ORIGINAL RESEARCH

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Effect of Sleep Quality on Hemodynamic Response to Exercise and Heart Rate Recovery in Apparently Healthy Individuals

Abstract

Purpose: Poor sleep quality has an unfavorable impact on autonomic nervous system activity, especially that of the cardiovascular (CV) system. The heart rate (HR) and blood pressure (BP) at rest and during exercise, along with the heart rate recovery (HRR), were examined in poor sleepers and compared with individuals with good sleep quality.

Methods: A total of 113 healthy individuals were enrolled to the study. All participants performed treadmill stress testing. Sleep quality of participants was assessed by using the Pittsburgh Sleep Quality Index (PSQI) questionnaire: 48 subjects were categorized as 'poor sleepers' (PSQI score > 6 points), and the rest were grouped as 'good sleepers'.

Results: The poor sleepers showed higher resting HR ($p<0.001$), higher diastolic BP ($p=0.006$), similar systolic BP ($p=0.095$), more frequent hypertensive response to exercise ($p=0.046$) and less HR increase with exercise (chronotropic incompetence) ($p=0.002$) compared with individuals who reported good sleep quality. In addition, the poor sleepers demonstrated reduced heart rate recovery at the 1st and 3rd minute of recovery ($p=0.005$ and 0.037 , respectively) compared with good sleepers. Multivariate logistic regression analysis revealed that only resting diastolic BP was the independent predictor of HRE. The PSQI score was positively correlated with resting HR; while it was negatively correlated with HR response to exercise, HRR1 and HRR index-1.

Conclusion: This cross-sectional study emphasizes the effect of poor sleep quality on unfavorable cardiovascular outcome indicators of the treadmill stress test.

Manuscript submitted 1st June, 2014
Manuscript accepted 8th November, 2014

Clin Invest Med 2014; 37 (6): E352-E362.

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Sleep is an essential physiological process of healthy life which ensures restorative, adaptive, and energy-conserving functions [1]. Abnormal sleep results in increased sympathetic activity, blood pressure (BP) and heart rate (HR). Due to changes in lifestyle, people experience sleep disturbances more frequently in the modern era [2]. Short- and long-term sleep deprivation are independently associated with an increased risk of coronary events [3]. After the detection of an abnormal BP behavior during sleep, several studies revealed that changes in the autonomic nervous system activity were associated with sleep disorders. More importantly, these findings marked sleep disorders as an important risk factor for cardiovascular (CV) and cerebrovascular disorders [2,4,5].

On the other hand, a number of treadmill stress test (TST) parameters reflecting autonomic nervous system activity have been well defined; including, heart rate recovery index, exaggerated BP response and impaired chronotropic response to exercise. Even in normotensive individuals, subjects with exaggerated BP responses to exercise were reported to be more prone to develop overt hypertension (HT) and associated unfavorable CV outcomes [6,7]. Impaired HR response to exercise, also known as chronotropic incompetence, is another parameter linked to adverse clinical outcomes in healthy adults and worse prognosis in patients with coronary artery disease (CAD) [8-12]. The heart rate recovery (HRR) defines the HR as it declines during the recovery period after a stress test and is determined by parasympathetic activity [13]. Impaired HRR, especially in the first minute following exercise, was shown to be a prognostic index and independent predictor for CV and all-cause mortality [14-16]. In addition, previous studies have reported that the resting HR is an independent prognostic indicator for worse CV outcome and for mortality among both healthy populations and patients with CV diseases, as well as a positive influence for controlling morbidity and mortality in the treatment of various CV diseases [17-20].

This is the first study investigating the relationship among poor sleep quality and hemodynamic response to exercise and HRR. We investigated whether unfavorable TST parameters, including exaggerated BP response to exercise, chronotropic incompetence, and attenuated HRR, are more common in healthy individuals with poor sleep quality.

