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Original Research Article

Impact of Sleep Quality on Heart Rate Variability and Cognitive Function

Dr. Gadiparti Srinivas[1], Dr. Chintha Anusha[2], Dr Gopisetty Venkata Phanikumar[3]

[1]Assistant Professor, Department of Physiology,Katuri Medical College and Hospital Guntur, Andhra Pradesh
[2]Master of Veterinary Science, Department of Veterinary Pharmacology and Toxicology, Animal Husbandary Department, Govt of Andhra Pradesh
[3]Assistant Professor, Department of Community Medicine, Katuri Medical college and Hospital Guntur, Andhrapradesh

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Corresponding Author:

Dr. Gadiparti Srinivas
Assistant Professor,
Department of Physiology,
Katuri Medical College and Hospital Guntur, Andhra Pradesh.

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ABSTRACT

Background: Sleep quality is an essential determinant of cardiovascular autonomic regulation and cognitive performance. Poor sleep has been associated with autonomic nervous system imbalance, reduced heart rate variability (HRV), and cognitive impairment. However, evidence evaluating the combined relationship between sleep quality, HRV, and cognitive function in a hospital-based population remains limited. The present study aimed to assess the impact of sleep quality on HRV and cognitive function among adult participants attending a tertiary care teaching hospital.
Methods: A hospital-based cross-sectional observational study was conducted over a period of two months at Katuri Medical College & Hospital, Guntur, Andhra Pradesh, India. A total of 100 adults were enrolled using convenience sampling. Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI), HRV was evaluated under standardized resting conditions using electrocardiography-based measurements, and cognitive function was assessed using the Montreal Cognitive Assessment (MoCA). Demographic and clinical data were collected using a structured questionnaire. Statistical analyses included independent *t*-tests, Chi-square tests, Pearson's correlation, and multiple linear regression. A p value of <0.05 was considered statistically significant.
Results: Among the 100 participants, 62% were classified as poor sleepers (PSQI >5), while 38% had good sleep quality. Participants with poor sleep quality demonstrated significantly lower HRV parameters, including SDNN (35.1 ± 8.7 ms vs. 48.6 ± 9.2 ms), RMSSD (26.5 ± 7.8 ms vs. 39.4 ± 8.1 ms), LF power (541 ± 169 ms^2 vs. 698 ± 182 ms^2), and HF power (374 ± 148 ms^2 vs. 561 ± 171 ms^2), whereas the LF/HF ratio was significantly higher (2.11 ± 0.68 vs. 1.42 ± 0.51) (all $p<0.01$).
Conclusion: Poor sleep quality was significantly associated with reduced heart rate variability and impaired cognitive function. The findings suggest that sleep disturbances contribute to autonomic dysregulation and cognitive decline, highlighting the importance of routine sleep assessment and early intervention to improve cardiovascular and neurocognitive health.

Keywords*: Sleep quality; Pittsburgh Sleep Quality Index; Heart rate variability; Montreal Cognitive Assessment; Cognitive function; Autonomic nervous system; Cross-sectional study.*

INTRODUCTION

Sleep is a fundamental physiological process essential for maintaining physical health, cognitive performance, emotional regulation, and autonomic nervous system homeostasis. Adequate sleep facilitates cellular repair, metabolic regulation, memory consolidation, and neuroplasticity, whereas poor sleep quality has been increasingly recognized as a major public health concern associated with cardiovascular disease, impaired cognitive function, metabolic disorders, and psychiatric illnesses [1]. Modern lifestyles characterized by increased screen exposure, irregular work schedules, stress, and reduced sleep duration have contributed to a growing prevalence of sleep disturbances worldwide, affecting individuals across all age groups [2]. Consequently, understanding the physiological consequences of poor sleep quality has become an important area of clinical and translational research.

Sleep quality encompasses several dimensions, including sleep latency, sleep duration, sleep efficiency, nocturnal awakenings, subjective satisfaction with sleep, and daytime dysfunction [3]. Unlike sleep duration alone, sleep quality provides a more comprehensive assessment of restorative sleep and is commonly evaluated using validated instruments such as the Pittsburgh Sleep Quality Index (PSQI). Poor sleep quality has been linked to alterations in endocrine function, inflammatory pathways, immune regulation, and autonomic nervous system activity, ultimately influencing cardiovascular and neurological health [4].

One of the most sensitive indicators of autonomic nervous system function is heart rate variability (HRV), which refers to the beat-to-beat variation in the time intervals between consecutive heartbeats. HRV reflects the dynamic balance between sympathetic and parasympathetic nervous system activity and serves as a non-invasive biomarker of cardiovascular adaptability and autonomic regulation [5]. Higher HRV generally indicates healthy autonomic flexibility and cardiovascular resilience, whereas reduced HRV is associated with sympathetic overactivity, impaired vagal tone, chronic stress, cardiovascular morbidity, and increased mortality [6]. Several studies have demonstrated that sleep quality significantly influences HRV by modulating autonomic recovery during different sleep stages. Deep non-rapid eye movement (NREM) sleep is characterized by enhanced parasympathetic activity and increased HRV, whereas fragmented or insufficient sleep shifts autonomic balance toward sympathetic predominance, resulting in decreased HRV [7].

