



Assessment of sleep quality and its association with autonomic function in adults

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ABSTRACT

Background: Sleep plays a fundamental role in maintaining autonomic homeostasis. Disturbances in sleep quality have been implicated in altered cardiovascular regulation, yet objective evaluation of autonomic function in relation to subjective sleep assessment remains limited. The present study examined the association between sleep quality and autonomic nervous system function in apparently healthy adults.

Material and Methods: This analytical cross-sectional study included 186 adults aged 20–50 years. Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI). Participants were categorized as good sleepers (PSQI \leq 5; n = 112) and poor sleepers (PSQI >5; n = 74).



Autonomic function was evaluated through short-term heart rate variability (HRV) analysis (time- and frequency-domain parameters) and standard cardiovascular reflex tests (deep breathing, active standing, and isometric handgrip). Intergroup comparisons were performed using appropriate statistical tests, and correlations between PSQI score and autonomic parameters were analyzed.

Results: Poor sleepers exhibited significantly higher body mass index (25.3 ± 3.4 vs 24.1 ± 3.0 kg/m²; $p = 0.021$), systolic (120.7 ± 10.3 vs 116.9 ± 8.8 mmHg; $p = 0.008$) and diastolic blood pressure (78.7 ± 7.1 vs 75.5 ± 6.2 mmHg; $p = 0.004$), and resting heart rate (77.6 ± 9.1 vs 72.1 ± 7.6 beats/min; $p < 0.001$). Time-domain HRV indices were significantly reduced in poor sleepers (SDNN: 36.5 ± 10.8 vs 48.7 ± 12.3 ms; RMSSD: 29.8 ± 11.2 vs 42.9 ± 14.1 ms; $p < 0.001$). HF power was lower (342.7 ± 156.9 vs 528.3 ± 198.4 ms²; $p < 0.001$), whereas LF/HF ratio was higher (2.12 ± 0.63 vs 1.28 ± 0.42 ; $p < 0.001$). Poor sleepers also demonstrated reduced E:I ratio and exaggerated sympathetic responses during orthostatic and handgrip testing ($p < 0.001$). PSQI score correlated negatively with SDNN ($r = -0.46$), RMSSD ($r = -0.51$), and HF power ($r = -0.48$), and positively with LF/HF ratio ($r = 0.54$) ($p < 0.001$).

Conclusion: Poor sleep quality is associated with reduced vagal modulation and relative sympathetic predominance, indicating subclinical autonomic imbalance in otherwise healthy adults.

Key words: Sleep quality; Heart rate variability; Autonomic function; Sympathovagal balance; Pittsburgh Sleep Quality Index

INTRODUCTION

Sleep is an essential physiological process that sustains homeostatic regulation and is closely interconnected with autonomic nervous system (ANS) function. The ANS, comprising sympathetic and parasympathetic branches, modulates cardiovascular dynamics during sleep and wakefulness, and alterations in this balance can have significant implications for health outcomes [1]. Heart rate variability (HRV), reflecting beat-to-beat variations in cardiac intervals, is widely recognized as a noninvasive marker of ANS modulation and sympathovagal balance [2]. Higher HRV is generally indicative of greater parasympathetic (vagal) influence and cardiovascular adaptability, whereas lower HRV suggests relative sympathetic predominance and diminished vagal modulation [3].

Disturbances in sleep quality, such as reduced sleep efficiency, fragmented rest, and short sleep duration, have been implicated in disrupted autonomic regulation. Experimental sleep restriction studies show trends toward reduced vagally mediated HRV and increased markers of sympathetic activity, indicating that insufficient sleep may shift autonomic balance toward sympathetic dominance [4]. In addition, epidemiological investigations demonstrate associations between subjectively poorer sleep quality—as assessed by standardized instruments such as the Pittsburgh Sleep Quality Index (PSQI)—and altered HRV indices in healthy individuals [5,6]. For example, cross-sectional analysis in young adults reported negative correlations between PSQI global scores and parasympathetic-related HRV measures (e.g., RMSSD, HF power), and positive correlations with sympathetic-related indices (LF and LF/HF ratio), suggesting that decreased sleep quality is accompanied by autonomic imbalance [6].

Given the established relationship between ANS dysregulation and cardiovascular risk, exploring how sleep quality relates to objective autonomic measures in apparently healthy adults may provide insights into early subclinical shifts in cardiovascular control. However, data in general



adult populations remain limited, especially outside specialized clinical groups. Therefore, the present study aimed to assess sleep quality using PSQI and examine its association with HRV and standard cardiovascular reflex tests in a cohort of healthy adults.