Materials and Methods

Study Population

A total of 113 normotensive consecutive healthy individuals, who were free of CV or pulmonary diseases (81 men and 32 women), who were not taking any antihypertensive or CV

medications and who underwent a TST between June 2013 and October 2013 were enrolled in the study. All subjects had normal baseline and exercise electrocardiograms (ECGs).

The Pittsburgh Sleep Quality Index (PSQI) questionnaire [21] was used to evaluate sleep quality and was completed by all of the participants after the TST. Office BP measurement and rest ECGs were recorded and a routine transthoracic echocardiography was performed on all subjects. A detailed medical history was noted. All participants gave informed consent after the nature of the procedures was explained. The study was approved by the local Ethics Committee.

Exclusion Criteria

A total of 385 individuals referred to TST were evaluated and 272 of them were excluded from the study due to following criteria: 106 with hypertension (HT) (defined as BP > 140/90 mmHg or those who were receiving anti-hypertensive medication), 21 with diabetes mellitus (defined as fasting serum glucose level above 126 mg/dL, or receiving oral anti-diabetic medication), 36 with CV disease (including coronary artery disease, congestive heart failure, congenital heart disease, moderate or severe valvular regurgitation, any degree of valvular stenosis and/or peripheral vascular disease), 12 with established chronic renal failure or serum creatinine levels ≥ 1.5 mg (132 μ mol/l), eight with chronic obstructive pulmonary disease, two with thyroid dysfunction, 18 with anemia (hemoglobin < 12 g/dL for men, < 11 g/dL for women), one with history of an implanted pacemaker, two with pre-excitation syndrome, two with left bundle branch block, 18 with ST segment depression/elevation during exercise stress test, 14 with atrial fibrillation, 12 with frequent ventricular premature beats, 15 with any medication or substance usage, including nasal decongestants and caffeine, that would affect BP or HR, and five with any medication that could potentially affect physical performance. Individuals unable to complete the TST or those with orthopedic problems that would preclude maximal effort on the treadmill were not included. Additionally individuals with a diagnosis or symptoms suggestive of psychiatric disorders that might be associated with sleep disturbance (i.e., anxiety disorder, mood disorders, psychotic disorders, eating disorders or substance use) were excluded from the study. Axis-I psychiatric disorders were scanned by a self-report symptom inventory called Symptom Checklist-90-Revised [22] (SCL-90R), which was translated to Turkish and verified by Dag *et al.* [23].

Assessment of Treadmill Stress Test

The study groups were similar in terms of exercise duration on TST, which was performed by all subjects on a treadmill (Quinton Treadmill System, Quinton, Bothell, WA, USA) in the morning according to the Bruce protocol [24]. A 12-lead ECG was obtained throughout the test. Resting SBP and DBP were the average of three measurements taken in a seated position by a cardiologist. All subjects completed the exercise stress tests without new-onset rhythm abnormalities, ST-segment deviations on ECG or other complications. Exercise BP values were measured during the last minute of each 3-minute stage and at peak exercise, and at 3rd and 5th minutes after the cessation of exercise, using an indirect arm-cuff oscillometric sphygmomanometer on the right arm. Exercise capacity (in minutes) was recorded as peak exercise time. Peak exercise workload was estimated on the basis of the speed and grade of the treadmill and recorded as metabolic equivalents (1 MET equals 3.5 ml of oxygen uptake per kilogram of body weight per minute). Target HR was determined as 85% of the age-predicted maximal HR (APMHR). Age-predicted maximal HR was defined as 220 minus age. Subjects were encouraged to exercise as much as they could, in order to achieve at least the target HR in the absence of symptoms or other indicators of ischemia. The hypertensive response to exercise (HRE) was defined as a peak exercise SBP $\geq$ 210 mmHg in men, or SBP $\geq$ 190 mmHg in women or DBP 100 mmHg in all subjects, in line with the Framingham criteria [25]. Heart rate response to exercise was defined as the difference between HR at peak exercise and HR at rest. Heart rate recovery was defined as the reduction in HR from the rate at peak exercise to the rate at the first, third and fifth minutes after cessation of exercise. These results were expressed as HRR₁, HRR₃, and HRR₅, respectively. Heart rate recovery index was defined as the HRR 1st, 3rd and 5th minutes divided by HR at peak exercise and expressed as HRR index-1, HRR index-3 and HRR index-5.