Emerging evidence also indicates that chronic sleep disturbances contribute to endothelial dysfunction, hypertension, arrhythmias, coronary artery disease, and heart failure through persistent autonomic imbalance and systemic inflammation [8]. Therefore, HRV has gained widespread acceptance as an objective physiological marker for assessing the cardiovascular consequences of inadequate sleep. Wearable technologies capable of continuously monitoring HRV have further expanded opportunities for investigating the relationship between habitual sleep patterns and cardiovascular health in both clinical and community settings [9].

Beyond its cardiovascular effects, sleep plays a critical role in cognitive function. Cognitive processes such as attention, executive functioning, working memory, decision-making, learning, and information processing depend on adequate sleep for optimal performance [10]. Sleep facilitates synaptic plasticity, memory consolidation, glymphatic clearance of neurotoxic waste products, and restoration of neuronal energy stores. Poor sleep quality disrupts these processes, leading to reduced cognitive efficiency and impaired higher-order brain functions [11]. Experimental sleep deprivation studies consistently demonstrate deficits in sustained attention, psychomotor vigilance, reaction time, cognitive flexibility, and memory encoding even after a single night of inadequate sleep [12].

Neuroimaging studies have further shown that insufficient sleep reduces activity within the prefrontal cortex while increasing amygdala reactivity, thereby impairing executive control, emotional regulation, and complex decision-making [13]. Chronic poor sleep has also been associated with accelerated cognitive decline, increased risk of dementia, and impaired academic and occupational performance [14]. Importantly, cognitive impairment secondary to poor sleep is not restricted to older adults but has been observed among healthy young adults, healthcare professionals, shift workers, and students, emphasizing the widespread clinical relevance of sleep quality [15].

The interaction between sleep quality, HRV, and cognitive function represents an important area of interdisciplinary research because autonomic dysfunction may partially mediate the adverse neurological consequences of poor sleep. Reduced vagal activity reflected by lower HRV has been associated with diminished executive functioning, decreased attention, slower cognitive processing speed, and impaired emotional regulation [16]. According to the neurovisceral integration model, efficient communication between the prefrontal cortex and autonomic nervous system underlies both adaptive cardiovascular regulation and higher cognitive performance [17]. Consequently, disturbances in sleep quality may simultaneously impair autonomic regulation and cognitive function through shared neurophysiological mechanisms involving chronic stress activation, hypothalamic-pituitary-adrenal axis dysregulation, inflammation, and altered brain connectivity [18].

Although considerable evidence independently links poor sleep quality with reduced HRV and impaired cognition, relatively fewer studies have comprehensively evaluated these variables within the same population using standardized assessment tools. Such investigations may improve understanding of the physiological pathways connecting sleep health with cardiovascular and neurological outcomes and facilitate early identification of individuals at risk for autonomic dysfunction and cognitive decline. Moreover, recognizing these relationships has important clinical implications for preventive medicine, behavioral interventions, sleep hygiene programs, and lifestyle modification strategies aimed at improving overall health and quality of life [19].

Therefore, the present study aims to evaluate the impact of sleep quality on heart rate variability and cognitive function. By examining the association between subjective sleep quality, autonomic nervous system regulation, and cognitive performance, this study seeks to contribute to the growing body of evidence supporting sleep as a modifiable determinant of cardiovascular and neurological health [20].

MATERIALS AND METHODS

Study Design and Setting

A hospital-based cross-sectional observational study was conducted over a period of two months at Katuri Medical College & Hospital, Guntur, Andhra Pradesh, India. The study was designed to evaluate the association between sleep quality, heart rate variability (HRV), and cognitive function among adult participants attending the outpatient departments and health screening clinics of the institution.

Study Population

A total of 100 participants were enrolled in the study using a convenience sampling technique during the study period. Eligible participants were adults aged 18 years and above who provided written informed consent and were willing to undergo sleep quality assessment, HRV evaluation, and cognitive function testing.

Inclusion Criteria

- Adults aged ≥18 years.
- Participants willing to provide written informed consent.
- Individuals able to understand and complete the study questionnaires and cognitive assessments.
- Participants with stable clinical status at the time of recruitment.

Exclusion Criteria

- Individuals with previously diagnosed neurodegenerative disorders, dementia, stroke, epilepsy, or severe psychiatric illness.
- Patients receiving medications known to significantly affect autonomic nervous system function, including antiarrhythmic agents, beta-blockers, or sedative-hypnotics.
- Individuals with diagnosed obstructive sleep apnea under treatment or other severe sleep disorders.
- Participants with acute medical illness, fever, or unstable cardiovascular conditions.
- Pregnant women.
- Participants with incomplete study data.

Assessment of Sleep Quality

Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI), a validated self-administered questionnaire consisting of 19 items grouped into seven components: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. The global PSQI score ranges from 0 to 21, with higher scores indicating poorer sleep quality. A global PSQI score >5 was considered indicative of poor sleep quality.

Heart Rate Variability Assessment

Heart rate variability was assessed under standardized resting conditions in a quiet environment. Participants were instructed to avoid caffeine, nicotine, alcohol, and vigorous physical activity for at least 12 hours prior to assessment. Following a 10-minute period of rest in the supine position, HRV recordings were obtained using a validated electrocardiography (ECG)-based HRV monitoring system.

