep(%), TOB (min), NOA (min) in severe SDB group, there were fluctuations between 5 d ( $P < 0.05$ ). SDB is one of the most common concomitant symptoms in AIS patients and is closely associated with multiple forms of altered sleep structure. AIS patients without SDB mainly showed changes in NREM3 sleep structure. AIS and SDB patients mainly showed changes in the structure of REM sleep. Different levels of SDB in AIS patients lead to different forms of structural changes in sleep.

**Keywords** Acute ischemic stroke, Acute phase, Sleep-disordered breathing, Sleep architecture, Variability

Numerous studies have shown that acute ischemic stroke (AIS) is often accompanied by sleep-disordered breathing (SDB) and changes in sleep architecture<sup>1–3</sup>. SDB is also a significant risk factor for ischemic stroke<sup>4</sup>, and it is closely related to other risk factors for ischemic stroke, such as hypertension, atherosclerosis, and hyperlipidemia<sup>5–7</sup>. Additionally, research indicates that changes in sleep architecture are associated with the severity and prognosis of post-stroke conditions<sup>8–12</sup>. Due to the limitations of sleep monitoring methods, the small sample sizes, and the short monitoring durations in most studies, there are significant discrepancies among the results. Therefore, we used a novel non-contact mattress-type sleep monitoring system to perform large-sample, long-term, dynamic, continuous monitoring of the sleep states of AIS patients. This study aims to identify the characteristics of SDB and sleep architecture changes in AIS patients. SIMACPS is a fast, simple and effective technology platform for clinical screening and diagnosis, as it is accurate in sleep structure and has high sensitivity and specificity in the diagnosis of sleep-related diseases such as sleep maintenance difficulties and OSAHS.

## Subjects and methods

### Research subjects

This study selected AIS patients who were hospitalized in the Department of Neurology at Kailuan General Hospital from November 2020 to December 2022. The study included 963 male and 404 female patients, aged 33 to 92 years ( $66.12 \pm 10.62$  years), with a BMI of  $27.32 \pm 4.39$  kg/m<sup>2</sup>. Throughout the entire hospitalization period, from admission to discharge, all eligible patients' sleep states were continuously and dynamically monitored using the non-contact mattress-type sleep monitoring system (intelligent mattress type sleep monitoring platform system, IMTSMPS) SC-500 (manufactured by Nanjing Bochuang Haiyun Electronic Technology Co., Ltd., China)<sup>13–19</sup>. Inclusion criteria: ① Age  $\geq 18$  years; ② Onset time within 48 h, with MRI-confirmed AIS; ③ Continuous sleep monitoring  $\geq 5$  days with valid data for at least 5 h/day. Exclusion criteria: ① Comatose

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patients;② Patients with hemorrhagic stroke;③ Recent use of central nervous system stimulants, anxiolytics/antidepressants, antiepileptic drugs, or sedatives and hypnotics;④ History of mental disorders;⑤ Severe cognitive impairment who are unable to cooperate with the examination;⑥ History of neuromuscular junction disorders, muscle diseases, other neurological disorders, or neurodegenerative diseases;⑦ Presence of malignant tumors;⑧ Presence of other severe physical diseases unable to cooperate with examinations;⑨ Patients who received thrombolytic therapy or endovascular treatment.

## Research methods

### Baseline data collection

Detailed records were kept of the patients' age, gender, smoking history, alcohol consumption history, habitual snoring history, past medical history (hypertension, diabetes, hyperlipidemia, hyperhomocysteinemia, stroke), and clinical characteristics at the time of admission for the current episode, especially the NIHSS score to assess stroke severity.

### Localization diagnosis of cerebral infarction

The lesion location of cerebral infarction was determined by neuroimaging and neurology specialists based on the patients' MRI brain scans and clinical manifestations. The infarction sites were categorized into cerebral hemispheres, thalamus, brainstem, cerebellum, and multiple cerebral infarctions.

### Sleep state monitoring indicators

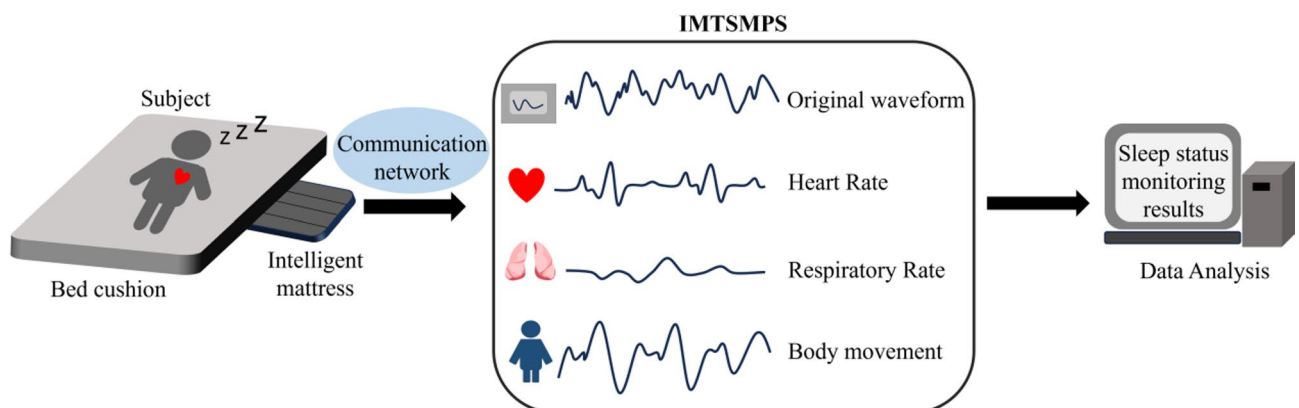
After admission and bed placement, continuous dynamic sleep architecture physiological indicators were monitored in a natural state using the SC-500 IMTSMPS system in a non-contact, non-interfering manner. Continuous sleep monitoring  $\geq 5$  days with valid data for at least 5 h/day. The results were displayed in real-time on the visualization system at the Neurobiology Laboratory Control Center and stored for data analysis. Monitoring indicators included total sleep time (TST, min), sleep latency (SL, min), rapid eye movement latency (REML, min), non-rapid eye movement 1–2 (NREM1–2, min) (light sleep), non-rapid eye movement 3 (NREM3, min) (deep sleep), rapid eye movement (REM, min), sleep efficiency (SE, min), number of awakenings (NoA, time), wake after sleep onset (WASO, min), time out of bed (TOB, min), proportion of non-rapid eye movement (%), proportion of REM (%), apnea-hypopnea index (AHI), respiratory variability index (RVI), and heart rate variability index (HRVI). Figures 1 and 2 show the schematic diagrams of the monitoring equipment.

