SSR Institute of International Journal of Life Sciences
ISSN (O): 2581-8740 | ISSN (P): 2581-8732
Baksh *et al*., 2026
doi: 10.21276/SSR-IIJLS.2026.12.2.30

Original Article

open access

Impact of Sleep Deprivation on Cardiac Autonomic Function Assessed by Heart Rate Variability in Healthy Adults

Mohammed Taufiq Baksh[1], Poonam Baksh[2], Waqas Alauddin[3]*

[1]Assistant Professor, Department of Physiology, Dr. N Y Tasgaonkar Institute of Medical Sciences and Raigad Hospital, Karjat, Maharashtra, India

[2]Medical Officer, Department of Community Medicine, Dr. N Y Tasgaonkar Institute of Medical Sciences and Raigad Hospital, Karjat, Maharashtra, India

[3]Associate Professor, Department of Physiology, Dr. N Y Tasgaonkar Institute of Medical Sciences and Raigad Hospital, Karjat, Maharashtra, India

***Address for Correspondence:** Dr. Waqas Alauddin, Associate Professor, Department of Physiology, Dr. N Y Tasgaonkar Institute of Medical Sciences and Raigad Hospital, Karjat, Maharashtra, India
E-mail: dr.waqas7@gmail.com

Received: 20 Nov 2025/ Revised: 24 Dec 2025/ Accepted: 26 Feb 2026

ABSTRACT

Background: The autonomic nervous system (ANS) plays a vital role in maintaining cardiovascular homeostasis by dynamically regulating sympathetic and parasympathetic activity. Heart rate variability (HRV) is a well-established, noninvasive marker of cardiac autonomic function.

Methods: A comparative observational study was conducted on 30 healthy male participants (mean age: 18.45±2.71 years). Cardiovascular parameters, including heart rate, systolic blood pressure, and diastolic blood pressure, were recorded under normal sleep and sleep-deprived conditions. HRV analysis was performed using frequency-domain parameters, including low-frequency (LF), high-frequency (HF), LF/HF ratio, and total power (TP). Data were expressed as mean±standard deviation, and statistical significance was set at $p<0.05$.

Results: A significant reduction in heart rate was observed following sleep deprivation (75.39 vs 70.93 bpm, p=0.0001). Diastolic blood pressure showed a significant increase (78 vs 80 mmHg, p=0.0002), while the change in systolic blood pressure was not statistically significant (p=0.07). HRV analysis revealed a significant increase in LF (54.28 vs 71.12 nu), LF/HF ratio (1.18 vs 2.46), and total power (595.17 vs 1317.98 ms^2), along with a significant decrease in HF (45.71 vs 28.87 nu) (p=0.0001 for all), indicating altered autonomic balance.

Conclusion: Sleep deprivation significantly affects cardiac autonomic function, characterised by a shift toward sympathetic predominance and reduced parasympathetic activity, as evidenced by alterations in HRV.

Key-words: Sleep deprivation, Heart rate variability, Autonomic nervous system, Cardiac autonomic function, Sympathetic activity

INTRODUCTION

Sleep is essential for maintaining physiological homeostasis and optimal functioning of multiple body systems, particularly the cardiovascular system.[1]

How to cite this article
Baksh MT, Baksh P, Alauddin W. Impact of Sleep Deprivation on Cardiac Autonomic Function Assessed by Heart Rate Variability in Healthy Adults. SSR Inst Int J Life Sci., 2026; 12(2): 9518-9523.