MATERIAL AND METHODS

Study Design and Setting: A community-based analytical cross-sectional study was conducted at a tertiary-care teaching institution. The investigation aimed to evaluate sleep quality in apparently healthy adults and examine its association with objective measures of autonomic nervous system function.

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Study Population: Participants were recruited from hospital staff (non-teaching), patient attendants, and community volunteers through public notices and direct approach.

Inclusion Criteria

- Age between 20 and 50 years
- Apparently healthy individuals without known chronic systemic disease
- Regular daytime work schedule (non-shift workers)
- Willingness to provide written informed consent

Exclusion Criteria

- Diagnosed sleep disorders (e.g., insomnia, obstructive sleep apnea)
- History of cardiovascular, endocrine, neurological, or psychiatric illness
- Diabetes mellitus or hypertension under treatment
- Use of medications influencing autonomic activity (beta-blockers, antidepressants, sedatives, antihypertensives)
- Tobacco use, alcohol dependence, or stimulant abuse
- Shift workers or individuals with irregular sleep-wake schedules
- Acute illness within the preceding two weeks

Participants were instructed to abstain from caffeine, heavy meals, and strenuous physical activity for at least 12 hours before autonomic evaluation.

Sample Size Determination: Sample size was estimated assuming a moderate correlation ($r = 0.30$) between sleep quality score and heart rate variability parameters, with a confidence level of 95% and power of 80%. The minimum calculated sample size was 138 participants. To compensate for incomplete data and potential recording artifacts, 200 individuals were enrolled. After excluding incomplete questionnaires and poor-quality ECG recordings, data from 186 participants were included in the final analysis.

Assessment of Sleep Quality: Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI). This validated self-administered instrument evaluates sleep characteristics over the preceding one month across seven domains: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, medication use, and daytime dysfunction. Each domain is scored from 0 to 3, yielding a global score ranging from 0 to 21. A global score greater



than 5 was considered indicative of poor sleep quality. Based on this criterion, participants were categorized into:

- Good sleep quality (PSQI ≤ 5)
- Poor sleep quality (PSQI > 5)

Anthropometric and Baseline Measurements: Height was measured using a wall-mounted stadiometer to the nearest 0.1 cm, and body weight was recorded using a calibrated digital weighing scale to the nearest 0.1 kg. Body mass index (BMI) was calculated as kg/m².

Resting blood pressure was measured in the seated position after five minutes of rest using an automated sphygmomanometer. Three readings were recorded at five-minute intervals, and the average was considered for analysis.

Assessment of Autonomic Function: All autonomic assessments were performed in a quiet, temperature-controlled laboratory (22–24°C) between 8:00 AM and 11:00 AM to reduce circadian variability. Participants rested in the supine position for 15 minutes before recordings.

Heart Rate Variability (HRV) Recording: Short-term HRV was recorded using a three-lead electrocardiographic system with a sampling frequency ≥ 500 Hz. A continuous 5-minute ECG was obtained under spontaneous breathing in the supine position. Artifacts and ectopic beats were visually inspected and corrected prior to analysis. HRV parameters were derived in accordance with established guidelines.

Time-domain measures:

- Mean RR interval
- Standard deviation of normal-to-normal intervals (SDNN)
- Root mean square of successive differences (RMSSD)

Frequency-domain measures:

- Low-frequency power (LF: 0.04–0.15 Hz)
- High-frequency power (HF: 0.15–0.40 Hz)
- LF/HF ratio

HF power was interpreted as an index of parasympathetic modulation, whereas LF/HF ratio was considered reflective of sympathovagal balance.

Cardiovascular Autonomic Reflex Tests: Standard non-invasive tests were performed to assess autonomic reactivity:

1. **Deep Breathing Test:** Participants performed six controlled breathing cycles per minute. The expiration-to-inspiration (E:I) ratio was calculated from RR intervals.
2. **Active Standing Test:** Heart rate and blood pressure responses were recorded immediately upon standing and at three minutes post-standing to evaluate orthostatic adjustment.

3. **Isometric Handgrip Test:** Sustained handgrip at 30% of maximal voluntary contraction was maintained for three minutes using a dynamometer. The rise in diastolic blood pressure was recorded as a marker of sympathetic reactivity.