### Diagnostic criteria for SDB

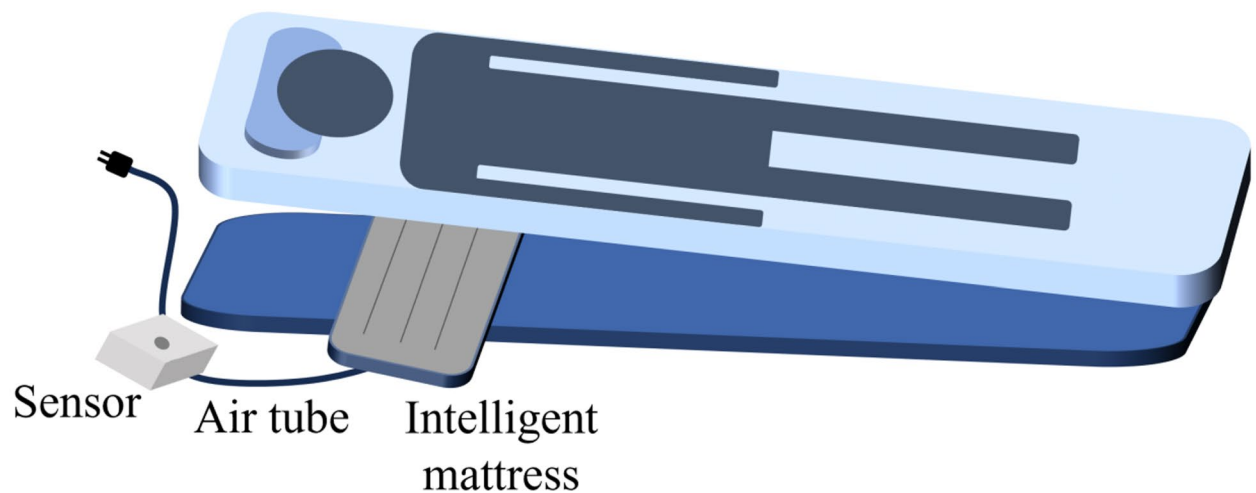
The diagnosis was based on the criteria from the “International Classification of Sleep Disorders, Third Edition,” published by the American Academy of Sleep Medicine (AASM)<sup>20,21</sup>. During the 5-day monitoring period, having an apnea-hypopnea index (AHI)  $\geq 5$  events/hour on at least one day was diagnosed as SDB<sup>22</sup>. Based on daily monitoring, the groups were classified as follows: non-SDB group (normal group) with AHI  $< 5$  events/hour; mild SDB group with  $5 \text{ events/hour} \leq \text{AHI} < 15 \text{ events/hour}$ ; moderate SDB group with  $15 \text{ events/hour} \leq \text{AHI} < 30 \text{ events/hour}$ ; and severe SDB group with  $\text{AHI} \geq 30 \text{ events/hour}$ . Patients with AHI fluctuating between two or more levels were classified as the SDB fluctuation group.

### Statistical methods

Statistical analysis was performed using SPSS 26.0 software. Measurement data were expressed as mean  $\pm$  standard deviation, and comparisons between two groups were conducted using the independent sample *T*-test. A one-way repeated measures ANOVA was used to analyze the differences in sleep architecture from day 1 to day 5 among AIS patients in the different degrees of SDB groups and the non-SDB group, calibrated with Bonferroni. A *P*-value of less than 0.05 was considered statistically significant.



**Fig. 1.** Schematic diagram of the working principle of the sleep monitor.



**Fig. 2.** Schematic diagram of the placement of the sleep monitor's intelligent air mattress.

## Results

### Demographical and clinical outcomes

A total of 1367 patients were included in the study. There were 963 male and 404 female cases. Age:  $33\text{--}92$  ( $66.12 \pm 10.62$ ) years; BMI:  $27.32 \pm 4.39$  kg/m<sup>2</sup>. Among them, 594 patients (43.45%) were smokers, 486 patients (35.55%) consumed alcohol, 918 patients (67.15%) had hypertension, 477 patients (34.89%) had diabetes, 602 patients (44.04%) had hyperhomocysteinemia, 345 patients (25.24%) had hyperlipidemia, and 158 patients (11.56%) had a history of habitual snoring. The NIHSS score at admission was  $14.58 \pm 8.47$ . The cerebral infarction lesions were located in the cerebral hemispheres in 423 cases (30.94%), thalamus in 302 cases (22.09%), brainstem in 323 cases (23.63%), cerebellum in 58 cases (4.24%), and multiple brain regions in 261 cases (19.09%).

### Continuous monitoring results of AHI in AIS patients

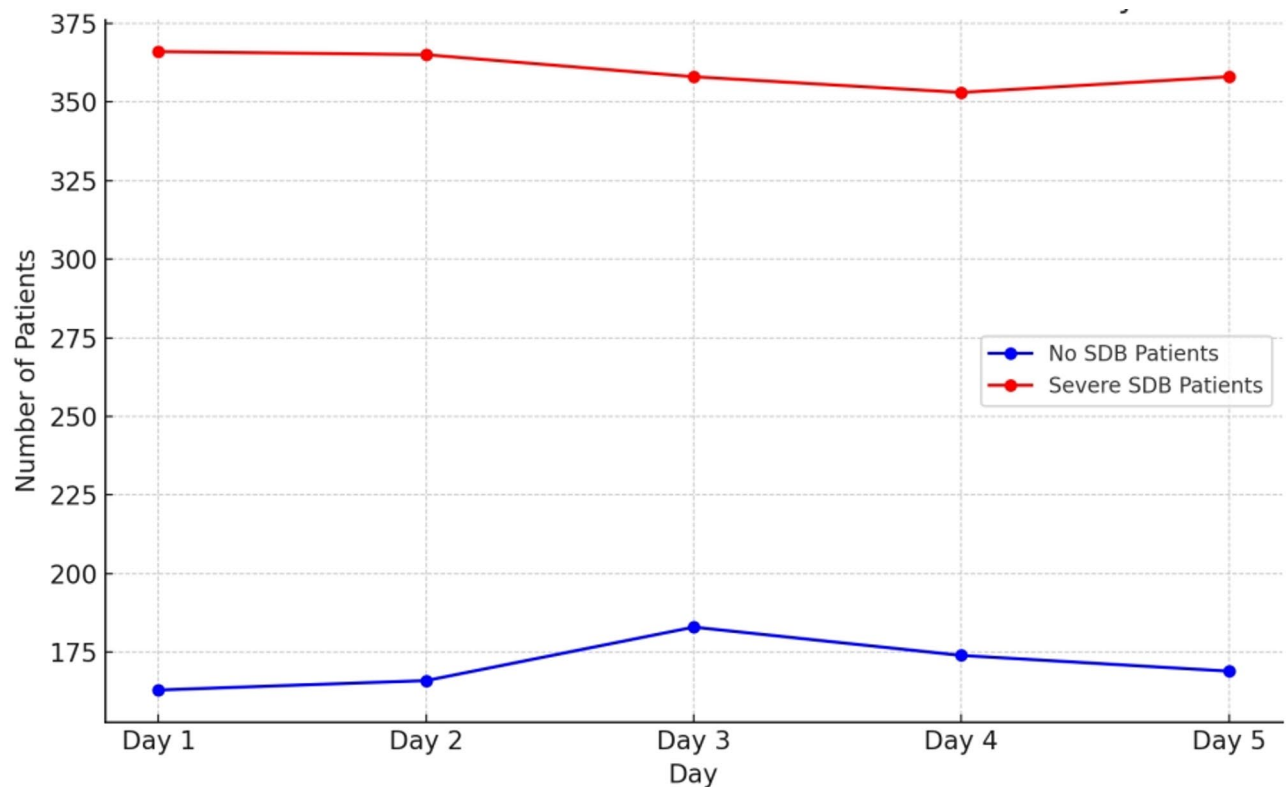
During the 5-day period of onset, the AHI of 1367 AIS patients was  $22.88 \pm 0.19$ . The AHI on days 1 to 5 of onset were  $23.2 \pm 16.11$ ,  $22.89 \pm 16.16$ ,  $22.79 \pm 15.6$ ,  $22.76 \pm 16.55$ , and  $22.74 \pm 16.15$ , respectively ( $F = 0.186$ ,  $P = 0.946$ ). Among them, 147 patients (10.75%) were in the non-SDB group, and 1220 patients (89.25%) were in the SDB group. In the SDB group, 282 patients (23.11%) had AHI fluctuations across more than two levels during the 5-day period, with 109 cases (38.65%) fluctuating between normal and abnormal states. Among the fluctuation group, 152 cases (53.90%) fluctuated between 2 levels, 120 cases (42.55%) between 3 levels, and 10 cases (3.55%) between 4 levels. The AHI of the SDB fluctuation group on days 1 to 5 of onset were  $25.98 \pm 17.79$ ,  $23.10 \pm 13.89$ ,  $23.52 \pm 15.18$ ,  $20.76 \pm 14.01$ , and  $16.11 \pm 12.73$ , respectively ( $F = 29.281$ ,  $P = 0.001$ ). refer to Figs. 3, 4, 5 and 6.