Access this article online
https://iijls.com/

Adequate sleep plays a crucial role in regulating the autonomic nervous system (ANS), which governs heart rate, vascular tone, and blood pressure through a dynamic balance between sympathetic and parasympathetic influences. [2] The autonomic nervous system maintains cardiovascular stability by continuously modulating cardiac function. Sympathetic activation increases heart rate and peripheral vascular resistance, whereas parasympathetic (vagal) activity promotes cardiac relaxation and recovery. [3] Disruption of this balance, often referred to as sympathovagal imbalance,

doi: 10.21276/SSR-IIJLS.2026.12.2.30

has been associated with increased cardiovascular morbidity and mortality. [4]

Heart rate variability (HRV) has emerged as a reliable, non-invasive tool for assessing cardiac autonomic function. It reflects beat-to-beat variations in heart rate and provides insight into autonomic regulation. [5] Frequency-domain analysis of HRV includes low-frequency (LF) and high-frequency (HF) components, with HF reflecting parasympathetic activity and LF reflecting both sympathetic and parasympathetic influences. [6] The LF/HF ratio is commonly used as an index of sympathovagal balance. [7]

Sleep deprivation is increasingly prevalent in modern society due to lifestyle changes, academic demands, and occupational stress. It has been shown to disrupt circadian rhythms and alter neuroendocrine function, leading to increased sympathetic activity and reduced parasympathetic tone. [8] Acute sleep deprivation has been associated with elevated catecholamine levels, increased blood pressure, and impaired cardiovascular regulation. [9-10]

Several studies have demonstrated that sleep deprivation adversely affects HRV, indicating a shift toward sympathetic dominance and reduced vagal modulation [11-12]. These autonomic alterations may predispose individuals to long-term cardiovascular risk if sustained over time. [13] Despite growing evidence, there remains a need for studies evaluating the immediate effects of sleep deprivation on cardiac autonomic function in healthy young adults using HRV analysis. Therefore, the present study aims to assess the impact of sleep deprivation on cardiovascular parameters and heart rate variability indices in healthy adults.

MATERIALS AND METHODS

Research Design- This was a comparative observational study evaluating the impact of sleep deprivation on cardiac autonomic function, using cardiovascular parameters and heart rate variability (HRV) analysis. The study was conducted in the Department of Physiology at Dr N. Y. Tasgaonkar Institute of Medical Sciences and Raigad Hospital from January 2024 to June 2024.

Methodology- A total of 30 healthy male volunteers with a mean age of 18.45±2.71 years were recruited for the study. All participants were familiarised with the study protocol before data collection to ensure compliance and uniformity of procedures.

Each participant was assessed under two standardized experimental conditions:

1. Normal Sleep Condition

Participants were instructed to maintain their regular sleep routine and obtain adequate sleep of 7–8 hours on the night preceding the recording. Compliance was confirmed through self-reporting before data acquisition.

2. Sleep Deprivation Condition

Participants were subjected to acute sleep deprivation**,** defined as continuous wakefulness for approximately 24 hours**.** This was carried out under supervised conditions to ensure adherence and to prevent unintended sleep episodes.

All recordings were conducted during the morning hours **(**8:00 AM to 11:00 AM) to minimise the potential influence of circadian rhythm variations on cardiovascular and autonomic parameters. [14-17]

On the day of recording, participants were asked to report in a fasting or light-fed state. They were instructed to avoid caffeine, heavy meals, nicotine, and strenuous physical activity for at least 12 hours before testing. Upon arrival, subjects were allowed a 10–15-minute rest period in a quiet, temperature-controlled environment to achieve a stable baseline. [14-17]

Anthropometric measurements, including height and weight**,** were recorded using standard instruments, and body mass index (BMI) was calculated using the formula weight (kg)/height (m^2).

Cardiovascular Assessment- Resting cardiovascular parameters were measured with the participant in the supine position. Heart rate was recorded from the electrocardiogram (ECG), and blood pressure was measured using a calibrated sphygmomanometer according to standard guidelines. Two readings were taken, and the average value was considered for analysis. [14-17]

Heart Rate Variability (HRV) Analysis- HRV was assessed using a standard ECG recording under resting conditions**.** obtained, and frequency-domain analysis was performed in accordance with established international guidelines. [14-17]

doi: 10.21276/SSR-IIJLS.2026.12.2.30

The following HRV parameters were analyzed: [14-17]

- **Low Frequency (LF) (nu)**– reflecting both sympathetic and parasympathetic influences
- **High Frequency (HF) (nu)**– representing parasympathetic (vagal) activity
- **LF/HF ratio**– indicating sympathovagal balance
- **Total Power (ms²)**– representing overall autonomic activity

Care was taken to minimise external disturbances during recording to avoid artefacts and ensure data reliability.

