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Effect of Acute Sleep Deprivation on Cardiac Autonomic Stress Reactivity in Healthy Young Adults

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ABSTRACT

Background: Sleep deprivation is increasingly recognized as a significant contributor to autonomic imbalance and cardiovascular risk, being associated with reduced parasympathetic activity and heightened sympathetic drive; therefore, this study aims to evaluate the effect of acute sleep deprivation on cardiac autonomic stress reactivity in healthy young adults.

Methods: This cross-sectional study included 30 healthy male participants. Cardiac autonomic function tests were performed under two conditions: after normal sleep and following acute sleep deprivation. Parasympathetic reactivity was assessed using the deep breathing test, Valsalva manoeuvre, and active standing test, while sympathetic reactivity was evaluated using the isometric handgrip test and orthostatic blood pressure response. Data were expressed as mean±SD or median (range).

Results: Acute sleep deprivation resulted in a significant reduction in parasympathetic activity, evidenced by decreased ΔHR during deep breathing (p=0.0001) and reduced Valsalva ratio (p=0.02). Tachycardia latency decreased (p=0.007), whereas bradycardia latency increased (p=0.03). Sympathetic reactivity showed a significant increase, with elevated ΔDBP during the handgrip test at 1 and 2 minutes (p<0.05). Orthostatic systolic blood pressure changes were not statistically significant.

Conclusion: Acute sleep deprivation leads to a significant shift toward sympathetic dominance with concomitant reduction in parasympathetic tone, indicating an autonomic imbalance that may predispose to cardiovascular morbidity.

Key-words: Sleep deprivation, autonomic nervous system, cardiovascular reactivity, parasympathetic, sympathetic

INTRODUCTION

Sleep is a fundamental physiological process essential for maintaining cardiovascular, metabolic, and neuroendocrine homeostasis. [1,2] Adequate sleep plays a critical role in regulating autonomic balance, hormonal secretion, and inflammatory pathways. [1,2] Disturbances in sleep, particularly sleep deprivation, have been increasingly recognized as significant contributors to adverse cardiovascular outcomes, including hypertension, arrhythmias, and ischemic heart disease. [1,2] In recent years, the growing prevalence of sleep deprivation due to modern lifestyle patterns has made it an important public health concern.

The autonomic nervous system (ANS), comprising sympathetic and parasympathetic divisions, is the principal regulator of cardiovascular function. It maintains dynamic control over heart rate, vascular tone, and blood pressure through a finely tuned balance between sympathetic excitation and parasympathetic (vagal) inhibition. Sleep deprivation disrupts this equilibrium, typically leading to reduced vagal activity

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and increased sympathetic drive, thereby promoting an autonomic imbalance. [3,4] Such alterations may lead to increased cardiovascular strain and reduced adaptability to physiological stress.

Cardiac autonomic function tests, including the deep breathing test, Valsalva maneuver, active standing test, and isometric handgrip test, are well-established, non-invasive tools for evaluating autonomic reactivity. [5,6] These tests provide insight into both parasympathetic and sympathetic components of autonomic control under standardized conditions. [5,6] Previous studies have reported alterations in autonomic parameters following sleep deprivation; however, many have focused predominantly on either heart rate variability or isolated autonomic indices. Comprehensive assessment of both parasympathetic and sympathetic stress reactivity within the same cohort remains relatively limited.

Furthermore, most available studies emphasize chronic sleep restriction, while the immediate effects of acute sleep deprivation on integrated autonomic responses are less clearly defined. Understanding these early changes is important, as they may represent initial physiological disturbances that precede long-term cardiovascular dysfunction.

Therefore, the present study was undertaken to evaluate the impact of acute sleep deprivation on cardiac autonomic stress reactivity, assessing both parasympathetic and sympathetic components in healthy young adults.

MATERIALS AND METHODS

Research Design- This cross-sectional analytical study was conducted in the Department of Physiology at Dr N.Y. Tasgaonkar Institute of Medical Science and Raigad Hospital between January 2024 and June 2024. The study was conducted in a controlled laboratory environment to ensure standardized testing conditions and minimize external variability.