### Continuous monitoring results of sleep architecture in AIS patients with SDB

In the non-SDB group of ACI patients, TST, SL, REML, SE, NREM3, proportion of NREM sleep, proportion of REM sleep, TOB, NOA, HRV1 were significantly different during the 5-day period of the onset of the disease ( $P < 0.01$ ), and there were no statistically significant differences ( $P > 0.05$ ) in NREM1-2, REM, WASO, NOA, and RVI (Table 1). The SDB group of ACI Among the patients, SL, REML, REM, WASO, TOB, NOA were significantly different during the 5-day period of onset ( $P < 0.01$ ), and there were no statistically significant differences in TST, SE, NREM1-2, NREM3, proportion of NREM sleep, proportion of REM sleep, RVI, and HRV1 ( $P > 0.05$ ) (Table 2).

### Continuous monitoring results of sleep architecture in AIS patients with different degrees of SDB

Among the ACI patients in the SDB mild group, SL, NREM1-2, NREM3, REM, WASO, TOB, and NOA were significantly different during the 5-day period of the onset of the disease ( $P < 0.01$ ), and there were no statistically significant differences between TST, REML, SE, proportion of NREM sleep, proportion of REM sleep, RVI, and HRV1 ( $P > 0.05$ ) (Table 3). Among the ACI patients in the SDB moderate group In them, SL, TOB, NOA, and HRV1 were significantly different during the 5-day period of onset ( $P < 0.01$ ), and there were no statistically significant differences in TST, REML, SE, NREM1-2, NREM3, REM, proportion of NREM sleep, proportion of REM sleep, WASO, and RVI ( $P > 0.05$ ) (Table 4). In the patients with ACI in the severe group of SDB, TST, SL, REML, NREM3, REM, proportion of REM sleep, TOB, and NOA were significantly different during the 5-day period of the onset of the disease ( $P < 0.01$ ), and there were no statistically significant differences in SE, NREM1-2, proportion of NREM sleep, WASO, RVI, and HRV1 ( $P > 0.05$ ) (Table 5).

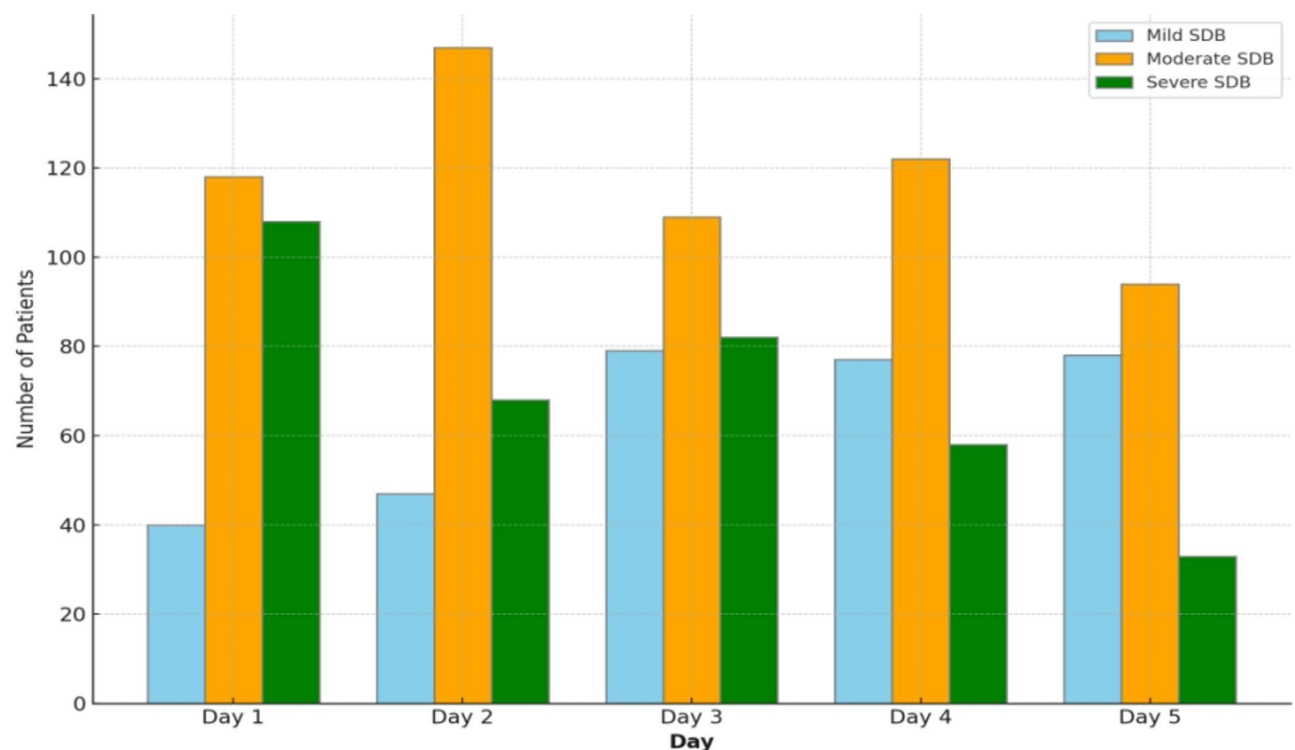


**Fig. 3.** Trend of non-SDB and severe SDB patients over 5 days. This time series chart illustrates the fluctuation in the number of patients in the non-SDB group compared to those in the severe SDB group over a period of 5 days. From the chart, it is evident that the number of patients in the non-SDB group exhibits slight fluctuations as the days progress, whereas the number of patients in the severe SDB group generally shows a slight decline and tends to stabilize. This trend of change helps us understand the evolution of SDB status in patients with ACI over time.