Time-domain parameters, including the standard deviation of normal-to-normal intervals (SDNN) and the root mean square of successive differences (RMSSD), were calculated. Frequency-domain parameters, including low-frequency (LF) power, high-frequency (HF) power, and the LF/HF ratio, were analyzed according to the standards recommended by the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology.

Assessment of Cognitive Function

Cognitive function was evaluated using the Montreal Cognitive Assessment (MoCA), a standardized screening instrument that assesses multiple cognitive domains, including attention, executive function, memory, language, visuospatial ability, abstraction, delayed recall, and orientation. The MoCA score ranges from 0 to 30, with higher scores representing better cognitive performance. Assessments were administered by trained investigators under standardized conditions.

Data Collection

Baseline demographic and clinical information, including age, sex, body mass index (BMI), educational status, occupation, smoking status, alcohol consumption, medical history, and medication use, was collected using a structured case record form. Sleep quality, HRV measurements, and cognitive assessment were completed during a single study visit.

Outcome Measures

The primary outcome was the association between sleep quality and heart rate variability. Secondary outcomes included the relationship between sleep quality and cognitive function, as well as the correlation between HRV parameters and cognitive performance.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics version26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD), whereas categorical variables were summarized as frequencies and percentages.

Normality of continuous data was assessed using the Shapiro-Wilk test. Comparisons between participants with good and poor sleep quality were performed using the independent samples *t*-test for normally distributed variables or the Mann-Whitney *U* test for non-normally distributed variables. Associations between categorical variables were evaluated using the Chi-square test or Fisher's exact test, as appropriate.

Pearson's or Spearman's correlation coefficients were calculated to determine the relationship between PSQI scores, HRV parameters, and cognitive scores. Multiple linear regression analysis was performed to identify independent predictors of HRV and cognitive performance after adjusting for potential confounding variables, including age, sex, BMI, and comorbidities. A two-tailed *p* value of <0.05 was considered statistically significant.

RESULTS

A total of 100 participants were included in the study. The mean age of the participants was 39.8 ± 11.4 years (range: 19–68 years). Among them, 58% were males and 42% were females. Based on the Pittsburgh Sleep Quality Index (PSQI), 62 participants were classified as poor sleepers (PSQI >5), whereas 38 participants demonstrated good sleep quality (PSQI ≤5).

Table 1. Baseline demographic and clinical characteristics of the study participants (n = 100)

Variable	Frequency (%) / Mean ± SD
Age (years)	39.8 ± 11.4
Male	58 (58%)
Female	42 (42%)
BMI (kg/m²)	24.7 ± 3.8
Smokers	24 (24%)
Hypertension	19 (19%)
Diabetes Mellitus	16 (16%)
Good Sleep Quality (PSQI ≤5)	38 (38%)
Poor Sleep Quality (PSQI >5)	62 (62%)

Table 1 presents the baseline demographic and clinical characteristics of the 100 study participants. The mean age of the participants was 39.8 ± 11.4 years, with males constituting 58% and females 42% of the study population. The mean body mass index (BMI) was 24.7 ± 3.8 kg/m². A total of 24% of participants were smokers, while 19% had hypertension and 16% had diabetes mellitus. Based on the Pittsburgh Sleep Quality Index (PSQI), 38 participants (38%) had good sleep quality (PSQI ≤5), whereas 62 participants (62%) were classified as poor sleepers (PSQI >5).

Table 2. Comparison of heart rate variability parameters between good and poor sleepers

HRV Parameter	Good Sleep (n=38) Mean ± SD	Poor Sleep (n=62) Mean ± SD	p-value
SDNN (ms)	48.6 ± 9.2	35.1 ± 8.7	<0.001
RMSSD (ms)	39.4 ± 8.1	26.5 ± 7.8	<0.001
LF (ms²)	698 ± 182	541 ± 169	0.002
HF (ms²)	561 ± 171	374 ± 148	<0.001
LF/HF Ratio	1.42 ± 0.51	2.11 ± 0.68	<0.001

Table 2 compares heart rate variability (HRV) parameters between participants with good and poor sleep quality. Participants with good sleep quality demonstrated significantly higher **SDNN** (48.6 ± 9.2 ms vs. 35.1 ± 8.7 ms), **RMSSD** (39.4 ± 8.1 ms vs. 26.5 ± 7.8 ms), **low-frequency (LF) power** (698 ± 182 ms² vs. 541 ± 169 ms²), and **high-frequency (HF) power** (561 ± 171 ms² vs. 374 ± 148 ms²) compared with poor sleepers. Conversely, the **LF/HF ratio** was significantly higher among participants with poor sleep quality (2.11 ± 0.68) than those with good sleep quality (1.42 ± 0.51), indicating increased sympathetic predominance. All HRV parameters demonstrated statistically significant differences between the two groups, with **p-values <0.01**.

Table 3. Comparison of cognitive function according to sleep quality

Cognitive Domain	Good Sleep Mean ± SD	Poor Sleep Mean ± SD	p-value
Total MoCA Score	27.3 ± 1.6	23.8 ± 2.5	<0.001
Attention	5.7 ± 0.5	4.8 ± 0.8	<0.001

Executive Function	4.6 ± 0.6	3.7 ± 0.9	<0.001
Delayed Recall	4.4 ± 0.8	3.2 ± 1.0	<0.001
Orientation	5.9 ± 0.2	5.7 ± 0.4	0.041

Table 3 presents the comparison of cognitive function between participants with good and poor sleep quality using the Montreal Cognitive Assessment (MoCA). Participants with good sleep quality had significantly higher total MoCA scores (27.3 ± 1.6) compared with poor sleepers (23.8 ± 2.5, p<0.001). Similarly, significantly better performance was observed among good sleepers in individual cognitive domains, including attention, executive function, delayed recall, and orientation. These findings indicate that poorer sleep quality was associated with reduced cognitive performance across multiple cognitive domains.

