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RESEARCH ARTICLE

Effect of sleep deprivation on heart rate recovery after treadmill testing in otherwise healthy young adults

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ABSTRACT

Background: Insufficient sleep has been shown to be associated with increased cardiovascular morbidity and overall mortality. Sleep deprivation can affect the cardiovascular autonomic control leading to sympathovagal imbalance. **Aims and Objectives:** This study was conceptualized to evaluate the effect of sleep deprivation on heart rate recovery (HRR) after treadmill exercise test in otherwise healthy adult subjects. **Materials and Methods:** The study included 50 healthy young adults in the age group of 18–35 years. The study subjects were divided into two groups of 25 each. Group 1 included subjects doing daytime duty. Group 2 included subjects who had completed their night shift duty. A maximal symptom-limited exercise stress test was performed by each subject in both the groups. The HRR was calculated as the reduction in HR from peak exercise to the 30th s (HRR 30), 1st min (HRR 1), 2nd min (HRR 2), 3rd min (HRR 3), and 5th min (HRR 5). **Results:** Subjects with sleep deprivation achieved a significantly lower level of the maximum HR at the peak of exercise stress test than the subjects with normal sleep ($P < 0.05$). HRR at 30 s (HRR 30 s) and 1 min (HRR 1) was found to be significantly lower in sleep-deprived subjects as compared to subjects taking normal sleep ($P < 0.01$). **Conclusion:** HRR after 1 min of exercise stress testing was significantly affected by sleep deprivation. This is explained by the effect of sleep deprivation on the cardiovascular autonomic control.

KEY WORDS: Heart Rate Recovery; Parasympathetic; Sleep Deprivation; Stress Test

INTRODUCTION

Adequate sleep is an important determinant of normal physiological growth and well-being. Insufficient sleep has been shown to be associated with increased cardiovascular morbidity and overall mortality.[1,2] This increased risk is related to adverse effect of sleep deprivation on important cardiovascular risk factors including blood pressure, inflammation, metabolism, and hormone regulation.[3] Sleep deprivation has also been shown to have a significant association with obesity and overweight, which is a major risk factor for cardiovascular diseases.[4] Recent research has shown that sleep deprivation can affect the cardiovascular autonomic control leading to sympathovagal imbalance. Autonomic nervous system plays an important role in the regulation of cardiovascular function, and impairment in this system is associated with increased cardiovascular mortality. Recent studies have also evaluated the effects of acute sleep deprivation (e.g., due to night duty shifts) on the cardiovascular physiology and autonomic functions.[5]

Exercise testing is a cardiovascular stress test that uses treadmill exercise with electrocardiography (ECG) and blood pressure monitoring. In addition to being an established

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tool for diagnosis and prognosis of coronary artery disease (CAD), exercise stress test also provide vital information about cardiovascular physiology including blood pressure and heart rate (HR) responses. These physiological responses are dependent on the normal functioning of the cardiovascular autonomic system.

The initiation of dynamic exercise results in increases in ventricular HR, stroke volume, and cardiac output as a result of vagal withdrawal and sympathetic stimulation. During strenuous exertion, sympathetic discharge is maximal and parasympathetic stimulation is withdrawn. Recovery of the HR immediately after exercise is a function of vagal (parasympathetic) reactivation.[6] A delayed decrease in the HR during the 1st min after graded exercise, which may be a reflection of decreased vagal activity, may signify autonomic dysfunction and cardiovascular morbidity. Therefore, HR recovery (HRR) after a treadmill exercise test can be a useful clinical parameter to predict cardiovascular mortality along with other parameters of a stress test. An abnormal value for the recovery of HR was defined as a reduction of 12 beats per minute or less from the HR at peak exercise.[7]

The measurement of HRR may be affected by various technique-related factors including type of exercise protocol used.[8] It may also be affected by physiological parameters such as age, sex, and physical fitness.[9] Sleep deprivation, by its effect on autonomic nervous system and cardiovascular physiology, may affect the HRR. There is a paucity of studies which have evaluated the effect of sleep deprivation on HRR after treadmill exercise test, specifically in Indian population. Therefore, this study was done to evaluate the effect of sleep deprivation on HRR after treadmill exercise test in otherwise healthy adult subjects.