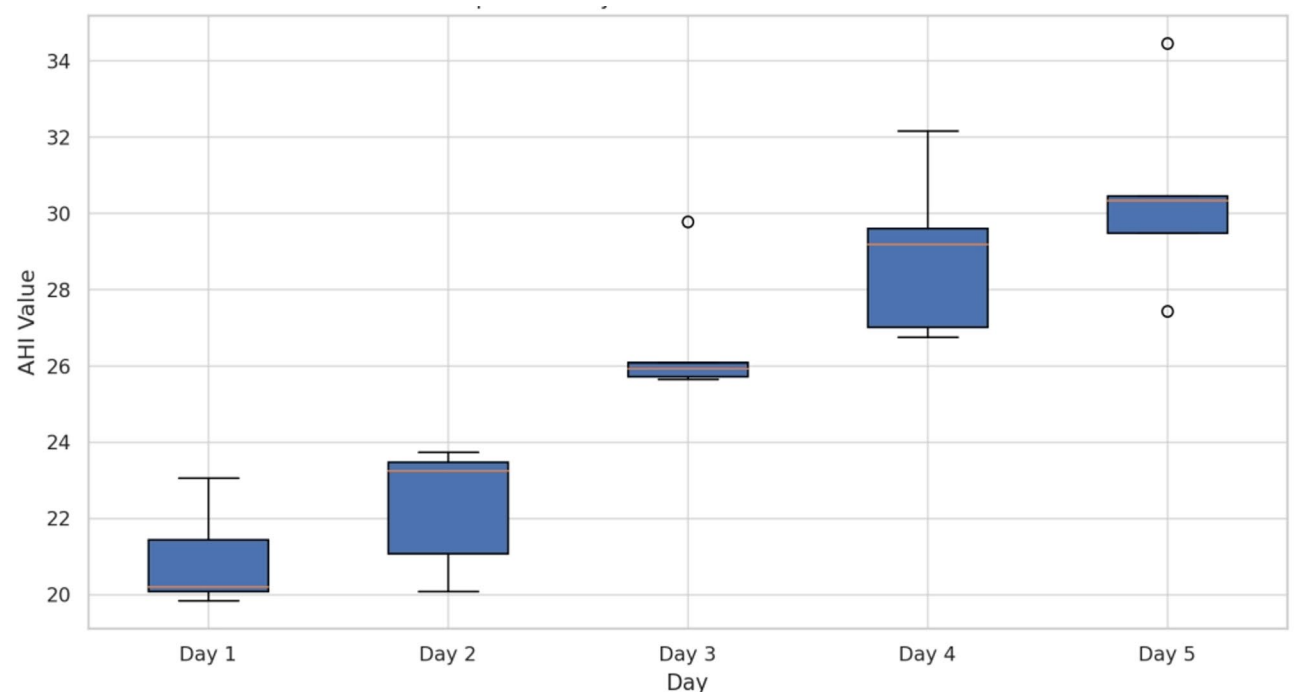
## Discussion

Currently, research on the relationship between stroke and sleep, especially the continuous changes in sleep states after stroke, is limited due to technical constraints. Studies on the changes in sleep architecture in AIS patients with SDB are also relatively scarce. As a result, despite the widespread presence of SDB in AIS patients, its use as an intervenable risk factor for stroke is limited in clinical prevention and treatment. This study used a non-contact mattress-type sleep monitoring system to continuously monitor the sleep states of AIS patients during the first 5 days of onset. The results showed that the incidence of SDB in AIS patients was as high as 89.25%. Among them, 23.11% of patients in the SDB group fluctuated between more than two levels of severity. Furthermore, AIS patients without SDB mainly exhibited fluctuations in NREM3 sleep architecture, while AIS patients with SDB primarily showed fluctuations in REM sleep architecture. Similarly, AIS patients with different degrees of SDB experienced different forms of sleep architecture changes. Therefore, continuous dynamic monitoring of sleep states in AIS patients is of significant clinical importance.

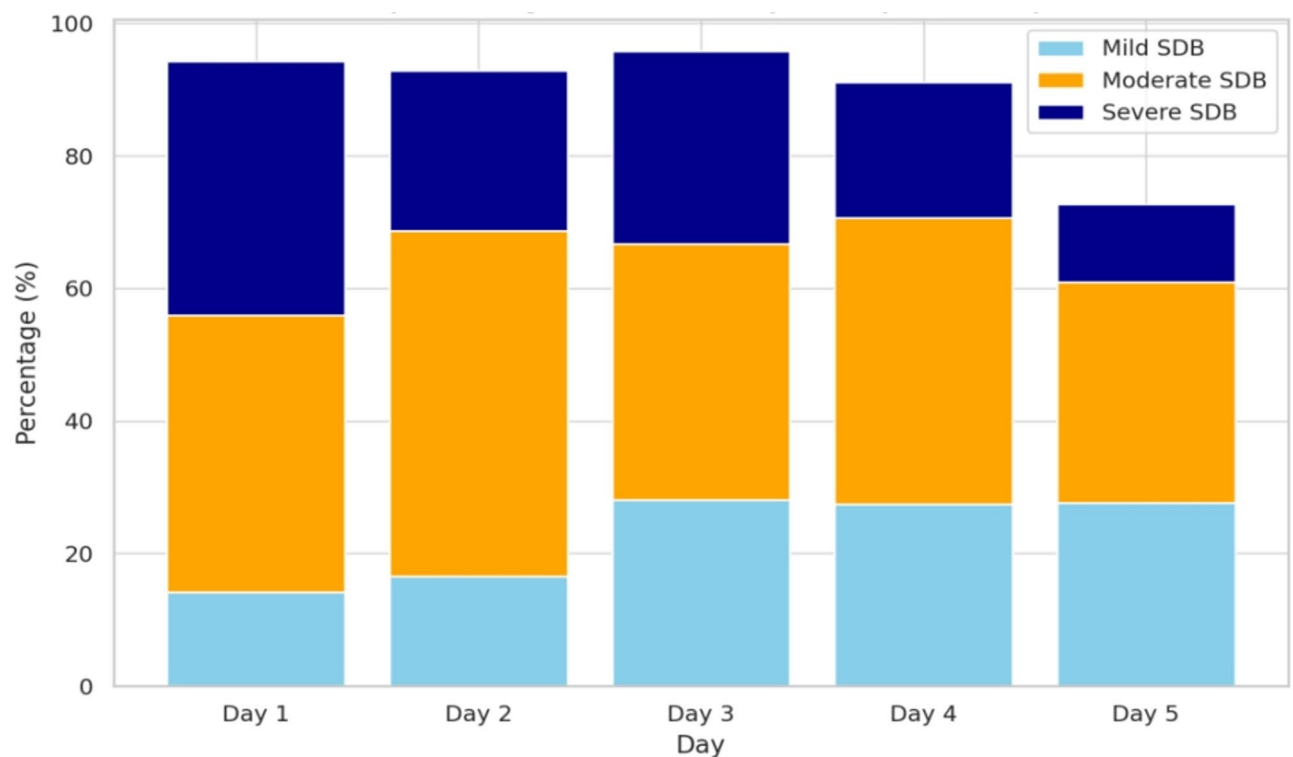
Previous research has shown that SDB is an independent risk factor for ischemic stroke<sup>4,23,24</sup>. Previous studies of poststroke apnea have focused on sleep status outcomes on a single day after stroke, and no studies have been constructed to report on continuous monitoring of sleep apnea over a period of time during the acute poststroke period. However, most studies have small sample sizes, monitor sleep only during one night, and the onset time of AIS at the start of sleep monitoring varies, leading to significant differences in reported incidence and sleep architecture changes in AIS patients with SDB<sup>25,26</sup>. This study conducted continuous sleep monitoring for 5 days during the early onset of AIS. The results showed that both the average AHI over 5 days and the daily AHI were at moderate SDB levels in this population. Among them, 38.65% of patients' AHI fluctuated between normal and different levels of SDB. This may be the main reason for the varying incidence rates of AIS with SDB reported in different studies. Furthermore, the results of this study showed that in the fluctuation group, the daily average AHI from day 1 to day 5 was at a severe SDB level only on the first day, while it remained at a moderate level on the other 4 days. The average AHI was higher on days 1–3 and lower on days 4–5. The actual monitoring results showed that 53.90% of patients fluctuated between 2 levels, 42.55% between 3 levels, and 3.55% between 4 levels. Currently, regarding the present study, it was shown that ischemic stroke patients with significant AHI variability were as high as 86.89%, which suggests that having significant fluctuations in apnea-sleep apnea is a major abnormality during the acute post-stroke period. There are no reports of constructing continuous nightly sleep monitoring studies in the general population. This suggests that the severity of SDB in patients with AIS



**Fig. 4.** Daily occurrence rates of SDB severity over 5 days. This bar chart displays the daily incidence rates of patients in the mild, moderate, and severe SDB groups over 5 days. The mild group is represented in sky blue, the moderate group in orange, and the severe group in green. From the chart, it is apparent that patients in the moderate SDB group constitute the highest proportion on most days, while the proportions of patients in the mild and severe SDB groups fluctuate across different days. Notably, the proportion of patients in the moderate SDB group reaches its peak on the second day of the monitoring period.