Methodology- A total of 30 healthy male volunteers aged 18–25 years were enrolled in the study. Participants were recruited from undergraduate students after obtaining informed written consent. All subjects underwent preliminary screening through detailed clinical history and physical examination to exclude any underlying medical conditions. [7-10]

Each participant underwent cardiac autonomic function testing under two standardized conditions: (1) after a normal night's sleep (baseline condition) and (2) after acute sleep deprivation (test condition). For the sleep deprivation protocol, participants were instructed to remain awake for one full night under supervision and report to the laboratory the following morning. [3-4]

All tests were conducted between 8:00 AM and 10:00 AM to minimize circadian influences. Participants were advised to abstain from caffeine, heavy meals, and strenuous physical activity for at least 12 hours before testing. [7-10]

Cardiac autonomic function was assessed using standard non-invasive tests according to established guidelines [7-10]. Parasympathetic reactivity was evaluated using the deep breathing test, Valsalva maneuver, tachycardia and bradycardia latencies, and the active standing test (HR_{15}/HR_{30} ratio). Sympathetic reactivity was assessed using the isometric handgrip test, with measurement of diastolic blood pressure changes (ΔDBP) at 1, 2, and 3 minutes, and the orthostatic test, which recorded changes in systolic blood pressure (ΔSBP). [7-10]

Electrocardiographic recordings and heart rate measurements were obtained using a standard computerized physiograph system. Blood pressure was measured using a calibrated sphygmomanometer. All measurements were recorded by trained personnel to ensure accuracy and reduce inter-observer variability.

Inclusion Criteria

- Healthy male individuals aged 18–25 years
- Willingness to participate and provide informed consent
- Regular sleep duration of 6–8 hours per night [3-4]

Exclusion Criteria

- History of cardiovascular, neurological, or endocrine disorders
- Diabetes mellitus or hypertension
- Use of medications affecting autonomic function
- Smoking, alcohol consumption, or substance abuse
- History of sleep disorders [3-4]

Statistical Analysis- Data were entered and analyzed using appropriate statistical software. Continuous variables were expressed as mean±standard deviation or median (range), depending on the distribution of data.

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Comparisons between normal sleep and sleep-deprived conditions were performed using paired statistical tests. A p-value of less than 0.05 was considered statistically significant.

Ethical Approval- Ethical approval for the study was obtained from the Institutional Ethics Committee before the commencement of the study. The study was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants after they were informed of the study's purpose and procedures.

RESULTS

A total of 30 healthy male subjects were included in the study. The mean age of participants was 18.45±2.71 years**,** and the mean body mass index (BMI) was 21.17±2.15 kg/m²**,** indicating a homogeneous study population with normal nutritional status. Baseline cardiovascular parameters were within physiological limits, with a mean resting heart rate of 75.96±6.20 beats/min**,** systolic blood pressure of 114±2.61 mmHg**,** and diastolic blood pressure of 76.80±3.08 mmHg as depicted in Table 1**.** The average habitual sleep duration was 7.0±0.9 hours**,** confirming adequate baseline sleep before experimental intervention.

Table 1: Baseline Characteristics of Study Participants

Parameter	Value
Number of subjects	30
Gender	Male
Age (years)	18.45±2.71
BMI (kg/m²)	21.17±2.15
Basal heart rate (beats/min)	75.96±6.20
Basal systolic blood pressure (mmHg)	114±2.61
Basal diastolic blood pressure (mmHg)	76.80±3.08
Average night sleep (hours)	7.0±0.9

Values are expressed as median (range).

Acute sleep deprivation produced a consistent and statistically significant attenuation of parasympathetic reactivity across multiple indices. The heart rate response to deep breathing (ΔHR) showed a marked reduction, declining from 18.00 (17–20) under normal sleep conditions to 13.00 (12–14) following sleep deprivation (*p=0.0001*), representing a substantial decrease in vagally mediated heart rate variability. Similarly, the Valsalva ratio decreased significantly from 1.70 (1.47–1.92) **to** 1.29 (1.19–1.83) (*p=0.0212*), further supporting diminished parasympathetic modulation, as depicted in Table 2.

Table 2: Comparison of Cardiac Autonomic Stress Reactivity After Normal Sleep and Sleep Deprivation

Parameter	After Normal Sleep	After Sleep Deprivation	p-value
Parasympathetic Reactivity			
Deep breathing test (ΔHR)	18.00 (17–20)	13.00 (12–14)	0.0001**
Valsalva ratio	1.70 (1.47–1.92)	1.29 (1.19–1.83)	0.02*
Tachycardia latency	10.87 (8.67–12.51)	9.60 (8.03–10.19)	0.007**
Bradycardia latency	8.07 (4.01–9.59)	9.10 (7.58–11.41)	0.03*
Active standing (HR_{15}/HR_{30})	1.35 (1.25–1.50)	1.25 (1.20–1.36)	0.05