Pittsburgh Sleep Quality Index

Just after completion of the TST, all participants were asked to complete a self-report questionnaire (the Turkish version of the Pittsburgh Sleep Quality Index; PSQI) examining the sleep quality of an individual over the last month. The PSQI consists of a scale with 19 items and measures seven components of sleep quality: (i) subjective sleep quality, (ii) sleep latency, (iii) sleep duration, (iv) habitual sleep efficiency, (v) sleep disturbances, (vi) use of sleeping medication and (vii) day-time dysfunction. A global PSQI score corresponding to the sum of individual scores from the seven components (ranges between

0 - 21 points). A PSQI score $\geq$ 6 points is considered to be indicative of poor sleep quality. The test has a high diagnostic specificity for detecting clinical sleep impairment. The reliability and validity of the Turkish version of this questionnaire has been verified by Agargun *et al.* [26].

Statistical Analysis

The normality of distribution for continuous variables was confirmed with the Kolmogorov-Smirnov test. Normally distributed continuous variables were defined as means and standard deviation, and as median and minimum-maximum ranges for the parameters not normally distributed; categorical variables were given as percentages. The independent samples Student's-t test, or the Mann-Whitney U test, were used for the continuous variables, and the Pearson's chi-square test or Yates' chi-squared test were used for categorical variables. Correlations were assessed by using either Pearson or Spearman rank tests if the distribution pattern of the variable was compatible with normal or not normal, respectively. The independent variables that were significantly correlated with PSQI score were also further evaluated with multivariate linear regression analysis. A multivariate logistic regression analysis was conducted to explore the independent predictors of HRE. All p values were calculated assuming 2-sided alternative hypotheses; p values <0.05 were taken to indicate statistically significant comparisons.

Results

A total of 113 individuals were enrolled to the study and 72% (81 subjects) of the study population was male. Mean age of the participants was 41.0 ± 10.7 years. The total study population was divided into two groups according to their PSQI scores: 48 participants were categorized as poor sleepers (PSQI score $\geq$ 6 points) and the 65 participants with PSQI score < 6 points were categorized as good sleepers. The median of total PSQI score of poor sleepers was 8 points (range 6 -15 points) and that of good sleepers was 4 points (range 2 - 5 points).

Basic characteristics of the population sample, including age, gender, smoking habit, waist circumference, BMI and total exercise time, as well as hematologic and biochemical analyses, were similar between the two groups (Table 1). Hemodynamic response to exercise, including SBP, DBP and HR, are listed in Table 2. Hypertensive response to exercise was more frequently seen among poor sleepers (27.1% *vs.* 12.3%, $p=0.046$; Table 2). The course of SBP and HR before, during, and after exercise is demonstrated in Figures 1 and 2, respectively.

TABLE 1. Baseline characteristics and laboratory measurements of study population

	Good Sleepers (n=65)	Poor Sleepers (n=48)	<i>p</i> value
Age, years	40.6 ± 11.1	41.6 ± 10.3	0.612
Male, n (%)	47 (72)	34 (71)	1.00
BMI, kg/m ²	26.6 ± 3.4	27.4 ± 4.8	0.277
Waist circumference, cm	86.6 ± 13.4	88.9 ± 14.8	0.372
Smoking	24 (37)	27 (56)	0.064
Total PSQI score, median (range)	4 (2-5)	8 (6-15)	
Hg, g/dL	14.1 ± 1.4	14.5 ± 1.5	0.204
Hematocrite, %	41.6 ± 3.8	42.4 ± 4.2	0.342
WBC, x10 ³	8.52 ± 2.06	8.46 ± 2.21	0.892
Platelet, x10 ³	263 ± 71	260 ± 78	0.841
Glucose, mg/dL	93 ± 9	95 ± 11	0.425
Total cholesterol, mg/dL	171 ± 40	168 ± 33	0.699
Triglycerides, mg/dL	123 ± 75	147 ± 70	0.155
HDL, mg/dL	42 ± 7	44 ± 8	0.388
LDL, mg/dL	102 ± 29	96 ± 28	0.272