Inclusion Criteria

- ✓ Healthy individuals aged 18–25 years
- ✓ Willingness to participate and provide written informed consent

Exclusion Criteria

- ✓ History of cardiovascular disease
- ✓ Diabetes mellitus or any chronic systemic illness
- ✓ Smoking or alcohol consumption
- ✓ Use of medications affecting autonomic function
- ✓ Known sleep disorders or irregular sleep patterns

Statistical Analysis- Data were expressed as mean±standard deviation (SD). Comparisons between normal sleep and sleep-deprived conditions were performed using appropriate paired statistical tests. A p-value<0.05 was considered statistically significant. Statistical analysis was carried out using standard statistical software.

Ethical Approval- The study protocol was approved by the Institutional Ethics Committee and written informed consent was obtained from all participants before inclusion in the study. The study was conducted in accordance with ethical principles for biomedical research involving human subjects.

RESULTS

Thirty healthy male participants were included. Mean age was 18.45±2.71 years and BMI 21.17±2.15 kg/m². Baseline cardiovascular parameters were within normal limits. The average sleep duration reported was 7±0.9 hours. Baseline characteristics are summarized in Table 1.

Table 1: Demographic and Baseline Clinical Characteristics of Study Participants (n=30)

Parameter	Mean±SD
Age (years)	18.45±2.71
Body Mass Index (kg/m²)	21.17±2.15
Basal Heart Rate (bpm)	75.96±6.20
Systolic Blood Pressure (mmHg)	114±2.61
Diastolic Blood Pressure (mmHg)	76.80±3.08
Average Sleep Duration (hours)	7±0.9

Values are expressed as median (range).

The comparison of cardiovascular parameters between normal sleep and sleep-deprived conditions is shown in Table 2. A statistically significant reduction in heart rate was observed following sleep deprivation (75.39±6.12 bpm vs 70.93±5.88 bpm; p=0.0001). Diastolic blood pressure showed a significant increase (78±3.10 mmHg vs 80±3.25 mmHg; p=0.0002). However, the increase in systolic blood pressure was not statistically significant (p=0.07).

Table 2: Comparison of Cardiovascular Parameters Between Normal Sleep and Sleep-Deprived Conditions

Parameter	Normal Sleep (Mean±SD)	Sleep Deprived (Mean±SD)	p-value	Significance
Heart Rate (bpm)	75.39±6.12	70.93±5.88	0.0001*	Significant
Systolic BP (mmHg)	114±2.61	116±3.02	0.07	Not Significant
Diastolic BP (mmHg)	78±3.10	80±3.25	0.0002*	Significant

*Values are expressed as median (range). *p<0.05 (significant)*

doi: 10.21276/SSR-IIJLS.2026.12.2.30

Changes in heart rate variability (HRV) parameters are presented in Table 3**.** Sleep deprivation resulted in a significant increase in low-frequency (LF) power (54.28±6.45 vs 71.12±7.03 nu; p=0.0001) and LF/HF ratio (1.18±0.32 vs 2.46±0.51; p=0.0001), indicating enhanced sympathetic activity. In contrast, high-frequency (HF) power significantly decreased (45.71±5.98 vs 28.87±6.12 nu; p=0.0001), reflecting reduced parasympathetic activity.

Additionally, total power increased significantly following sleep deprivation (595.17±120.45 vs 1317.98±210.62 ms^2; p=0.0001), suggesting increased overall autonomic responsiveness.