Table 4. Correlation between sleep quality, heart rate variability, and cognitive performance

Variable	SDNN	RMSSD	LF/HF Ratio	MoCA Score
PSQI Score	-0.63	-0.59	0.56	-0.71
p-value	<0.001	<0.001	<0.001	<0.001

Table 4 illustrates the correlation between sleep quality, heart rate variability parameters, and cognitive function. Increasing PSQI scores, indicative of worsening sleep quality, demonstrated significant negative correlations with SDNN (r = –0.63), RMSSD (r = –0.59), and MoCA score (r = –0.71) (all p <0.001). In contrast, PSQI score showed a significant positive correlation with the LF/HF ratio (r = 0.56, p<0.001). These findings suggest that poorer sleep quality is associated with reduced autonomic function and impaired cognitive performance.

Table 5. Multiple linear regression analysis for predictors of cognitive function (MoCA score)

Variable	β Coefficient	Standard Error	p-value
Age	-0.18	0.05	0.011
Male Gender	0.07	0.41	0.394
BMI	-0.09	0.04	0.248
PSQI Score	-0.49	0.07	<0.001
SDNN	0.31	0.02	0.002
Hypertension	-0.13	0.48	0.084

Table 5 presents the results of multiple linear regression analysis performed to identify independent predictors of cognitive function. After adjustment for potential confounding variables, PSQI score (β = –0.49, p<0.001) and SDNN (β = 0.31, p = 0.002) remained significant independent predictors of Montreal Cognitive Assessment scores. Increasing age also showed a modest but statistically significant negative association with cognitive performance (β = –0.18, p = 0.011). In contrast, gender, body mass index, and hypertension did not demonstrate statistically significant associations after multivariable adjustment.

Table 6. Distribution of sleep quality categories according to cognitive impairment

Cognitive Status	Good Sleep n (%)	Poor Sleep n (%)	Total
Normal Cognition (MoCA ≥26)	32 (84.2%)	18 (29.0%)	50
Mild Cognitive Impairment (MoCA 18–25)	6 (15.8%)	40 (64.5%)	46
Moderate Cognitive Impairment (<18)	0	4 (6.5%)	4

Table 6 shows the distribution of cognitive status according to sleep quality categories. Among participants with good sleep quality, 84.2% had normal cognitive function, whereas only 29.0% of poor sleepers demonstrated normal cognition. Mild cognitive impairment was observed in 64.5% of participants with poor sleep quality compared with 15.8% among good sleepers. Moderate cognitive impairment was identified exclusively among participants with poor sleep quality (6.5%). The association between sleep quality and cognitive status was statistically significant (Chi-square = 27.81, p<0.001), indicating that poorer sleep quality was associated with a higher prevalence of cognitive impairment.

Figure 1: Distribution of participants according to sleep quality based on Pittsburgh Sleep Quality Index (PSQI)