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Sympathetic Reactivity			
ΔDBP (1 min)	10 (0–10)	20 (10–20)	0.01*
ΔDBP (2 min)	10 (10–20)	20 (16–30)	0.001**
ΔDBP (3 min)	24 (10–30)	30 (20–30)	0.09
Orthostatic ΔSBP	8 (6–10)	7 (6–9)	0.28

Values are expressed as median (range).
***p<0.05 (significant), **p<0.01 (highly significant)*

DISCUSSION

The present study demonstrates that acute sleep deprivation produces a significant shift in cardiac autonomic balance, characterized by reduced parasympathetic activity and enhanced sympathetic reactivity. This pattern reflects autonomic dysregulation, which may represent an early functional marker of cardiovascular risk.

The observed reduction in heart rate variability during deep breathing and the significant decline in the Valsalva ratio indicate impaired vagal modulation of cardiac function. These findings are consistent with previous studies showing that sleep deprivation suppresses parasympathetic activity and reduces baroreflex sensitivity.[11-13] Vagal withdrawal is particularly important because parasympathetic tone exerts a protective effect on the heart by stabilizing electrical activity and limiting excessive sympathetic stimulation. [4] Therefore, even short-term reductions in vagal activity may predispose individuals to arrhythmogenic and hypertensive states.

In parallel, the present study demonstrated a significant increase in sympathetic reactivity, as evidenced by the augmented diastolic blood pressure response during the isometric handgrip test. This finding aligns with earlier experimental studies showing increased sympathetic nerve activity and elevated catecholamine levels following sleep deprivation.[14,15] The underlying mechanism is likely multifactorial, involving activation of the hypothalamic–pituitary–adrenal axis, increased cortisol secretion, and heightened central sympathetic outflow.[16] This sympathetic predominance contributes to increased vascular tone and peripheral resistance, which may explain the exaggerated pressor response observed in this study.

An important observation in the present study is the differential effect of sleep deprivation on various autonomic tests. While parasympathetic indices were consistently impaired and sympathetic reactivity was enhanced, the orthostatic blood pressure response did not differ significantly. This suggests that acute sleep deprivation may selectively affect dynamic autonomic responses rather than basal reflex mechanisms. Such selective vulnerability of autonomic pathways has also been reported in prior studies and may indicate early-stage autonomic imbalance rather than overt dysfunction.[12,17]

From a clinical perspective, these findings are particularly relevant in the context of modern lifestyle patterns, where acute and chronic sleep deprivation are increasingly common. Repeated exposure to such an autonomic imbalance may contribute to the development of hypertension, endothelial dysfunction, and increased cardiovascular morbidity.[18,19] Moreover, reduced parasympathetic activity combined with heightened sympathetic tone has been associated with increased inflammatory responses and metabolic dysregulation, further amplifying cardiovascular risk [20].

STRENGTHS

The present study provides a comprehensive evaluation of cardiac autonomic function by assessing both parasympathetic and sympathetic components using standardized, non-invasive tests. The use of multiple autonomic function parameters enhances the reliability of findings and allows for a more integrated understanding of autonomic responses to acute sleep deprivation. Additionally, conducting the study under controlled laboratory conditions minimized external variability and improved the internal validity of the results.

LIMITATIONS

Despite its strengths, the study has certain limitations. The sample size was relatively small, which may limit the statistical power and generalizability of the findings. Including only male participants further limits the applicability of the results to the broader population. Moreover, the study focused solely on acute sleep deprivation; therefore, the long-term effects of chronic sleep restriction on autonomic function could not be assessed.

Future studies should include larger and more diverse populations, along with advanced measures such as heart rate variability analysis and biochemical markers of stress. Longitudinal study designs would be valuable in understanding the progression from acute autonomic imbalance to clinically significant cardiovascular disease.

CONCLUSIONS

The present study demonstrates that acute sleep deprivation significantly alters cardiac autonomic function, with reduced parasympathetic activity and increased sympathetic reactivity, reflecting autonomic imbalance. These changes suggest that even short-term sleep loss may impair cardiovascular regulation and increase susceptibility to hypertension and arrhythmias. Given the growing prevalence of sleep deprivation, these findings highlight the importance of adequate sleep in maintaining autonomic and cardiovascular health. Further studies with larger and more diverse populations are needed to evaluate the long-term clinical implications.