Table 3: Comparison of Heart Rate Variability Parameters Between Normal Sleep and Sleep-Deprived Conditions

Parameter	Normal Sleep (Mean±SD)	Sleep Deprived (Mean±SD)	p-value	Significance
LF (nu)	54.28±6.45	71.12±7.03	0.0001*	Significant
HF (nu)	45.71±5.98	28.87±6.12	0.0001*	Significant
LF/HF Ratio	1.18±0.32	2.46±0.51	0.0001*	Significant
Total Power (ms^2)	595.17±120.45	1317.98±210.62	0.0001*	Significant

*Values are expressed as median (range). *p<0.05 (significant)*

DISCUSSION

The present study evaluated the impact of sleep deprivation on cardiac autonomic function using cardiovascular parameters and heart rate variability (HRV) indices in healthy adults. The findings demonstrate that acute sleep deprivation significantly alters autonomic balance, with a shift toward sympathetic predominance and reduced parasympathetic activity.

A significant reduction in heart rate was observed following sleep deprivation. Although sleep deprivation is generally associated with increased sympathetic activity, this paradoxical reduction in heart rate may be explained by compensatory vagal mechanisms or altered baroreceptor sensitivity[1]. Similar findings have been reported in studies suggesting complex autonomic adjustments during acute sleep loss. [2]

Diastolic blood pressure showed a significant increase, while systolic blood pressure remained unchanged. The rise in diastolic pressure may reflect increased peripheral vascular resistance mediated by sympathetic activation[3]. Sleep deprivation has been shown to elevate catecholamine levels and impair endothelial function, thereby increasing vascular tone.[4]

The HRV findings in the present study strongly support a shift toward sympathetic dominance. A significant increase in low-frequency (LF) power and LF/HF ratio, along with a decrease in high-frequency (HF) power, indicates reduced vagal modulation and enhanced sympathetic activity. These results are consistent with previous studies demonstrating autonomic imbalance following sleep deprivation.[5,6]

The LF component of HRV reflects both sympathetic and parasympathetic influences, whereas HF is primarily mediated by vagal activity[7]. The observed increase in LF/HF ratio is widely interpreted as an indicator of sympathovagal imbalance, which has been associated with increased cardiovascular risk. [8]

Interestingly, total power was significantly increased in the sleep-deprived state. This suggests an overall increase in autonomic activity rather than suppression.[14-17] Similar observations have been reported in studies indicating heightened physiological arousal and autonomic reactivity during acute sleep deprivation.[9]

The underlying mechanisms linking sleep deprivation to autonomic dysfunction include activation of the hypothalamic–pituitary–adrenal (HPA) axis, increased cortisol secretion, and elevated sympathetic nervous system activity.[10] Disruption of circadian rhythms further contributes to impaired autonomic regulation and cardiovascular instability.[18-20]

These findings have important clinical implications. Persistent sleep deprivation may lead to chronic autonomic imbalance, thereby increasing the risk of hypertension, arrhythmias, and cardiovascular disease. [18-20] Early identification of such changes through HRV analysis may help in preventive strategies. [14-17]

doi: 10.21276/SSR-IIJLS.2026.12.2.30

STRENGTHS

The present study has several strengths. It employed a within-subjects design, allowing each participant to serve as their own control, thereby minimising inter-individual variability. The study used standardised experimental conditions, including controlled sleep deprivation and uniform timing of data collection, which reduced the influence of circadian rhythms. Furthermore, the use of heart rate variability (HRV) analysis, a well-established and non-invasive tool, enhanced the reliability of assessing cardiac autonomic function. The inclusion of multiple HRV parameters (LF, HF, LF/HF ratio, and total power) provided a comprehensive evaluation of autonomic balance**.** Additionally, participants were screened using strict inclusion and exclusion criteria, ensuring a homogeneous, healthy study population and thereby improving internal validity.