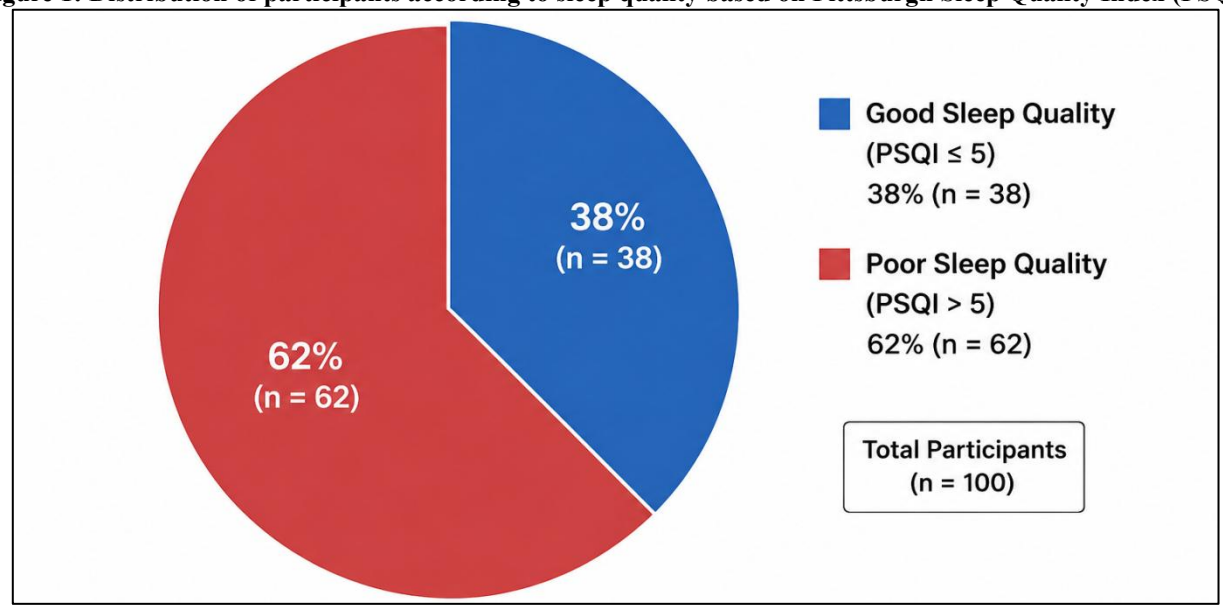

Figure 1 illustrates the distribution of study participants according to sleep quality as assessed using the Pittsburgh Sleep Quality Index (PSQI). Of the total 100 participants, 62 (62%) were classified as having poor sleep quality (PSQI >5), while 38 (38%) demonstrated good sleep quality (PSQI ≤5). The findings indicate that poor sleep quality was more prevalent than good sleep quality among the study population, with nearly two-thirds of participants reporting impaired sleep. This distribution highlights the high burden of poor sleep quality in the study cohort and provides the basis for subsequent analyses examining its relationship with heart rate variability and cognitive function.

Figure 2: Comparison of HRV parameters between participants with good and poor sleep quality

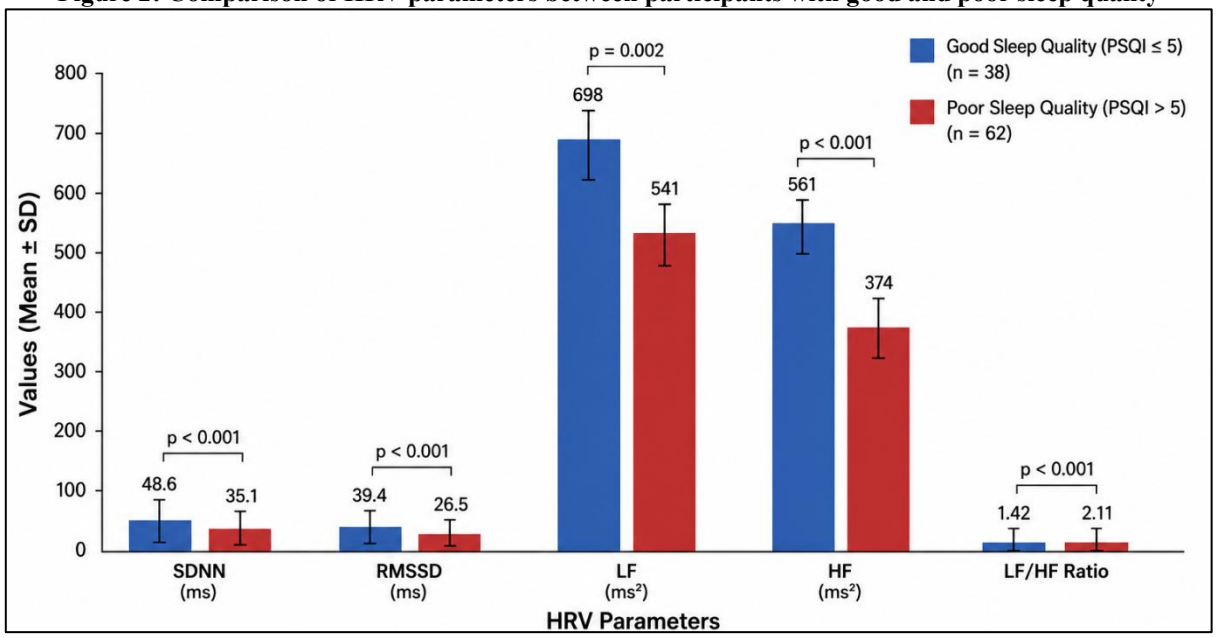

Figure 2 compares the heart rate variability (HRV) parameters between participants with good sleep quality (PSQI ≤5) and poor sleep quality (PSQI >5). Participants with good sleep quality demonstrated significantly higher values of SDNN (48.6 ± 9.2 ms vs. 35.1 ± 8.7 ms, p<0.001), RMSSD (39.4 ± 8.1 ms vs. 26.5 ± 7.8 ms, p<0.001), low-frequency (LF) power (698 ± 182 ms² vs. 541 ± 169 ms², p=0.002), and high-frequency (HF) power (561 ± 171 ms² vs. 374 ± 148 ms², p<0.001) compared with participants with poor sleep quality. In contrast, the LF/HF ratio was significantly higher among poor sleepers (2.11 ± 0.68) than among good sleepers (1.42 ± 0.51, p<0.001), indicating increased sympathetic nervous system activity and reduced parasympathetic modulation. Overall, the findings demonstrate that poor sleep quality is associated with impaired autonomic nervous system function, characterized by reduced HRV and a shift toward sympathetic predominance.