MATERIALS AND METHODS

This observational study was conducted in the Department of Physiology, Maharishi Markandeshwar Institute of Medical Sciences and Research, Mullana. The study was conducted over duration of 6 months. The study was approved by the Institutional ethics committee. The study included 50 healthy young adults in the age group of 18–35 years. The subjects for the study were taken among the health-care staff (residents, interns, and nurses) of MMIMSR hospital working on shift duty. Subjects having known chronic illness including diabetes, hypertension or CAD were excluded from the study. The study also excluded subjects with renal or hepatic dysfunction, persons with any cognitive/psychiatric illness such as schizophrenia and pregnant females. Subjects who refused consent were also excluded.

Each participant in the study was explained about the aims and objectives of the study and informed written consent for the study was taken. The study subjects were divided into two groups of 25 each. Group 1 included subjects doing daytime duty. Group 2 included subjects who had completed their night shift duty. Detailed present and medical history were recorded followed by a thorough physical examination in all the subjects. Demographic and anthropometric data including age, sex, and body mass index (BMI) of the patients were recorded. The average sleep duration in the two groups was noted. Resting HR was recorded. Blood pressure was measured in sitting position after 5 min of rest with a standard sphygmomanometer.

A maximal symptom-limited exercise stress test was performed by each subject in both the groups. The test was performed under the supervision of a physician for safety reasons, and to obtain reliable information on exercise test variables. The subjects were asked to avoid smoking, alcohol and caffeine before the testing. The procedure was done on a treadmill using the Bruce Protocol with continuous ECG monitoring. HR and blood pressure measurements were taken during each stage of the protocol and at the peak exercise. The total duration of exercise was noted for all subjects and averaged in both the groups. The subjects were encouraged to achieve the target of 85% of the age-predicted peak HR, which is calculated by 220 - age beats/min. HR reserve is calculated by formula - (Peak exercise HR – resting HR) beats/min. Any symptoms experienced by the subjects including chest pain, fatigue, and dyspnea were noted. After achieving the maximum exercise, the subjects were asked to relax in sitting position, and measurements during the recovery period were taken. The HRR was calculated as the reduction in HR from peak exercise to the 30th s (HRR 30), 1st min (HRR 1), 2nd min (HRR 2), 3rd min (HRR 3), and 5th min (HRR 5).

Statistical Analysis

The data collected were analyzed statistically to determine the effect of sleep deprivation on HRR. Variables were expressed as mean ± standard deviation (SD) and percentage. Mean differences for continuous variables between groups were examined by the independent Student *t*-test. Chi-square test (or Fisher exact test if applicable) was used for categorical variables. $P < 0.05$ was considered significant. The statistical analysis was carried out with SPSS PC software version 20.0.

RESULTS

Table 1 shows the baseline demographic and anthropometric characteristics of the two study groups. There was no statistically significant difference between the two groups with respect to age, sex, and BMI. Table 2 compares the exercise stress test parameters including blood pressure response and post-exercise HRR in the two study groups. HRR at 30 s and 1 min was significantly lower in sleep-deprived subjects as compared to the other group.

Table 1: Baseline clinical and anthropometric parameters in the two groups

Parameter	Group 1 (*n*–25)	Group 2 (*n*–25)	*P* value
Age (years)	26.60±2.74	26.16±2.81	0.577
Gender			
Males	14 (56%)	15 (60%)	0.774
Females	11 (44%)	10 (40%)	
Weight (kg)	61.92±8.08	59.12±8.21	0.231
Height (m)	1.61±0.08	1.62±0.06	0.492
BMI (kg/m^2)	23.91±3.48	22.34±2.90	0.905
Pre-exercise HR (beats/min)	78.16±7.57	77.28±6.53	0.662
Pre-exercise SBP (mm Hg)	122.48±9.84	121.2±10.13	0.652
Pre-exercise DBP (mm Hg)	75.60±6.13	74.96±5.83	0.707

BMI: Body mass index, HR: Heart rate, SBI: Systolic blood pressure, DBI: Diastolic blood pressure

Table 2: Comparing exercise stress test parameters between the two groups

Parameter	Group 1	Group 2	*P* value
Peak SBP (mm Hg)	174.16±10.45	179.84±11.07	0.068
Peak DBP (mm Hg)	87.04±6.08	87.92±5.84	0.604
Peak HR (beats/min)	166.4±6.84	161.36±7.73	0.019*
HRR at 30 sec	14.96±3.28	11.64±3.26	0.001**
HRR at 1 min	29.92±5.44	25.36±6.26	0.008**
HRR at 2 min	46.76±7.29	44.40±7.70	0.271
HRR at 3 min	57.24±8.16	56.72±8.46	0.826
HRR at 5 min	65.80±8.47	64.92±8.71	0.718