**Fig. 5.** Updated daily AHI values for SDB patients. This boxplot shows the daily variation and central tendency in AHI values among SDB patients, as well as the severity of their condition. Notably, the data for the third day highlight a significant change in the median and quartile values of AHI compared to other days.



**Fig. 6.** Daily percentages of SDB cases by severity over 5 days. This stacked bar chart shows the daily incidence percentages of patients in the mild, moderate, and severe SDB groups over a period of 5 days. Different colors represent the various degrees of SDB: mild (sky blue), moderate (orange), and severe (dark blue). The chart displays the proportion of each SDB severity level for each day, stacking them to illustrate the total SDB percentage. Notably, there is an increase in the SDB incidence rate on the third day.

Group/Parameter	Day 1 (n = 163)	Day 2 (n = 166)	Day 3 (n = 183)	Day 4 (n = 174)	Day 5 (n = 169)	F value	P value
TST (min)	521.59 ± 92.02	504.05 ± 103.73 <sup>a</sup>	517.52 ± 95.54	506.31 ± 100.15 <sup>a</sup>	519.5 ± 98.33b <sup>d</sup>	3.138	0.014
SL (min)	22.30 ± 7.46	21.71 ± 6.92	17.68 ± 5.86 <sup>a</sup>	21.18 ± 7.37 <sup>c</sup>	17.74 ± 5.44 <sup>ad</sup>	21.506	0.001
REML (min)	84.24 ± 19.96	84.96 ± 20.60	76.14 ± 21.53 <sup>ab</sup>	84.17 ± 22.80 <sup>c</sup>	81.47 ± 19.50	5.030	0.001
SE (%)	81 ± 11	83 ± 9	84 ± 6 <sup>a</sup>	84 ± 7 <sup>a</sup>	84 ± 8 <sup>a</sup>	5.546	0.001
NREM1 ~ 2 (min)	305 ± 89.37	301.42 ± 89.57	312.17 ± 85.18	302.04 ± 89.17	302.52 ± 93.35	0.694	0.596
NREM3 (min)	57.18 ± 18.87	63.02 ± 20.93 <sup>a</sup>	61.47 ± 21.04	62.32 ± 21.26 <sup>a</sup>	60.21 ± 21.03	2.981	0.019
REM (min)	112.24 ± 32.38	112.51 ± 32.82	114.15 ± 31.56	109.90 ± 34.35	110.03 ± 34.16	0.886	0.472
NREM (%)	70 ± 16	74 ± 15 <sup>a</sup>	72 ± 15	72 ± 14	71 ± 16	2.552	0.038
REM (%)	24 ± 8	24 ± 7	26 ± 9	23 ± 5	22 ± 6 <sup>c</sup>	7.051	0.001
WASO (min)	56.42 ± 18.45	54.92 ± 18.60	58.71 ± 17.89	54.19 ± 19.00	58.25 ± 19.08	2.347	0.053
TOB (min)	31.04 ± 10.8	29.63 ± 9.32	35.65 ± 10.21 <sup>b</sup>	34.96 ± 10.70 <sup>b</sup>	35.69 ± 10.27 <sup>b</sup>	13.684	0.001
NOA (times/h)	13.88 ± 3.09	14.70 ± 3.81	14.94 ± 3.96	14.57 ± 4.07	14.89 ± 3.90	2.233	0.064
RVI	1.27 ± 0.38	1.26 ± 0.40	1.26 ± 0.44	1.25 ± 0.42	1.27 ± 0.37	0.231	0.921
HRVI	5.76 ± 1.11	5.86 ± 1.14	5.77 ± 1.11	5.95 ± 1.11 <sup>a</sup>	6.17 ± 1.02 <sup>a</sup>	5.976	0.001

**Table 1.** One-way repeated measures ANOVA results of sleep architecture indicators in AIS patients of the non-SDB group from day 1 to day 5. <sup>a</sup>Indicates comparison with the normal group,  $P < 0.05$ . <sup>b</sup>Indicates comparison with the mild group,  $P < 0.05$ . <sup>c</sup>Indicates comparison with the moderate group,  $P < 0.05$ . <sup>d</sup>Indicates comparison with the severe group,  $P < 0.05$ .

during the acute phase is highly variable. Since the treatment methods differ for patients with different SDB severity, it is clinically necessary to conduct routine continuous sleep monitoring for AIS patients with SDB during the first 1–3 days of onset to guide the development of individualized treatment plans for AIS patients. This suggests that oxygen therapy in the acute stage of acute ischemic stroke patients with SDB may be of great significance in improving the prognosis of the patients. In future studies, sleep monitoring should be further conducted for a longer period after the onset of acute ischemic stroke to guide the formulation of treatment plans

Group/Parameter	Day 1 (n = 1204)	Day 2 (n = 1201)	Day 3 (n = 1184)	Day 4 (n = 1193)	Day 5 (n = 1198)	F value	P value
TST (min)	507.82 ± 102.24	509.87 ± 104.59	513.98 ± 103.12	508.21 ± 102.02	507.82 ± 103.54	1.061	0.374
SL (min)	27.42 ± 9.05	26.04 ± 7.82	24.11 ± 7.85 <sup>a</sup>	25.60 ± 7.29	24.19 ± 9.16 <sup>a</sup>	34.775	0.001
REML (min)	88.34 ± 21.75	89.73 ± 27.07	83.34 ± 21.61 <sup>b</sup>	84.84 ± 22.61	84.77 ± 22.54	17.260	0.001
SE (%)	79 ± 12	80 ± 12	80 ± 11	79 ± 13	80 ± 13	2.176	0.069
NREM1 ~ 2 (min)	315.43 ± 84.44	317.11 ± 86.31	320.82 ± 82.10	316.66 ± 82.79	312.81 ± 89.06	2.018	0.089
NREM3 (min)	60.11 ± 14.26	60.27 ± 14.93	61.30 ± 16.08	61.29 ± 15.45	60.62 ± 16.12	1.999	0.092
REM (min)	104.97 ± 32.43	109.54 ± 31.48	109.81 ± 31.96	106.79 ± 32.77	100.97 ± 32.43 <sup>c</sup>	22.803	0.001
NREM (%)	75 ± 13	75 ± 11	75 ± 10	75 ± 12	74 ± 12	2.240	0.062
REM (%)	23 ± 6	23 ± 4	23 ± 5	22 ± 7	23 ± 8	1.944	0.100
WASO (min)	66.26 ± 13.30	65.49 ± 13.30	69.54 ± 13.86 <sup>b</sup>	68.12 ± 14.02	68.97 ± 16.54 <sup>b</sup>	21.843	0.001
TOB (min)	34.16 ± 9.53	35.64 ± 9.41	37.50 ± 8.75	35.30 ± 9.91	36.06 ± 10.07 <sup>ab</sup>	20.063	0.001
NOA (times/h)	14.37 ± 5.96	15.25 ± 7.14	17.24 ± 6.14 <sup>ab</sup>	16.58 ± 6.54 <sup>ab</sup>	16.32 ± 6.31 <sup>ab</sup>	38.343	0.001
RVI	1.28 ± 0.29	1.27 ± 0.29	1.28 ± 0.32	1.28 ± 0.31	1.29 ± 0.33	1.393	0.234
HRVI	5.74 ± 1.14	5.76 ± 1.08	5.76 ± 1.13	5.68 ± 1.02	5.75 ± 1.15	1.886	0.110

**Table 2.** One-way repeated measures ANOVA results of sleep architecture indicators in AIS patients of the SDB group from day 1 to day 5. <sup>a</sup>Indicates comparison with the normal group,  $P < 0.05$ . <sup>b</sup>Indicates comparison with the mild group,  $P < 0.05$ . <sup>c</sup>Indicates comparison with the moderate group,  $P < 0.05$ . <sup>d</sup>Indicates comparison with the severe group,  $P < 0.05$ .