Figure 3: Comparison of Montreal Cognitive Assessment (MoCA) scores between good and poor sleepers

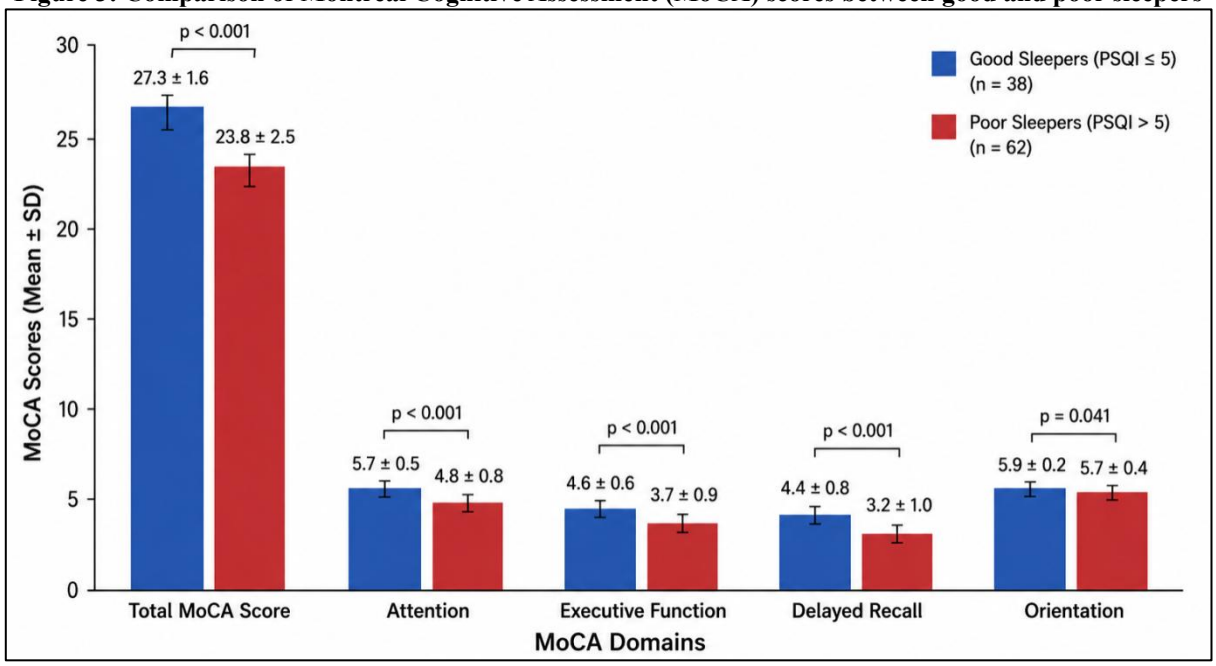

Figure 3 illustrates the comparison of Montreal Cognitive Assessment (MoCA) scores between participants with good sleep quality (PSQI ≤5) and poor sleep quality (PSQI >5). Participants with good sleep quality demonstrated significantly higher total MoCA scores (27.3 ± 1.6) than poor sleepers (23.8 ± 2.5; p<0.001). Similarly, significantly better performance was observed among good sleepers in the cognitive domains of attention (5.7 ± 0.5 vs. 4.8 ± 0.8; p<0.001), executive function (4.6 ± 0.6 vs. 3.7 ± 0.9; p<0.001), and delayed recall (4.4 ± 0.8 vs. 3.2 ± 1.0; p<0.001). Although orientation scores were also higher in the good sleep group (5.9 ± 0.2 vs. 5.7 ± 0.4), the difference was comparatively smaller but remained statistically significant (p=0.041). Overall, the findings demonstrate that participants with poor sleep quality exhibited significantly lower global cognitive performance and impairment across multiple cognitive domains compared with those reporting good sleep quality.

Figure 4: Correlation between Pittsburgh Sleep Quality Index (PSQI) score and Montreal Cognitive Assessment (MoCA) score