Statistical Analysis: Data were analyzed using SPSS software (version 25.0). Continuous variables were expressed as mean \pm standard deviation or median (interquartile range) depending on data distribution. Categorical variables were presented as frequency and percentage. Normality was assessed using the Shapiro–Wilk test. Intergroup comparisons were performed using independent t-test. Chi-square test was used for categorical variables. Correlation between PSQI global score and autonomic parameters was assessed using Pearson correlation. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 200 individuals were enrolled in the study. After exclusion of incomplete questionnaires and recordings with excessive artifacts, data from 186 participants were included in the final analysis. Based on the PSQI global score, 112 participants (60.2%) were categorized as having good sleep quality, whereas 74 participants (39.8%) were classified as poor sleepers.

The mean age of the study population was 32.8 ± 7.4 years. There was no statistically significant difference in age distribution between good and poor sleepers ($p = 0.148$). Sex distribution was comparable between the two groups ($p = 0.932$). Body mass index was significantly higher among poor sleepers compared to good sleepers (25.3 ± 3.4 vs 24.1 ± 3.0 kg/m²; $p = 0.021$). Both systolic and diastolic blood pressure values were significantly elevated in participants with poor sleep quality (120.7 ± 10.3 mmHg and 78.7 ± 7.1 mmHg, respectively) compared to those with good sleep (116.9 ± 8.8 mmHg and 75.5 ± 6.2 mmHg; $p = 0.008$ and $p = 0.004$, respectively). Resting heart rate was significantly higher in poor sleepers (77.6 ± 9.1 beats/min) compared to good sleepers (72.1 ± 7.6 beats/min; $p < 0.001$). The mean PSQI global score was markedly greater in the poor sleep group (9.1 ± 2.0) than in the good sleep group (3.4 ± 1.1 ; $p < 0.001$) (Table 1).

Participants with poor sleep quality demonstrated significantly reduced time-domain HRV indices. The mean RR interval was lower in poor sleepers (776.8 ± 88.7 ms) compared to good sleepers (842.6 ± 95.4 ms; $p < 0.001$). Similarly, SDNN and RMSSD values were significantly decreased in the poor sleep group (36.5 ± 10.8 ms and 29.8 ± 11.2 ms, respectively) compared to the good sleep group (48.7 ± 12.3 ms and 42.9 ± 14.1 ms; $p < 0.001$ for both) (Table 2).

Frequency-domain analysis revealed significantly lower high-frequency (HF) power among poor sleepers (342.7 ± 156.9 ms²) compared to good sleepers (528.3 ± 198.4 ms²; $p < 0.001$). Low-frequency (LF) power was modestly but significantly higher in the poor sleep group (689.6 ± 235.1 ms²) than in the good sleep group (612.4 ± 210.7 ms²; $p = 0.041$). The LF/HF ratio was markedly elevated in participants with poor sleep quality (2.12 ± 0.63) compared to those with good sleep quality (1.28 ± 0.42 ; $p < 0.001$) (Table 3).

Autonomic reactivity differed significantly between the two groups. The E:I ratio was significantly lower in poor sleepers (1.21 ± 0.08) compared to good sleepers (1.31 ± 0.09 ; $p < 0.001$). Heart rate increment on standing was greater among poor sleepers (18.9 ± 4.2 beats/min) than good sleepers (14.6 ± 3.8 beats/min; $p < 0.001$). Additionally, the fall in systolic blood pressure on standing was



significantly more pronounced in the poor sleep group (7.6 ± 2.8 mmHg vs 4.2 ± 2.1 mmHg; $p < 0.001$). The rise in diastolic blood pressure during the isometric handgrip test was significantly higher in poor sleepers (19.5 ± 4.1 mmHg) compared to good sleepers (14.8 ± 3.6 mmHg; $p < 0.001$) (Table 4).

PSQI global score demonstrated a moderate negative correlation with SDNN ($r = -0.46$, $p < 0.001$), RMSSD ($r = -0.51$, $p < 0.001$), and HF power ($r = -0.48$, $p < 0.001$). In contrast, PSQI score showed a positive correlation with LF/HF ratio ($r = 0.54$, $p < 0.001$) and resting heart rate ($r = 0.39$, $p < 0.001$) (Table 5).