HRR: HR recovery, HR: Heart rate, SBI: Systolic blood pressure, DBP: Diastolic blood pressure. *P<0.05: Significant; **P<0.01: Highly significant

DISCUSSION

The potential value of post-exercise HRR in predicting cardiovascular morbidity and mortality has been demonstrated in large clinical studies over the years.[7,10-13] Watanabe *et al*. showed that an abnormal HRR is a powerful and independent predictor of mortality even after adjusting for age, sex, exercise capacity, presence or absence of myocardial ischemia, and left ventricular systolic function.[10] In a retrospective analysis including 6546 patients without any known cardiovascular disease who were referred for exercise testing, the association between exercise test parameters and all-cause mortality was evaluated in a referral population. The study showed that all-cause mortality significantly correlated with HRR and functional aerobic capacity.[12] A prospective cross-sectional study included 208 patients in the age group of 34–74 years showed that even after adjusting for established CAD risk factors, abnormal post-exercise HRR at 1 min correlated significantly with the extent of major epicardial coronary involvement.[14]

The effect of inadequate sleep or sleep disorders on normal cardiovascular physiology and its relation to cardiovascular disease has been documented in the previous studies.[15-17] In a systematic review and meta-analysis of prospective studies done to evaluate the role of sleep duration in cardiovascular morbidity and mortality, Cappuccio *et al*. showed that patients with sleep deprivation had a significantly higher risk of developing CAD or stroke. It also had a significant effect on cardiovascular mortality.[15]

In the present study, important effects of sleep deprivation on exercise stress test parameters were observed. Subjects with sleep deprivation achieved a significantly lower level of maximum HR at the peak of exercise stress test than the subjects with normal sleep ($P < 0.05$). HRR at 30 s (HRR 30 s) and 1 min (HRR 1) was found to be significantly lower in sleep-deprived subjects as compared to subjects taking normal sleep ($P < 0.01$). No significant difference was seen between the two groups with respect to HRR at 2nd, 3rd, and 5th min of post-exercise. Although peak systolic blood pressure achieved in sleep-deprived subjects was higher than the normal sleep group, it was not statistically significant ($P > 0.05$). Similarly, no significant difference was found between the two groups with regard to peak diastolic blood pressure ($P > 0.05$).

Our results corroborated with other studies done to evaluate the effect of sleep deprivation on HRR. In a recent study done by Cincin *et al*. on 30 healthy subjects (including 21 security officers and nine nurses), treadmill exercise test was applied once after a night with regular sleep and once after a night shift in the hospital. There was a significant difference in HRR 30 s and HRR 1 min. The ratio of these indices to peak HR was also significantly lower with SD. They concluded that sleep deprivation blunts cardiovascular autonomic response, and consequences of this relation might be more pronounced in subjects who are exposed to sleeplessness regularly or in subjects with baseline cardiovascular disease.[18] In another study done on 113 healthy subjects, the effect of sleep quality (assessed using the Pittsburgh sleep quality index questionnaire) on hemodynamic response and HRR after exercise was evaluated. The study showed that poor sleepers had significantly more frequent hypertensive response to exercise, chronotropic incompetence and reduced HRR at the 1st and 3rd min of peak exercise as compared to those with good sleep quality.[19]

These results are explained by the effect of sleep deprivation on the normal functioning of cardiovascular autonomic control leading to sympathovagal imbalance. It has been shown previously that acute sleep deprivation results in increased sympathetic and decreased parasympathetic cardiovascular modulation in normal humans.[20] In a study done to evaluate the effect of acute sleep deprivation in physicians, Tobaldini *et al*. showed that even one night of sleep deprivation may affect the autonomic control of the cardiovascular system and immune modulation, independently by the activation of the Hypothalamic-Pituitary axis.[21]

There are some limitations in our study. Since our study included only young adults, so the effect of age on HRR was not dealt with in this study. Similarly, the potential effect of the level of physical activity on the study parameters was not included. Our study did not evaluate the cumulative effect of regular night shifts on the HRR.

CONCLUSION

HRR after exercise is an important indicator of normal cardiovascular physiology. Our study showed that HRR after 1 min of exercise stress testing was significantly affected by sleep deprivation. This is explained by the effect of sleep deprivation on the cardiovascular autonomic control.

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