LIMITATIONS

Despite its strengths, the study has certain limitations. The sample size was relatively small**,** which may limit the generalizability of the findings. The inclusion of only male participants restricts the applicability of the results to the broader population, particularly females. The study focused on acute sleep deprivation, and therefore, the findings may not reflect the long-term effects of chronic sleep deprivation on cardiac autonomic function. Although sleep deprivation was conducted under supervised conditions, it was not objectively verified using polysomnography or actigraphy**,** which could have provided a more accurate assessment of sleep status. Additionally, potential confounding factors such as psychological stress, dietary habits, and individual circadian preferences were not controlled, which may have influenced autonomic responses.

CONCLUSIONS

The present study demonstrates that acute sleep deprivation significantly alters cardiac autonomic function, as evidenced by a shift toward sympathetic predominance and reduced parasympathetic activity. These findings underscore the critical role of adequate sleep in maintaining optimal autonomic balance and cardiovascular health. Early identification of such autonomic alterations through heart rate variability analysis may help prevent long-term cardiovascular complications.

ACKNOWLEDGMENTS

The authors would like to acknowledge the support of the Department of Physiology at Dr. N. Y. Tasgaonkar Institute of Medical Sciences and Raigad Hospital for providing the necessary facilities to conduct this study. The authors are also grateful to all the participants who volunteered for the study and contributed to its successful completion.

CONTRIBUTION OF AUTHORS

Research concept- Mohammed Taufiq Baksh
Research design- Mohammed Taufiq Baksh
Supervision- Waqas Alauddin
Materials- Mohammed Taufiq Baksh
Data collection- Mohammed Taufiq Baksh
Data analysis and interpretation- Mohammed Taufiq Baksh, Poonam Baksh
Literature search- Waqas Alauddin, Mohammed Taufiq Baksh, Poonam Baksh
Writing article- Waqas Alauddin, Mohammed Taufiq Baksh, Poonam Baksh
Critical review-Mohammed Taufiq Baksh, Poonam Baksh
Article editing- Mohammed Taufiq Baksh
Final approval- Mohammed Taufiq Baksh, Waqas Alauddin

REFERENCES

[1] Zhang S, Niu X, Ma J, Wei X, et al. 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.
[2] Jain N, Lehrer HM, Chin BN, Tracy EL, Evans MA, et al. Heart rate and heart rate variability following sleep deprivation in retired night shift workers and retired day workers. Psychophysiology., 2023; 60(12): e14374. doi: 10.1111/psyp.14374.
[3] Takase B, Akima T, Satomura K, Ohsuzu F, et al. Effects of chronic sleep deprivation on autonomic activity by examining heart rate variability, plasma catecholamine, and intracellular magnesium levels. Biomed Pharmacother., 2004; 58(Suppl 1): S35–39. doi: 10.1016/s0753-3322(04)80007-6.
[4] Crooks E, Hansen DA, Satterfield BC, Layton ME, Van Dongen HPA. Cardiac autonomic activity during sleep deprivation with and without caffeine administration. Physiol Behav., 2019; 210: 112643. doi: 10.1016/j.physbeh.2019.112643.

doi: 10.21276/SSR-IIJLS.2026.12.2.30

[5] Oliveira-Silva L, Peçanha T, Fecchio RY, Rezende RA, Abreu A, et al. Poor sleep quality is associated with cardiac autonomic dysfunction in treated hypertensive men. J Clin Hypertens (Greenwich), 2020; 22(8): 1484–90. doi: 10.1111/jch.13949.

[6] Xu Y, Qu B, Liu F, Gong Z, Zhang Y, et al. Sleep deprivation and heart rate variability in healthy volunteers: effects of REM and SWS sleep deprivation. Comput Math Methods Med., 2023; 2023: 7121295. doi: 10.1155/2023/7121295.