Group/Parameter	Day 1 (n = 327)	Day 2 (n = 324)	Day 3 (n = 326)	Day 4 (n = 325)	Day 5 (n = 327)	F value	P value
TST (min)	486.38 ± 105.69	495.03 ± 102.46	487.03 ± 104.17	492.46 ± 103.74	495.64 ± 104.34	0.929	0.446
SL (min)	23.36 ± 7.6	22.56 ± 7.47	21.09 ± 7.22	20.65 ± 6.42	18.84 ± 6.63 <sup>a</sup>	19.665	0.001
REML (min)	85.39 ± 25.80	84.39 ± 24.38	85.20 ± 25.50	84.69 ± 24.59	87.24 ± 25.44	0.759	0.552
SE (%)	0.79 ± 0.12	0.79 ± 0.11	0.79 ± 0.13	0.80 ± 0.11	0.80 ± 0.10	1.281	0.275
NREM1 ~ 2 (min)	282.65 ± 80.81	298.10 ± 86.87	292.11 ± 85.05	300.61 ± 81.70	303.05 ± 84.20 <sup>a</sup>	5.606	0.001
NREM3 (min)	61.87 ± 12.26	64.33 ± 15.64	62.88 ± 18.63	65.41 ± 18.19 <sup>a</sup>	62.62 ± 14.09	3.802	0.004
REM (min)	101.56 ± 29.00	107.28 ± 25.95 <sup>a</sup>	103.36 ± 22.40	103.11 ± 26.27	104.14 ± 24.41	4.278	0.002
NREM (%)	0.74 ± 0.13	0.74 ± 0.15	0.73 ± 0.17	0.74 ± 0.11	0.74 ± 0.12	0.507	0.731
REM (%)	0.22 ± 0.07	0.22 ± 0.04	0.21 ± 0.06	0.22 ± 0.05	0.22 ± 0.06	1.661	0.157
WASO (min)	59.26 ± 18.31	65.00 ± 18.24	66.78 ± 19.26	64.75 ± 16.22	68.50 ± 17.33 <sup>a</sup>	16.414	0.001
TOB (min)	28.06 ± 8.21	31.18 ± 8.14	36.98 ± 10.61 <sup>a</sup>	37.23 ± 11.76 <sup>a</sup>	37.99 ± 12.90 <sup>a</sup>	64.592	0.001
NOA (times/h)	13.5 ± 4.22	14.91 ± 4.5	16.67 ± 4.45 <sup>ab</sup>	16.59 ± 5.66 <sup>ab</sup>	16.46 ± 5.15 <sup>ab</sup>	26.906	0.001
RVI	1.33 ± 0.38	1.33 ± 0.38	1.35 ± 0.38	1.34 ± 0.43	1.35 ± 0.40	0.246	0.912
HRVI	5.81 ± 1.2	5.77 ± 1.06	5.67 ± 1.01	5.74 ± 1.03	5.65 ± 1.29	0.946	0.436

**Table 3.** One-way repeated measures ANOVA results of sleep architecture indicators in AIS patients of the mild SDB group from day 1 to day 5. <sup>a</sup>Indicates comparison with the normal group,  $P < 0.05$ . <sup>b</sup>Indicates comparison with the mild group,  $P < 0.05$ . <sup>c</sup>Indicates comparison with the moderate group,  $P < 0.05$ . <sup>d</sup>Indicates comparison with the severe group,  $P < 0.05$ .

in the chronic stage. Since the application of IMTSMPS in this study requires a specific dedicated cyberspace to conduct various monitoring information, this study, at this time, can only be applied to hospital wards with such dedicated cyberspace, and not to post-discharge homes or rehabilitation facilities for patients originating from different regions.

Earlier research have reported significant transient changes in sleep architecture during the acute phase of ischemic stroke<sup>27–30</sup>. However, there are few reports on the continuous long-term monitoring of sleep architecture changes in patients with AIS. The results of this study showed fluctuating changes in sleep architecture, including TST, SL, REML, and NREM3, during the 5-day period in AIS patients without SDB. Similarly, AIS patients with SDB exhibited significant fluctuations in sleep architecture, including SL, REML, REM, WASO, TOB, and NOA. This suggests that AIS patients without SDB experience sleep architecture changes characterized by extended TST and shortened NREM3, while AIS patients with SDB primarily exhibit REM sleep architecture changes, such as shortened REM and extended WASO. These findings differ from previous studies that reported acute sleep architecture changes post-stroke, such as decreased TST and reduced NREM sleep<sup>31–34</sup>. The discrepancy may be due to our study's continuous 24-hour monitoring of patients, whereas other studies primarily monitored the nighttime sleep states of stroke patients. However, our results are similar to some studies that found abnormal reductions in REM time and increases in REML within the first 3 days post-stroke<sup>35–38</sup>.

Group/Parameter	Day 1 (n = 514)	Day 2 (n = 511)	Day 3 (n = 489)	Day 4 (n = 517)	Day 5 (n = 507)	F value	P value
TST (min)	514.75 ± 95.83	509.82 ± 99.26	508.62 ± 98.89	508.60 ± 97.14	509.31 ± 102.15	0.335	0.854
SL (min)	22.92 ± 7.73	23.45 ± 7.45	21.53 ± 7.10	23.74 ± 7.28	19.68 ± 7.06 <sup>d</sup>	25.537	0.001
REML (min)	84.59 ± 20.27	86.52 ± 23.49	84.84 ± 20.78	86.37 ± 18.89	83.87 ± 22.16	1.485	0.204
SE (%)	78 ± 11	78 ± 13	79 ± 11	79 ± 10	79 ± 13	0.922	0.450
NREM1 ~ 2 (min)	320.71 ± 78.28	324.15 ± 74.99	320.15 ± 76.40	318.73 ± 79.83	310.67 ± 87.37	1.974	0.096
NREM3 (min)	63.58 ± 16.18	62.67 ± 14.30	65.05 ± 16.70	62.41 ± 13.36	62.46 ± 19.35	2.322	0.055
REM (min)	114.83 ± 31.62	114.27 ± 31.81	112.41 ± 30.36	110.84 ± 32.21	111.68 ± 31.77	1.420	0.225
NREM (%)	75 ± 9	76 ± 9	76 ± 8	75 ± 10	74 ± 13	2.309	0.056
REM (%)	22 ± 6	22 ± 4	22 ± 5	22 ± 9	22 ± 8	1.568	0.180
WASO (min)	73.27 ± 18.08	73.15 ± 17.38	74.41 ± 17.07	72.32 ± 13.68	72.40 ± 10.39	1.445	0.217
TOB (min)	33.30 ± 17.33	33.73 ± 16.51	37.03 ± 15.85	37.17 ± 17.82 <sup>a</sup>	38.66 ± 17.53 <sup>a</sup>	9.188	0.001
NOA (times/h)	15.47 ± 4.47	16.29 ± 4.50	17.55 ± 4.25 <sup>a</sup>	17.31 ± 5.18 <sup>a</sup>	17.46 ± 4.65 <sup>a</sup>	18.556	0.001
RVI	1.35 ± 0.41	1.35 ± 0.37	1.36 ± 0.43	1.35 ± 0.39	1.36 ± 0.39	0.111	0.979
HRVI	6.09 ± 2.15	6.07 ± 2.16	5.85 ± 2.10	5.74 ± 2.16 <sup>a</sup>	5.81 ± 2.00	2.853	0.023

**Table 4.** One-way repeated measures ANOVA results of sleep architecture indicators in AIS patients of the moderate SDB group from day 1 to day 5. <sup>a</sup>Indicates comparison with the normal group,  $P < 0.05$ ; <sup>b</sup> Indicates comparison with the mild group,  $P < 0.05$ ; <sup>c</sup> Indicates comparison with the moderate group,  $P < 0.05$ ; <sup>d</sup> Indicates comparison with the severe group,  $P < 0.05$ .