Abbreviations: BMI, body mass index; HDL, high-density lipoprotein; Hg, haemoglobin; LDL, low-density lipoprotein; PSQI, Pittsburgh Sleep Quality Index; WBC, white blood cell count.

All subjects were normotensive as shown by office BP measurements. There was no significant difference in resting systolic BP (SBP) between groups, while diastolic BP was higher in poor sleepers ($p=0.006$; Table 2). Systolic BP at the end of stage 1 and 2 were significantly higher in poor sleepers ($p=0.046$, and $p=0.008$, respectively). SBP at peak exercise, increase in SBP with exercise, and decrease of SBP from peak exercise to recovery at 3rd and 5th minutes were comparable between the two groups (Table 2). In addition, SBP at peak exercise was significantly correlated with SBP at rest ($r=0.572$, $p<0.001$) and DBP at rest ($r=0.413$, $p<0.001$). In multivariate logistic regression analyses, only resting DBP was an independent predictor of HRE (Table 3).

Resting HR was significantly higher in poor sleepers and positively correlated with PSQI score. Multivariate linear regression analysis revealed that age, total PSQI score and HRR₁ are independently associated with resting HR (Table 4). Heart rate response to exercise was significantly lower in poor sleepers

and was negatively correlated with total PSQI score (Table 5); while HR at peak exercise was comparable between the two groups (Table 2). Only age and resting HR were independently associated with HR response to exercise in the multivariate linear regression analysis (Table 5). HRR₁, and HRR₃ were significantly lower in poor sleepers ($p=0.005$ and $p=0.037$, respectively) and HRR₅ tended to be lower in poor sleepers but the difference did not reach statistical significance ($p=0.053$; Table 2). HRR₁ and HRR index-1 showed a significant negative correlation with PSQI score. Multivariate linear regression analysis revealed that age, BMI, resting HR and total PSQI score were the independent variables associated with HRR₁; whereas age and BMI were the only variables independently associated with HRR index-1 (Table 6). Figure 3 demonstrates the correlation between PSQI score and resting HR, HR response to exercise, HRR₁ and HRR index-1 on scatter dot graphs.

Smoking tended to be more common among poor sleepers (37% vs. 56%, $p=0.064$). Smokers and non-smokers were compared within each group and among all participants in terms of hemodynamic parameters; including, resting HR, HR response to exercise, HRR-1, HRR index-1, total exercise time, age and BMI: there were no differences between smokers and non-smokers for these parameters.

Discussion

A good sleep is essential to refresh the body and to decrease sympathetic tonus, BP, HR and cardiac workload. This study suggests that resting HR was higher; while HR response to exercise, HRR₁ and HRR index-1 were lower among poor sleepers. The total PSQI score was positively correlated with resting HR, while it was negatively correlated with HR response to exercise, HRR₁ and HRR index-1. In multivariate linear regression analysis, the total PSQI score was independently associated with resting HR, HR response to exercise and HRR₁. Additionally, DBP at rest was higher among poor sleepers, whereas SBP at rest, at peak exercise and SBP decrease after cessation of exercise were comparable between the two groups. The presence of a higher resting HR, DBP and exaggerated SBP response to exercise at early stages may be related to increased sympathetic activity and decreased parasympathetic tonus secondary to the limited duration of restful sleep among poor sleepers. The statistically significant difference in SBP at early exercise stages between groups was not seen at the peak exercise stage, although the difference in SBP at peak exercise may reach significance with a larger study sample.