Table 1. Baseline Characteristics of Study Participants (n = 186)

Variable	Total (n = 186)	Good Sleep (n = 112)	Poor Sleep (n = 74)	p-value
Age (years)	32.8 ± 7.4	32.1 ± 7.2	33.9 ± 7.6	0.148
Male/Female (n)	98 / 88	60 / 52	38 / 36	0.932
BMI (kg/m ²)	24.6 ± 3.2	24.1 ± 3.0	25.3 ± 3.4	0.021
Systolic BP (mmHg)	118.4 ± 9.6	116.9 ± 8.8	120.7 ± 10.3	0.008
Diastolic BP (mmHg)	76.8 ± 6.7	75.5 ± 6.2	78.7 ± 7.1	0.004
Resting Heart Rate (beats/min)	74.3 ± 8.5	72.1 ± 7.6	77.6 ± 9.1	<0.001
PSQI Global Score	6.2 ± 2.9	3.4 ± 1.1	9.1 ± 2.0	<0.001

Table 2. Time-Domain Heart Rate Variability Parameters

Parameter	Good Sleep (n = 112)	Poor Sleep (n = 74)	p-value
Mean RR Interval (ms)	842.6 ± 95.4	776.8 ± 88.7	<0.001
SDNN (ms)	48.7 ± 12.3	36.5 ± 10.8	<0.001
RMSSD (ms)	42.9 ± 14.1	29.8 ± 11.2	<0.001

Table 3. Frequency-Domain Heart Rate Variability Parameters

Parameter	Good Sleep (n = 112)	Poor Sleep (n = 74)	p-value
LF Power (ms ²)	612.4 ± 210.7	689.6 ± 235.1	0.041
HF Power (ms ²)	528.3 ± 198.4	342.7 ± 156.9	<0.001
LF/HF Ratio	1.28 ± 0.42	2.12 ± 0.63	<0.001

Table 4. Cardiovascular Autonomic Reflex Tests

Test	Good Sleep (n = 112)	Poor Sleep (n = 74)	p-value
E:I Ratio	1.31 ± 0.09	1.21 ± 0.08	<0.001
HR Increase on Standing (beats/min)	14.6 ± 3.8	18.9 ± 4.2	<0.001
Fall in SBP on Standing (mmHg)	4.2 ± 2.1	7.6 ± 2.8	<0.001
Rise in DBP (Handgrip) (mmHg)	14.8 ± 3.6	19.5 ± 4.1	<0.001

Table 5. Correlation between PSQI Score and Autonomic Parameters (n = 186)

Parameter	Correlation Coefficient (r)	p-value
SDNN	-0.46	<0.001
RMSSD	-0.51	<0.001



HF Power	-0.48	<0.001
LF/HF Ratio	+0.54	<0.001
Resting Heart Rate	+0.39	<0.001

DISCUSSION

The present study demonstrated that poorer subjective sleep quality was accompanied by alterations in autonomic cardiovascular control, reflected as higher resting heart rate and blood pressure, reduced HRV parameters indicative of vagal tone, and increased sympathovagal ratio in adults without diagnosed disease. These findings are consistent with previous observations in healthy populations showing that indices of autonomic function, including HRV measures like HF power and LF/HF ratio, are adversely influenced by impaired sleep quality or sleep disturbances [7,8]. Specifically, elevated LF/HF and depressed parasympathetic HRV measures have been linked to poorer self-reported sleep quality and fragmented sleep patterns, aligning with our results pointing toward relative sympathetic predominance in poor sleepers.

Autonomic imbalance associated with poor sleep is not limited to otherwise healthy individuals. Research in clinical cohorts, such as middle-aged hypertensive patients, documented that those with poorer sleep quality exhibited lower parasympathetic modulation and attenuated baroreflex sensitivity alongside correlations between PSQI scores and autonomic markers [9,10]. Although our cohort differed by being free of overt cardiovascular disease, the direction of associations—higher heart rate, sympathetic predominance, and reduced vagal modulation with poor sleep quality—echoes these findings, reinforcing the concept that sleep quality has pervasive influences on autonomic regulation across different populations.

Moreover, broader evidence from systematic evaluations confirms that sleep disruption, whether through deprivation or poor subjective quality, is associated with autonomic dysregulation, reflected by reduced vagal modulation and relative sympathetic activation in various HRV indices. This altered autonomic profile has implications for cardiovascular stress and adaptive capacity [11,12]. A shift toward sympathetic predominance may contribute to elevated cardiometabolic risk over time, highlighting the clinical relevance of assessing sleep quality when investigating early markers of autonomic dysfunction.