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REFERENCES

[1] Schmid SM, Hallschmid M, Jauch-Chara K, Kück MC, Lehnert H, et al. Partial sleep restriction modulates secretory activity of thyrotropic axis in healthy men. J Sleep Res., 2013; 22(2): 166–69. doi: 10.1111/jsr.12004.

[2] Mullington JM, Haack M, Toth M, Serrador JM, Meier-Ewert HK. Cardiovascular, inflammatory, and metabolic consequences of sleep deprivation. Prog Cardiovasc Dis., 2009; 51(4): 294–302. doi: 10.1016/j.pcad.2008.10.003.

[3] Somers VK, White DP, Amin R, Abraham WT, Costa F, et al. Sleep apnea and cardiovascular disease: an American Heart Association/American College of Cardiology Foundation scientific statement. J Am Coll Cardiol., 2008; 52(8): 686–717. doi: 10.1016/j.jacc.2008.05.002.

[4] Thayer JF, Lane RD. The role of vagal function in the risk for cardiovascular disease and mortality. Biol Psychol., 2007; 74(2): 224–42. doi: 10.1016/j.biopsycho.2005.11.013.

[5] 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.

[6] Freeman R. Assessment of cardiovascular autonomic function. Clin Neurophysiol., 2006; 117(4): 716–30. doi: 10.1016/j.clinph.2005.09.027.

[7] 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.

[8] Alauddin W, Alam S, Mishra M, Radke PM, Shree R, et al. A cross-sectional study of cardiovascular autonomic reactivity in Ehlers-Danlos syndrome. Cureus., 2024; 16(7): e64542. doi: 10.7759/cureus.64542.

[9] 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.

[10] Bharti I, Alauddin W, Radke PM, Patil A, Sunasra R, et al. Cardiac autonomic reactivity tests in type A and

type B personalities. Cureus, 2024; 16(2): e53690. doi: 10.7759/cureus.53690.

[11]Cheung BY, Takemura K, Ou C, Gale A, Heine SJ. Considering cross-cultural differences in sleep duration between Japanese and Canadian university students. PLoS One, 2021; 16(4): e0250671. doi: 10.1371/journal.pone.0250671.

[12]Meerlo P, Sgoifo A, Suchecki D. Restricted and disrupted sleep: effects on autonomic function, neuroendocrine stress systems and stress responsivity. Sleep Med Rev., 2008; 12(3): 197–210. doi: 10.1016/j.smrv.2007.07.007.

[13]Gangwisch JE, Heymsfield SB, Boden-Albala B, Buijs RM, Kreier F, et al. Short sleep duration as a risk factor for hypertension. Hypertension, 2006; 47(5): 833–39. doi: 10.1161/01.HYP.0000217362.34748.e0.

[14]Cappuccio FP, Cooper D, D'Elia L, Strazzullo P, Miller MA. Sleep duration predicts cardiovascular outcomes: a systematic review and meta-analysis of prospective studies. Eur Heart J., 2011; 32(12): 1484–92. doi: 10.1093/eurheartj/ehr007.

[15]Irwin MR. Sleep and inflammation: partners in sickness and in health. Nat Rev Immunol., 2019; 19(11): 702–15. doi: 10.1038/s41577-019-0190-z.

[16]Tobaldini E, Nobili L, Strada S, Casali KR, Braghiroli A, et al. Heart rate variability in normal and pathological sleep. Front Physiol., 2013; 4: 294. doi: 10.3389/fphys.2013.00294.

[17]Boudreau P, Yeh WH, Dumont GA, Boivin DB. Circadian variation of heart rate variability across sleep stages. Sleep, 2013; 36(12): 1919–28. doi: 10.5665/sleep.3230.

[18]Balint EM, Gander M, Pokorny D, Funk A, Waller C, Buchheim A. High prevalence of insecure attachment in patients with primary hypertension. Front Psychol., 2016; 7: 1087. doi: 10.3389/fpsyg.2016.01087.

[19]Cincin A, Sari I, Oğuz M, Sert S, Bozbay M, Ataş H, et al. Effect of acute sleep deprivation on heart rate recovery in healthy young adults. Sleep Breath, 2015; 19(2): 631–36. doi: 10.1007/s11325-014-1066-x.

[20]Sgoifo A, Buwalda B, Roos M, Costoli T, Merati G, Meerlo P. Effects of sleep deprivation on cardiac autonomic and pituitary-adrenocortical stress reactivity in rats. Psychoneuroendocrinol., 2006; 31(2): 197–208. doi: 10.1016/j.psyneuen.2005.06.009.