Diastolic BP may not be a reliable indicator for BP response to exercise since it may decrease with exercise due to

FIGURE 1. Systolic blood pressure course during treadmill stress test.

FIGURE 2. Heart rate course during treadmill stress test.

TABLE 2. Data Showing Hemodynamic Response to Treadmill Stress Test

	Good Sleepers (n=65)	Poor Sleepers (n=48)	<i>p</i> value
Total exercise time, min.	8.9 ± 1.6	8.5 ± 1.5	0.172
Resting HR, beat/min	80 ± 13	91 ± 17	<0.001
Stage 1 HR, beat/min	108 ± 15	115 ± 23	0.08
Stage 2 HR, beat/min	121 ± 17	132 ± 19	0.003
Stage 3 HR, beat/min	138 ± 17	148 ± 17	0.005
Peak exercise HR, beat/min	156 ± 13	158 ± 15	0.481
HR increase with exercise, beat/min	76 ± 15	67 ± 15	0.002
Recovery 1 st min HR, beat/min	118 ± 16	126 ± 19	0.013
Recovery 3 rd min HR, beat/min	95 ± 12	102 ± 17	0.017
Recovery 5 th min HR, beat/min	89 ± 11	96 ± 15	0.011
HRR ₁ , beat/min	38 ± 11	32 ± 13	0.005
HRR ₃ , beat/min	61 ± 12	56 ± 14	0.037
HRR ₅ , beat/min	67 ± 13	62 ± 13	0.053
Rest SBP, mmHg	121 ± 15	127 ± 17	0.095
Rest DBP, mmHg	75 ± 11	81 ± 13	0.006
Stage 1 SBP, mmHg	142 ± 20	150 ± 23	0.046
Stage 1 DBP, mmHg	75 ± 12	80 ± 12	0.034
Stage 2 SBP, mmHg	151 ± 20	163 ± 25	0.008
Stage 2 DBP, mmHg	74 ± 12	78 ± 12	0.046
Stage 3 SBP, mmHg	162 ± 19	170 ± 26	0.064
Stage 3 DBP, mmHg	75 ± 12	79 ± 13	0.101
Peak exercise SBP, mmHg	169 ± 20	175 ± 25	0.186
Peak exercise DBP, mmHg	78 ± 12	82 ± 12	0.103
SBP increase with exercise, mmHg	48 ± 18	48 ± 19	0.893
SBP recovery 1 st minute, mmHg	155 ± 21	160 ± 25	0.220
SBP recovery 3 rd minute, mmHg	143 ± 18	149 ± 20	0.091
SBP recovery 5 th minute, mmHg	136 ± 18	142 ± 20	0.064
HRE, n (%)	8 (12.3)	13 (27.1)	0.046

Abbreviations: DBP, diastolic blood pressure; HR, heart rate; HRE, hypertensive response to exercise; HRR, heart rate recovery; SBP, systolic blood pressure.

FIGURE 3. Scatter dot graphs showing correlation between total Pittsburgh Sleep Quality Index (PSQI) score and resting heart rate (HR), HR response to exercise, heart rate recovery (HRR) 1st minute (HRR-1) and HRR index-1.

peripheral arterial vasodilatation, which results in decreased peripheral vascular resistance. In addition, SBP was higher in poor sleepers at early stages of the TST. Therefore, only SBP was considered as an indicator of BP response to exercise.