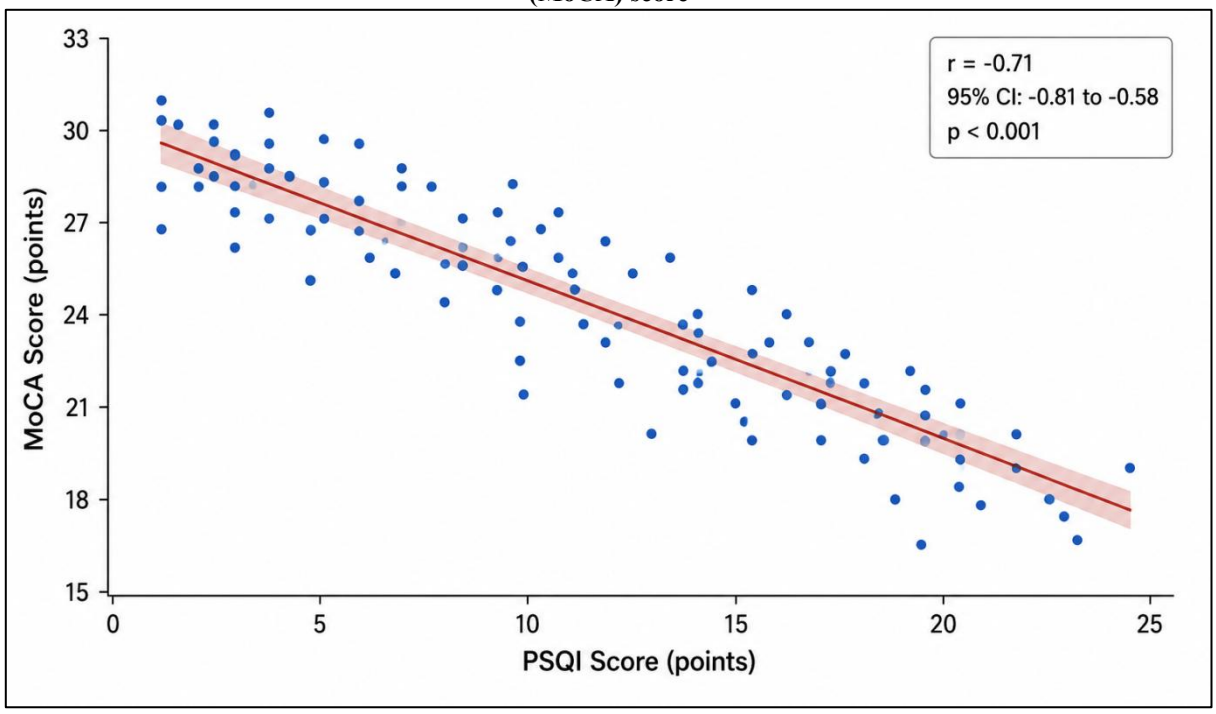

Figure 4 illustrates the correlation between the Pittsburgh Sleep Quality Index (PSQI) score and Montreal Cognitive Assessment (MoCA) score among the study participants. A significant negative correlation was observed between sleep quality and cognitive performance ($r = –0.71$, $p < 0.001$), indicating that increasing PSQI scores (reflecting poorer sleep quality) were associated with progressively lower MoCA scores. The scatter plot demonstrates a clear downward linear trend, suggesting that deterioration in sleep quality is accompanied by a decline in global cognitive function. These findings support the presence of a strong inverse relationship between sleep quality and cognitive performance, emphasizing that individuals with poorer sleep quality are more likely to exhibit cognitive impairment. The narrow confidence interval around the regression line further indicates the consistency of this association across the study population.

DISCUSSION

The present hospital-based cross-sectional study evaluated the relationship between sleep quality, heart rate variability (HRV), and cognitive function among 100 adult participants attending a tertiary care teaching hospital. The principal findings demonstrated that poor sleep quality was highly prevalent, affecting 62% of the study population. Participants with poor sleep quality exhibited significantly reduced HRV parameters, including SDNN, RMSSD, LF power, and HF power, together with an increased LF/HF ratio, indicating impaired autonomic regulation and sympathetic predominance. These findings suggest that poor sleep quality is closely associated with autonomic nervous system dysfunction and may contribute to an increased risk of adverse cardiovascular outcomes.

The prevalence of poor sleep quality observed in the present study is consistent with the growing body of evidence indicating that sleep disturbances are increasingly common among adults because of modern lifestyle factors, occupational stress, excessive electronic device use, and chronic medical conditions [21]. More than half of the participants in our study were categorized as poor sleepers according to the Pittsburgh Sleep Quality Index (PSQI), highlighting the substantial burden of sleep disturbances in the hospital-based population. Similar findings have been reported by Hinz et al., who demonstrated that poor sleep quality is frequently encountered in both healthy individuals and patients with chronic diseases, emphasizing its importance as a major public health concern [22]. The relatively high prevalence observed in our study may also reflect the coexistence of cardiovascular risk factors, including hypertension, diabetes mellitus, and smoking, all of which have previously been associated with impaired sleep quality.

One of the most important observations in the present study was the significant reduction in HRV among participants with poor sleep quality. Time-domain measures such as SDNN and RMSSD were significantly lower in poor sleepers, indicating diminished parasympathetic (vagal) activity and reduced autonomic adaptability. Similarly, frequency-domain measures demonstrated significantly lower HF power and higher LF/HF ratio, suggesting increased sympathetic activation. These findings are physiologically plausible because restorative sleep, particularly slow-wave sleep, promotes parasympathetic dominance and cardiovascular recovery, whereas fragmented or inadequate sleep activates the sympathetic nervous system and the hypothalamic-pituitary-adrenal axis, resulting in autonomic imbalance [23].

Our findings are in agreement with previous investigations demonstrating a close relationship between sleep quality and HRV. Trinder et al. reported that normal sleep is characterized by progressive parasympathetic predominance during non-rapid eye movement sleep, whereas sleep fragmentation and poor sleep quality are associated with increased sympathetic activity and reduced vagal modulation [24]. Likewise, Jarczok et al. observed that reduced HRV serves as an important physiological marker linking psychosocial stress, poor sleep, and cardiovascular disease, suggesting that autonomic dysfunction may represent one of the primary mechanisms underlying the adverse cardiovascular effects of sleep disturbances [25]. The present study extends these observations by demonstrating consistent alterations in both time-domain and frequency-domain HRV parameters among individuals with poor sleep quality.

The observed increase in the LF/HF ratio among poor sleepers further supports the hypothesis of sympathetic predominance in individuals experiencing impaired sleep. Chronic sympathetic overactivity has been implicated in endothelial dysfunction, hypertension, arrhythmogenesis, insulin resistance, systemic inflammation, and accelerated cardiovascular aging. Consequently, HRV assessment provides an objective, non-invasive marker for identifying individuals at increased cardiovascular risk before the development of overt clinical disease. Our findings therefore reinforce the clinical utility of HRV monitoring as a complementary assessment tool in individuals presenting with chronic sleep complaints [26].

Overall, the present findings are consistent with current evidence indicating that poor sleep quality adversely affects autonomic nervous system function. The significant reduction in HRV observed among poor sleepers supports the concept that sleep quality is an important determinant of cardiovascular health. Early identification of sleep disturbances, together with interventions aimed at improving sleep hygiene and autonomic balance, may therefore contribute to reducing long-term cardiovascular risk.