[7] Bourdillon N, Jeanneret F, Nilchian M, Albertoni P, Ha P, Millet GP. Sleep deprivation deteriorates heart rate variability and photoplethysmography. Front Neurosci., 2021; 15: 642548. doi: 10.3389/fnins.2021.642548.

[8] Onimaru LJ, Christofaro DGD, Valente HB, Leoci IC, Andersen ML, et al. The relationship between sleep quality and cardiac autonomic modulation according to physical activity levels in adults: a cross-sectional study. Sleep Breath., 2025; 29(4): 216. doi: 10.1007/s11325-025-03388-y.

[9] Glos M, Fietze I, Blau A, Baumann G, Penzel T. Cardiac autonomic modulation and sleepiness: physiological consequences of sleep deprivation due to 40 h of prolonged wakefulness. Physiol Behav., 2014; 125: 45–53. doi: 10.1016/j.physbeh.2013.11.011.

[10]Schlagintweit J, Laharnar N, Glos M, Zemann M, Demin AV, et al. Effects of sleep fragmentation and partial sleep restriction on heart rate variability during night. Sci Rep., 2023; 13(1): 6202. doi: 10.1038/s41598-023-33013-5.

[11]Lins-Filho OL, Andrade-Lima A, Torres AD, Oliveira LM, Luiz do-Prado W, et al. Association between sleep quality and cardiac autonomic modulation in adolescents: a cross sectional study. Sleep Sci., 2023; 16(4): e462–67. doi: 10.1055/s-0043-1776750.

[12]Quintana DS, Elvsåshagen T, Zak N, Norbom LB, Pedersen PO, et al. Diurnal variation and twenty-four hour sleep deprivation do not alter supine heart rate variability in healthy male young adults. PLoS One., 2017; 12(2): e0170921. doi: 10.1371/journal.pone.0170921.

[13]Oliver MD, Baldwin DR, Datta S. The relationship between sleep and autonomic health. J Am Coll Health, 2020; 68(5): 550–56. doi: 10.1080/07448481.2019.1583652.

[14]Alauddin W, Radke PM, Janardhana N, Singh I, et al. Heart rate variability and intrinsic autonomic coupling in Ehlers-Danlos syndrome. Cureus, 2025; 17(12): e98693. doi: 10.7759/cureus.98693.

[15]Alauddin W, Radke PM, Pardhi P, Mishra M, Arora S. Effects of revascularization on cardiac autonomic neuropathy in type 2 diabetes mellitus patients with chronic stable angina. Cureus, 2024; 16(10): e72278. doi: 10.7759/cureus.72278.

[16]Alauddin W, Chaswal M, Bashir M, Isser HS. Cardiovascular autonomic modulation in chronic coronary syndrome following percutaneous coronary intervention. Cureus, 2024; 16(7): e65092. doi: 10.7759/cureus.65092.

[17]Alauddin W, Chaswal M, Bashir M, Isser HS. A study of cardiac autonomic functions in patients with chronic stable angina undergoing percutaneous coronary revascularization. Medeni Med J., 2021; 36(2): 91–97. doi: 10.5222/MMJ.2021.24603.

[18]Słomko J, Zawadka-Kunikowska M, Kozakiewicz M, Klawe JJ, Tafil-Klawe M, et al. Hemodynamic, autonomic, and vascular function changes after sleep deprivation for 24, 28, and 32 hours in healthy men. Yonsei Med J., 2018; 59(9): 1138–42. doi: 10.3349/ymj.2018.59.9.1138.

[19]Citi L, Klerman E, Brown E, Barbieri R. Point process heart rate variability assessment during sleep deprivation. Comput Cardiol., 2010; 37: 721–24.

[20]Nano M, Fonseca P, Overeem S, Vullings R, Aarts RM. Lying awake at night: cardiac autonomic activity in relation to sleep onset and maintenance. Front Neurosci., 2020; 13: 1405. doi: 10.3389/fnins.2019.01405.