Poor sleep quality may lead to hypertension through extended exposure to raised 24-hour BP and HR, elevated sympathetic nervous system activity and increased salt and water retention due to adrenergic hormones. In addition, poor sleep quality was found to be an independent predictor of non-dipping HT in newly diagnosed hypertensive and even in normotensive subjects. A threefold increase in non-dipper HT was also shown in poor sleepers compared with good sleepers [27,28],

In a healthy body, HR is expected to be low at rest, adequately increase with exercise and then decrease towards resting levels soon after the cessation of exercise. Previous studies have demonstrated the association of resting HR and attenuated exercise-induced HR increase with CV mortality [20,29,30]. In a multicenter large-scale prospective study by Dyer *et al.*, resting HR was found to be a significant risk factor for sudden cardiac death and non-cardiac death, after controlling for traditional CV risk factors [17]. In the Framingham Heart Study, involving 5,070 subjects (free of CV disease at entry into study), all-cause, CV and coronary mortality rates were found to increase progressively in relation to antecedent HRs over 30 years of follow-up [20]. In our study population, poor sleepers

TABLE 3. Multivariate logistic regression analysis of independent variables to predict hypertensive response to exercise

Variables	Odds ratio	95% Confidence interval	p value
Age, years	1.002	0.933 – 1,077	0.953
Gender (male)	1,830	0.419 – 8.003	0.422
Smoking	0.348	0.095 – 1.271	0.110
PSQI score	0.989	0.782 – 1.252	0.929
Exercise duration	1.218	0.597 – 2.483	0.588
Resting SBP, mmHg	1.041	0.986 – 1.098	0.148
Resting DBP, mmHg	1.086	1.016 – 1.160	0.015

Abbreviations: PSQI, Pittsburgh Sleep Quality Index; SBP, systolic blood pressure; DBP, diastolic blood pressure.

TABLE 4. Correlation and linear regression analyses to evaluate the association and relative importance of independent variables on resting HR

Independent variables	Pearson Correlation Analysis		Linear Regression Analysis	
	r	p value	β coefficient	p value
Age, years	-0.087	0.361	-0.312	0.006
BMI, kg/m ²	0.112	0.242	0.016	0.860
Total PSQI score	0.301	0.001	0.199	0.031
Exercise duration, min	-0.001	0.990	-0.042	0.696
HRR ₁ , beat/min	-0.366	<0.001	-0.412	<0.001

Abbreviations: BMI, body mass index; HR, heart rate; HRR₁, heart rate recovery 1st minute; PSQI, Pittsburgh sleep quality index

had significantly higher resting HR, which exposed them to an increased cardiac workload and may be an important indicator for unfavorable future CV outcomes, compared with good sleepers. Additionally, total PSQI score was positively correlated and independently associated with resting HR, after adjustment for age and BMI.

An attenuated HR response to exercise has been shown to be predictive of all-cause mortality and risk of CAD, even after accounting for age, physical fitness and standard CV risk factors [9,10,30-32]. The prognostic implication of attenuated HR response was apparent even with a minor decrease in the HR response to maximal exercise [32]. In a prospective study by Sandvik *et al.*, both HR increment with exercise and maxi-

TABLE 5. Correlation and Linear regression analyses to evaluate the association and relative importance of independent variables on HR response to exercise

Independent variables	Pearson Correlation Analysis		Linear Regression Analysis	
	r	p value	β coefficient	p value
Age, years	-0.436	<0.001	-0.485	<0.001
BMI, kg/m ²	-0.134	0.159	0.089	0.143
Total PSQI score	-0.245	0.009	0.027	0.662
Exercise duration, min	0.334	<0.001	0.094	0.192
Resting HR, beat/min	-0.607	<0.001	-0.682	<0.001

Abbreviations: BMI, body mass index; HR, heart rate; HRR₁, heart rate recovery 1st minute; HRR index-1, heart rate recovery index 1st minute; min, minute; PSQI, Pittsburgh sleep quality index.