Group/Parameter	Day 1 (n = 366)	Day 2 (n = 365)	Day 3 (n = 358)	Day 4 (n = 353)	Day 5 (n = 358)	F value	P value
TST (min)	522.24 ± 108.48	507.10 ± 113.31	534.12 ± 148.11 <sup>b</sup>	514.13 ± 106.89	519.40 ± 104.62	2.452	0.044
SL (min)	26.70 ± 9.79	25.63 ± 6.28	22.92 ± 6.45 <sup>a</sup>	25.53 ± 7.45	23.23 ± 7.73	15.804	0.001
REML (min)	88.63 ± 21.03	90.75 ± 26.59	82.86 ± 25.40 <sup>ab</sup>	85.60 ± 23.04	83.56 ± 23.40	6.800	0.001
SE (%)	77 ± 15	77 ± 14	78 ± 14	78 ± 12	78 ± 13	0.954	0.432
NREM1 ~ 2 (min)	326.96 ± 85.56	325.05 ± 90.05	328.42 ± 88.13	323.56 ± 88.37	327.06 ± 89.19	0.514	0.725
NREM3 (min)	52.10 ± 13.34	57.47 ± 15.29	58.44 ± 15.78	60.69 ± 16.31 <sup>a</sup>	58.53 ± 15.31	15.882	0.001
REM (min)	105.29 ± 22.24	101.00 ± 21.94	110.53 ± 23.48 <sup>b</sup>	108.23 ± 22.33	100.97 ± 22.87 <sup>c</sup>	12.772	0.001
NREM (%)	73 ± 11	74 ± 10	73 ± 0.12	74 ± 0.11	74 ± 0.13	0.906	0.459
REM (%)	24 ± 0.05	23 ± 0.06	23 ± 0.05	23 ± 0.05	22 ± 0.06 <sup>a</sup>	6.130	0.001
WASO (min)	73.47 ± 15.69	69.80 ± 17.37	72.99 ± 16.09	71.12 ± 21.23	71.60 ± 19.49	2.361	0.051
TOB (min)	32.79 ± 8.77	34.00 ± 10.16	37.83 ± 10.50 <sup>a</sup>	37.89 ± 10.31 <sup>a</sup>	36.32 ± 10.89	17.705	0.001
NOA (times/h)	16.05 ± 4.50	16.69 ± 4.45	17.26 ± 4.25 <sup>ab</sup>	17.53 ± 4.51 <sup>ab</sup>	17.17 ± 4.99 <sup>ab</sup>	5.578	0.001
RVI	1.30 ± 0.36	1.31 ± 0.39	1.31 ± 0.31	1.32 ± 0.31	1.31 ± 0.30	0.076	0.990
HRVI	5.65 ± 1.08	5.66 ± 1.07	5.52 ± 1.14	5.58 ± 1.02	5.60 ± 1.04	2.125	0.075

**Table 5.** One-way repeated measures ANOVA results of sleep architecture indicators in AIS patients of the severe SDB group from day 1 to day 5. <sup>a</sup>Indicates comparison with the normal group,  $P < 0.05$ . <sup>b</sup>Indicates comparison with the mild group,  $P < 0.05$ . <sup>c</sup>Indicates comparison with the moderate group,  $P < 0.05$ . <sup>d</sup>Indicates comparison with the severe group,  $P < 0.05$ .

Previous studies have revealed that varying degrees of SDB are common complications in AIS patients<sup>36,39,40</sup>. This study further explored the impact of SDB severity on the sleep architecture of AIS patients. The results showed that in mild, moderate, and severe SDB groups, different sleep architecture indicators exhibited varying degrees of fluctuation within 5 days of onset. In the mild SDB group of AIS patients, SL, NREM1-2, NREM3, REM, WASO, TOB, and NOA showed significant fluctuations during the study period, indicating marked variability in light sleep, deep sleep, and REM sleep in these patients. In the moderate SDB group, only SL, TOB, NOA, and RVI showed significant fluctuations, suggesting that moderate SDB mainly induces noticeable changes in RVI. For AIS patients in the severe SDB group, TST, SL, REML, NREM3, REM, proportion of REM sleep, TOB, and NOA showed significant fluctuations within the 5 days post-onset, indicating that severe SDB primarily causes significant variability in TST, deep sleep, and REM sleep. These results highlight the distinct impacts of different degrees of SDB on the sleep architecture of AIS patients, providing further evidence for the importance of investigating the clinical effects of SDB on AIS patients. Additionally, multivariate analysis showed that brainstem infarction increased the risk of SDB by 2.29 times, and elevated RVI also increased the risk of SDB by 2.015 times. The medulla oblongata contains centers that control respiration through a circulatory gain feedback system and receives inputs from respiratory centers within the cerebral bridges that innervate the pharyngeal muscles, which play an important role in maintaining the patency of the upper airway<sup>41</sup>. However, the risk of SDB in AIS patients was negatively correlated with REM, TOB, and RVI, indicating that these specific

sleep architecture parameters play an important role in assessing the risk of SDB in AIS patients. These findings underscore the key role of brainstem infarction and specific types of sleep architecture changes in the occurrence of SDB in AIS patients.

## Limitations

This study was designed as a single-center study. To enhance the representativeness and statistical power of the research, future studies will adopt a multi-center collaborative approach, expand the sample size, and use stratified random sampling and mixed-effects models suitable for repeated measures data for analysis.

## Conclusion

SDB is one of the most common accompanying symptoms of AIS, and its severity often fluctuates across more than two levels. Additionally, AIS patients with SDB primarily exhibit fluctuations in REM sleep architecture, but different degrees of SDB can lead to various forms of sleep architecture changes. Routine long-term continuous dynamic monitoring of sleep states is crucial in developing individualized treatment plans for AIS and improving the prognosis of these patients.

## Data availability

The datasets used and/or analyzed during the current study can be obtained from the corresponding author upon reasonable request.