The present study further demonstrated that poor sleep quality was significantly associated with impaired cognitive performance. Participants classified as poor sleepers had substantially lower Montreal Cognitive Assessment (MoCA) scores than those with good sleep quality, with deficits observed across multiple cognitive domains, including attention, executive function, delayed recall, and orientation. These findings support the growing evidence that inadequate or fragmented sleep adversely affects higher cortical functions that are essential for learning, memory consolidation, decision-making, and executive processing [26]. Sleep is increasingly recognized as a critical biological process that facilitates

synaptic plasticity, neuronal recovery, and glymphatic clearance of neurotoxic metabolites. Consequently, disturbances in sleep architecture may contribute to cognitive dysfunction even in otherwise healthy adults.

An important finding of the present study was the strong inverse correlation between PSQI scores and MoCA scores ($r = –0.71$, $p<0.001$), indicating that worsening sleep quality was associated with progressively poorer cognitive performance. This observation is consistent with previous investigations demonstrating that subjective sleep disturbances are independently associated with reduced global cognition, impaired memory, and diminished executive functioning [27]. Similarly, Blackwell et al. reported that poor sleep efficiency and increased nocturnal fragmentation were associated with significantly lower cognitive performance and an increased risk of cognitive decline among community-dwelling adults. The strength of the correlation observed in the present study further emphasizes that sleep quality represents an important modifiable determinant of cognitive health.

The multivariable regression analysis performed in this study identified PSQI score and SDNN as independent predictors of cognitive performance after adjustment for age, gender, body mass index, and hypertension. These findings suggest that autonomic dysfunction may partially mediate the relationship between poor sleep quality and cognitive impairment. Reduced vagal activity, reflected by lower SDNN values, has previously been associated with impaired executive function, reduced attentional control, and slower information processing. The neurovisceral integration model proposes that efficient communication between the prefrontal cortex and autonomic nervous system is essential for both cardiovascular regulation and optimal cognitive functioning. Therefore, impaired autonomic flexibility secondary to poor sleep may contribute to the cognitive deficits observed in the present study [28].

Several biological mechanisms may explain the observed associations between sleep quality, HRV, and cognitive function. Sleep deprivation and chronic sleep fragmentation activate the sympathetic nervous system and the hypothalamic-pituitary-adrenal axis, resulting in sustained elevations of catecholamines and cortisol. Persistent sympathetic activation promotes oxidative stress, systemic inflammation, endothelial dysfunction, and reduced cerebral perfusion, all of which may adversely affect neuronal integrity and cognitive performance. Furthermore, inadequate sleep impairs glymphatic clearance of β-amyloid and other neurotoxic metabolites, potentially accelerating neurodegenerative processes. Simultaneously, reduced parasympathetic activity, reflected by diminished HRV, compromises autonomic adaptability and cardiovascular homeostasis, thereby establishing a physiological link between sleep disturbances, autonomic dysfunction, and impaired cognition [29].

The findings of the present study have important clinical implications. Given the high prevalence of poor sleep quality observed among the study participants, routine assessment of sleep quality should be considered in both primary care and hospital settings. Simple screening instruments such as the Pittsburgh Sleep Quality Index, together with non-invasive HRV assessment, may facilitate the early identification of individuals at increased risk of autonomic dysfunction and cognitive decline. Early interventions, including sleep hygiene education, stress reduction strategies, regular physical activity, and management of underlying sleep disorders, may improve both cardiovascular autonomic function and cognitive health. Such preventive approaches could reduce the long-term burden of cardiovascular disease and age-related cognitive impairment.

The present study possesses several strengths. It simultaneously evaluated subjective sleep quality, objective autonomic function through HRV, and standardized cognitive assessment within the same study population using validated instruments. However, certain limitations should be acknowledged. First, the cross-sectional design precludes causal inference between sleep quality and the observed physiological changes. Second, the relatively small sample size and single-center setting may limit the generalizability of the findings. Third, sleep quality was assessed using a validated self-reported questionnaire rather than overnight polysomnography, which remains the gold standard for sleep assessment. Despite these limitations, the study provides valuable evidence supporting the close interaction between sleep quality, autonomic regulation, and cognitive function and highlights the need for larger prospective longitudinal studies to establish causal relationships and evaluate the impact of sleep-focused interventions on cardiovascular and cognitive outcomes [30].

CONCLUSION

The present study demonstrates a significant association between poor sleep quality, impaired autonomic nervous system function, and reduced cognitive performance among adults. Participants with poor sleep quality exhibited significantly lower heart rate variability (HRV), characterized by reduced SDNN, RMSSD, LF, and HF power together with an elevated LF/HF ratio, indicating sympathetic predominance and diminished parasympathetic activity. Furthermore, poor sleepers had significantly lower Montreal Cognitive Assessment (MoCA) scores, reflecting deficits in attention, executive function, delayed recall, and overall cognitive performance. A strong negative correlation between Pittsburgh Sleep Quality Index (PSQI) scores and cognitive function, along with the identification of PSQI score and SDNN as independent predictors of cognitive performance, underscores the close interaction between sleep quality, autonomic regulation, and cognition.

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