TABLE 6. Correlation and Linear regression analyses to evaluate the association and relative importance of independent variables on HRR₁ and HRR index-1

Independent variables	Pearson Correlation Analysis		Linear Regression Analysis	
	r	p value	β coefficient	p value
Age ^a , years	-0.422	<0.001	-0.415	<0.001
Age ^b , years	-0.244	0.009	-0.255	0.011
BMI ^a , kg/m ²	-0.362	<0.001	-0.218	0.007
BMI ^b , kg/m ²	-0.366	<0.001	-0.234	0.005
Resting HR ^a , beat/min	-0.366	<0.001	-0.366	<0.001
Resting HR ^b , beat/min	-0.457	<0.001	-0.401	<0.001
Total PSQI score ^a	-0.309	0.001	-0.173	0.034
Total PSQI score ^b	-0.300	0.001	-0.162	0.054
Exercise duration ^a , min	0.248	0.008	0.076	0.421
Exercise duration ^b , min	0.130	0.171	-0.107	-0.275

^a for HRR₁, ^b for HRR index-1

Abbreviations: BMI, body mass index; HR, heart rate; HRR₁, heart rate recovery 1st minute; HRR index-1, heart rate recovery index 1st minute; min, minute; PSQI, Pittsburgh sleep quality index

mal exercise-induced HR were found to be strong, graded, long-term predictors of CV mortality among apparently

healthy middle-aged men, independent of conventional coronary risk factors [11]. Girotra *et al.*, demonstrated that impaired chronotropic response during a 6-minute walking test was associated with an increased risk of mortality and coronary heart disease among an elderly population consist of 2,224 individuals [33]. In the present study, poor sleepers showed attenuated HR response to exercise compared with good sleepers, which may implicate an increased future CV risk due to poor sleep quality. Total PSQI score was negatively correlated with HR response to exercise, indicating poorer HR response with poorer sleep quality.

Poor sleep quality has a negative impact on autonomic functions, which can be indirectly assessed with HRR and HRR index [14]. A delayed decrease in the heart rate during the first minute after graded exercise, which may reflect decreased vagal activity, is a powerful predictor of overall mortality (independent of workload), the presence or absence of myocardial perfusion defects and changes in HR during exercise. Previous large scale, prospective studies suggest that impaired HRR predicts mortality [15,16,34], even after accounting for ischemia, [34,35] chronotropic incompetence, [15,16,34,36] and the Duke treadmill score [36]. In addition, Hargens *et al.* found that obstructive sleep apnea causes alterations in the CV response after cessation of exercise, which is reflected in an attenuated HRR, and may indicate an imbalance in the autonomic regulation of HR [37]. In our study population, HRR₁ and HRR₃ were significantly lower, while HRR₅ was tended to be lower in the poor sleeper group. These results suggest that poor sleepers may be under increased risk of future CV events compared with good sleepers. Also, total PSQI score was independently associated with HRR₁ after adjustment for age, BMI and resting HR value.

Previous studies proposed that an exaggerated BP response during exercise is predictive of the development of hypertension, independent of other important risk factors. It is a valuable means of identifying normotensive individuals at high risk for developing hypertension and further end-organ damage risk secondary to HT [38-40]. In addition, an association between exaggerated BP response to exercise and masked HT has been reported [7]. In a recent study, normotensive men with metabolic syndrome, but with exercise-induced HT, were found to have greater insulin resistance and lower HR variability indices [41]. In our study population, poor sleepers showed higher SBP response to exercise compared with good sleepers at the early stages of exercise, which may indicate an increased risk of future HT development. In addition, HRE was found to be more common among poor sleepers, which may prove useful for CV risk assessment. Also, resting DBP was found to

be the only independent variable for predicting HRE in the multivariate logistic regression analysis. Future studies with larger sample population may clarify the association between sleep quality and HRE.

Study Limitations

The study was conducted at a single center and included a relatively small number of patients. Another limitation was its cross-sectional design. The use of a subjective, self-report questionnaire for evaluation of sleep quality may have a risk of decreased objectivity in categorization of the participants as poor sleepers. Further large-scale, prospective studies using objective measures of sleep quality are needed to confirm the association between poor sleep quality and unfavorable hemodynamic parameters for adverse CV events.

Conclusion

This cross-sectional study emphasizes the effect of poor sleep quality on unfavorable CV outcome indicators of TST.

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