Taken together, our results extend current evidence by demonstrating that even subclinical variation in sleep quality among healthy adults corresponds to measurable changes in autonomic indices, supporting the notion that sleep quality and cardiovascular autonomic regulation are integrally connected.

CONCLUSION

In this adult population, impaired sleep quality was associated with significant alterations in autonomic regulation, characterized by elevated resting heart rate and blood pressure, reduced time-domain and high-frequency heart rate variability indices, and an increased LF/HF ratio, indicating relative sympathetic predominance and diminished parasympathetic modulation. Poor sleepers also demonstrated attenuated vagal responses and exaggerated sympathetic reactivity during standard autonomic reflex testing. Furthermore, sleep quality scores showed meaningful correlations with key HRV parameters, reinforcing the relationship between disturbed sleep and autonomic imbalance. Collectively, these findings suggest that compromised sleep quality is linked to subclinical dysregulation of cardiovascular autonomic control, which may have implications for long-term cardiometabolic risk.



REFERENCES

1. Cribbet MR, Thayer JF, Jarczok MN, Fischer JE. High-Frequency Heart Rate Variability Is Prospectively Associated With Sleep Complaints in a Healthy Working Cohort. *Psychosom Med.* 2024;86(4):342-348. doi:10.1097/PSY.0000000000001302
2. Zhang S, Niu X, Ma J, Wei X, Zhang J, Du W. Effects of sleep deprivation on heart rate variability: a systematic review and meta-analysis. *Front Neurol.* 2025;16:1556784. doi:10.3389/fneur.2025.1556784
3. Fein T, Muhammad T, Lee S. The interaction between exercise and sleep with heart rate variability: cross-sectional study. *Eur J Appl Physiol.* 2026;126(1):223-237. doi:10.1007/s00421-025-05887-y
4. Chalmers T, Hickey BA, Newton P, et al. Associations between sleep quality and heart rate variability: implications for a biological model of stress detection using wearable technology. *Int J Environ Res Public Health.* 2022;19(9):5770. doi:10.3390/ijerph19095770.
5. Dawer P, Alam KK, Mishra G, Gupta M. Effect of Sleep Quality on Heart Rate Variability in Medical Students: A Cross-sectional Study. *J Assoc Physicians India.* 2025;73(11):33-36. doi:10.59556/japi.73.1209.
6. Patel MC, Singh SK. The relation of sleep quality with heart rate variability parameters in Gujarati Ethnic adolescents. *Natl J Physiol Pharm Pharmacol.* 2021;11(10):1141-1146.
7. Chalmers T, Hickey BA, Newton P, et al. Associations between Sleep Quality and Heart Rate Variability: Implications for a Biological Model of Stress Detection Using Wearable Technology. *Int J Environ Res Public Health.* 2022;19(9):5770. doi:10.3390/ijerph19095770
8. MacNeil S, Deschênes SS, Caldwell W, Brouillard M, Dang-Vu TT, Gouin JP. High-Frequency Heart Rate Variability Reactivity and Trait Worry Interact to Predict the Development of Sleep Disturbances in Response to a Naturalistic Stressor. *Ann Behav Med.* 2017;51(6):912-924. doi:10.1007/s12160-017-9915-z
9. Oliveira-Silva L, Peçanha T, Fecchio RY, et al. Poor sleep quality is associated with cardiac autonomic dysfunction in treated hypertensive men. *J Clin Hypertens (Greenwich).* 2020;22(8):1484-1490. doi:10.1111/jch.13949
10. Ooi JH, Lim R, Seng H, et al. Non-invasive parameters of autonomic function using beat-to-beat cardiovascular variations and arterial stiffness in hypertensive individuals: a systematic review. *Biomed Eng Online.* 2024;23(1):23. doi:10.1186/s12938-024-01202-6
11. da Estrela C, McGrath J, Booi L, Gouin JP. Heart Rate Variability, Sleep Quality, and Depression in the Context of Chronic Stress. *Ann Behav Med.* 2021;55(2):155-164. doi:10.1093/abm/kaaa039
12. Zabara-Antal A, Crisan-Dabija R, Arcana RI, et al. Heart Rate Variability (HRV) in Patients with Sleep Apnea and COPD: A Comprehensive Analysis. *J Clin Med.* 2025;14(13):4630. doi:10.3390/jcm14134630