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## References

- Schütz, S. G. et al. Ten-Year trends in Sleep-Disordered breathing after ischemic stroke: 2010 to 2019 data from the BASIC project. *J. Am. Heart Assoc.* **11**, e024169 (2022).
- Baillieux, S. et al. Sleep Apnoea and ischaemic stroke: current knowledge and future directions. *Lancet Neurol.* **21**, 78–88 (2022).
- Mohsenin, V. Sleep-related breathing disorders and risk of stroke. *Stroke* **32**, 1271–1278 (2001).
- Titova, O. E. et al. Sleep-disordered breathing-related symptoms and risk of stroke: cohort study and Mendelian randomization analysis. *J. Neurol.* **269**(5), 2460–2468 (2022).
- Xu, H. et al. Association between obstructive sleep apnea and lipid metabolism during REM and NREM sleep. *J. Clin. Sleep. Med.* **16**, 475–482 (2020).
- Martínez-Cerón, E. et al. Contribution of sleep characteristics to the association between obstructive sleep apnea and dyslipidemia. *Sleep. Med.* **84**, 63–72 (2021).
- Drager, L. F., Jun, J. & Polotsky, V. Y. Obstructive sleep apnea and dyslipidemia: implications for atherosclerosis. *Curr. Opin. Endocrinol. Diabetes Obes.* **17**, 161–165 (2010).
- Matsuura, D. et al. Effect on functional outcome, and treatment of sleep-disordered breathing in patients with subacute stroke. *J. Clin. Sleep. Med.* **15**, 891–897 (2019).
- Kang, D. O. et al. Impact of Sleep-Disordered breathing on functional outcomes in ischemic stroke: A cardiopulmonary coupling analysis. *Stroke* **51**, 2188–2196 (2020).
- Zhang, L. et al. Obstructive sleep apnea before ischemic stroke: clinical relevance to infarction volume and neurological recovery. *J. Stroke Cerebrovasc. Dis.* **28**, 2132–2139 (2019).
- Hoang-Anh, T., Duong-Minh, Q., Nguyen-Thi, Y. N. & Duong-Quy, S. Study of the obstructive sleep apnea syndrome in cerebral infarction patients. *Front. Neurol.* **14**, 1132014 (2023).
- Nacafaliyev, V., Ortan, P. & Sayin, S. S. Relationship between obstructive sleep Apnoea syndrome and silent brain infarction. *Postgrad. Med. J.* **22**, 67–78 (2022).
- Collop, N. A. et al. Clinical guidelines for the use of unattended portable monitors in the diagnosis of obstructive sleep apnea in adult patients. *Portable Monit. Task Force Am. Acad. Sleep. Med. J. Clin. Sleep. Med.* **3**, 737–747 (2007).
- Kurihara, Y. & Watanabe, K. Sleep-stage decision algorithm by using heartbeat and body-movement signals. *IEEE Trans. Syst. Man. Cybernetics-Part A: Syst. Hum.* **42**, 1450–1459 (2012).
- Ding, F. et al. Polysomnographic validation of an under-mattress monitoring device in estimating sleep architecture and obstructive sleep apnea in adults. *Sleep. Med.* **96**, 20–27 (2022).
- Qaseem, A. et al. Diagnosis of obstructive sleep apnea in adults: a clinical practice guideline from TheAmerican. *Coll. Physicians Ann. Intern. Med.* **161**, 210–220 (2014).
- Han, B. *Research on Non-contact Sleep Staging Algorithm Based on Heart Rate and Body Movement* (2018).
- Xia, J. S., Zhu, W. W. & Yang, T. Advances in cardiac impulse signal research and its applications in medicine. *Chin. Med. Equip.* **36**, 168–172 (2021).
- Zhang, P. S. et al. Construction and evaluation of an intelligent monitoring and analysis system for a sleep health cloud platform. *Chin. J. Health Psychol.* **30**, 1481–1488 (2022).
- American Academy of Sleep Medicine. *International Classification of Sleep Disorders* 3rd edn (American Academy of Sleep Medicine, 2014).
- Berry, R. B. B. R. et al. *The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications, Version 2.0.3* (American Academy of Sleep Medicine, 2014).
- Foldvary-Schaefer, N. R. & Waters, T. E. Sleep-Disordered breathing. *Continuum (Minneapolis Minn)*. **23**, 1093–1116 (2017).
- Wu, Z., Chen, F., Yu, F., Wang, Y. & Guo, Z. A meta-analysis of obstructive sleep apnea in patients with cerebrovascular disease. *Sleep. Breath.* **22**, 729–742 (2018).
- Tanayapong, P. & Kuna, S. T. Sleep disordered breathing as a cause and consequence of stroke: A review of pathophysiological and clinical relationships. *Sleep. Med. Rev.* **59**, 101499 (2021).
- Plomaritis, P. et al. Sleep-disordered breathing in acute stroke: A single-center, prospective, longitudinal study. *J. Clin. Med.* **12** (2023).
- McDermott, M., Brown, D. L. & Chervin, R. D. Sleep disorders and the risk of stroke. *Expert Rev. Neurother.* **18**, 523–531 (2018).
- McDermott, M. & Brown, D. L. Sleep apnea and stroke. *Curr. Opin. Neurol.* **33**, 4–9 (2020).
- Chen, Q. et al. Effect of sleep-Disordered breathing during rapid eye movement sleep and Non-Rapid eye movement sleep on acute ischemic stroke. *J. Stroke Cerebrovasc. Dis.* **30**, 105913 (2021).
- Wu, B. et al. Relationship between sleep architecture and severity of obstructive sleep apnea. *Zhejiang Da Xue Xue Bao Yi Xue Ban.* **49**, 455–461 (2020).

30. Tian, H. et al. Influence of occlusion site and baseline ischemic core on outcome in patients with ischemic stroke. *Neurology* **92**, e2626–e2643 (2019).
31. Le Bon, O. Relationships between REM and NREM in the NREM-REM sleep cycle: a review on competing concepts. *Sleep. Med.* **70**, 6–16 (2020).
32. Raven, F., Van der Zee, E. A., Meerlo, P. & Havekes, R. The role of sleep in regulating structural plasticity and synaptic strength: implications for memory and cognitive function. *Sleep. Med. Rev.* **39**, 3–11 (2018).
33. Sacchetti, M. L. & Della Marca, G. Are stroke cases affected by sleep disordered breathings all the same? *Med. Hypotheses*. **83**, 217–223 (2014).
34. Su, X. et al. A long-term ischemic stroke risk score model in patients aged 60 years and older with obstructive sleep apnea: a multicenter prospective cohort study. *Nan Fang Yi Ke Da Xue Xue Bao*. **42**, 338–346 (2022).
35. Gonzalez-Aguines, A. et al. Obstructive sleep apnea syndrome and its relationship with ischaemic stroke. *Rev. Neurol.* **69**, 255–260 (2019).
36. Šiarnik, P. et al. Sleep apnea prediction in acute ischemic stroke (SLAPS score): a derivation study. *Sleep. Med.* **77**, 23–28 (2021).
37. Liu, X. et al. Prevalence and determinants of sleep apnea in patients with stroke: A Meta-Analysis. *J. Stroke Cerebrovasc. Dis.* **30**, 106129 (2021).
38. Kawada, T. Obstructive sleep apnea and ischemic stroke: a risk assessment. *Neurol. Sci.* **40**, 2183 (2019).
39. Boulos, M. I., Dharmakulaseelan, L., Brown, D. L. & Swartz, R. H. Trials in sleep apnea and stroke: learning from the past to direct future approaches. *Stroke* **52**, 366–372 (2021).
40. Leino, A. et al. Acute stroke and TIA patients have specific polygraphic features of obstructive sleep apnea. *Sleep. Breath.* **24**, 1495–1505 (2020).
41. Trevizan-Baú, P., Stanić, D., Furuya, W. I., Dhingra, R. R. & Dutschmann, M. Neuroanatomical frameworks for volitional control of breathing and orofacial behaviors. *Respir. Physiol. Neurobiol.* **323**, 104227 (2024).

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## Author contributions

P Zhang was responsible for the organization, management, and implementation of the study. L Wang was in charge of on-site study management and data handling. Y Ou provided on-site guidance and management for the study. J Xue, Q Ma, and Y Fu were responsible for data collection and analysis. X Yuan conceived, designed, and supervised the study and was primarily responsible for writing the manuscript.

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## Declarations

## Competing interests

The authors declare no competing interests.

## Ethical approval

This study fully complies with the ethical principles of the Declaration of Helsinki. Informed consent was obtained from all participants. The study was approved by the Medical Ethics Committee of Kailuan General Hospital (Approval No. 2023005) and was registered in the Chinese Clinical Trial Registry in 2020 (Registration No. ChiCTR2000029767).

## Additional information

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