

## Impact of acute sleep deprivation on aortic elastic properties in healthy workers

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**Aim:** People are more frequently exposed to acute sleep loss nowadays due to extensive working. Epidemiologic studies suggest that sleep deprivation (SD) increases the risk of cardiovascular events. Aortic stiffness is also shown to predict cardiovascular events, stroke, and death even in healthy populations. The association between SD and aortic stiffness remains unknown. We sought to determine the impact of acute SD on aortic elastic properties in healthy workers.

**Materials and methods:** The study population consisted of 34 healthy medical personnel working in shifts (17 men and 17 women). Less than half of the mean daily sleep time, or less than 4 hours a day, was defined as SD. Blood pressure and echocardiographic measurements for aortic stiffness including B-index, strain, and distensibility were obtained before and after SD.

**Results:** Systolic blood pressure of participants increased significantly after SD compared to control values, which were recorded after normal sleep. There was also a statistically significant difference in diastolic blood pressures ( $118.29 \pm 13.17$  vs.  $119.52 \pm 11.94$ ,  $P = 0.001$ ;  $70.70 \pm 12.04$  vs.  $71.08 \pm 9.51$ ,  $P = 0.029$ ; respectively). There was a statistically significant decrease in distensibility after SD compared to basal values ( $4.80 \pm 2.88$  vs.  $4.42 \pm 2.49$ ,  $P = 0.042$ ; respectively). The association between acute SD and B-index and strain did not reach statistical significance ( $11.18 \pm 6.22$  vs.  $12.25 \pm 6.24$ ,  $P = 0.064$ ;  $46.44 \pm 43.71$  vs.  $48.17 \pm 35.09$ ,  $P = 0.07$ ; respectively).

**Conclusion:** Acute SD triggers an increase in blood pressure and is associated with increased aortic stiffness.

**Key words:** Sleep deprivation, blood pressure, aortic stiffness, echocardiography

### 1. Introduction

Sleep is essential for metabolic and physiologic regulation and is also known for its positive effects on resting and restoration of the body. During sleep, sympathetic tone regresses and the parasympathetic system is activated in the cardiovascular system. This change is reflected by high frequency rhythms on heart rate variability (1) and a dipper effect on blood pressure (2). Along with these effects on the cardiovascular system, healthy sleeping habits also have favorable effects on glucose metabolism and the immune system (3).

Nowadays, people are more frequently exposed to acute sleep deprivation (SD) due to extensive working. A disruption in neurohormonal mechanisms caused by SD further deteriorates multiple systems including cognitive, metabolic, and immune, and many other vital functions. Studies suggest that SD increases the risk of diabetes and hypertension (4). In addition, decreased duration of sleep is shown to be independently associated with coronary events in epidemiologic studies (5). The association of

acute SD and QT duration and P wave dispersion, which are accepted as predictors of cardiovascular events, has already been shown by studies performed in young healthy volunteers in our clinic (6,7).

The tunica media layer of the aorta is composed of tissue rich in elastic fibers. This is an important feature for the continuity of the circulatory system. Vast arrays of factors are known to disrupt this elasticity. Impairment in aortic elasticity is an independent predictor of cardiovascular mortality in hypertensive subjects (8). Coronary artery disease (9), chronic kidney disease (10), diabetes (11), connective tissue disease (12), obstructive sleep apnea syndrome (13), and neurocardiogenic syncope (14), which was revealed by our previous study, are all diseases in which disruption in aortic elasticity was documented. There is also an associated poor prognosis in these diseases. Based on the literature, and to the best of our knowledge, this is the first study to evaluate the effects of acute SD on aortic elastic properties.

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## 2. Methods

### 2.1. Study population

Thirty-four medical personnel (17 men and 17 women) in our hospital were enrolled in the study. Volunteers were all free from acute or chronic diseases and were not taking any medications. Study investigators evaluated the healthy volunteers with detailed physical examinations and laboratory workups. Subjects with hypertension, cardiovascular disease, diabetes, cerebrovascular disease, chronic obstructive pulmonary disease, connective tissue disease, or psychiatric disease were excluded. Tobacco and alcohol consumption were also exclusion criteria. Volunteers were instructed not to consume caffeinated beverages for the duration of the study. Local ethics committee approval was obtained before enrollment.

### 2.2. Study protocol

Less than half of the mean daily sleep time, or less than 4 hours a day, was defined as SD. The study population consisted of medical personnel working in shifts. The working hours of these personnel were between 1600 and 0800. Between 0800 and 1600, these subjects slept for a maximum of 3.5 h. Basal blood pressure and echocardiographic measurements were obtained at 1600. After 16 h of work, control blood pressure and echocardiographic measurements were obtained again at 0800 and B-index, strain, and distensibility were calculated as markers of aortic stiffness.

### 2.3. Calculation of aortic stiffness

Aortic stiffness was calculated noninvasively by beat-to-beat changes in aortic diameter in association with blood pressure. M-mode measurements were obtained in parasternal long axis view 2–3 cm above the aortic valve. Aortic systolic and diastolic diameters were measured from anterior and posterior innermost borders. Systolic diameters were measured while the aorta was foremost in motion, and diastolic diameters were measured during the peak of the QRS complex of the surface electrocardiography recorded simultaneously. Measurements were recorded in millimeters. Blood pressures were recorded at the same time with an external sphygmomanometer in the supine position and recorded as mmHg.

Strain (15), B-index, and distensibility (16) were used as parameters of aortic stiffness. The formulae used to calculate these parameters were as follows:

$$\text{Strain (\%)} = (\text{systolic diameter} - \text{diastolic diameter}) \times 100 / \text{diastolic diameter}$$

$$\text{Beta index} = \ln (\text{systolic pressure} / \text{diastolic pressure}) / \text{strain}$$

$$\text{Distensibility (cm}^2 \text{ dyn}^{-1}\text{)} = 2 \times (\text{strain}) / (\text{systolic blood pressure} - \text{diastolic blood pressure})$$

### 2.4. Statistics

Statistical analysis was performed using SPSS 16.0 and findings were recorded as mean  $\pm$  standard deviation. Descriptive statistical methods (mean, standard deviation) were used in the evaluation of the study data. When comparing quantitative data, if the number of the data set was greater than 30, it was accepted as having a normal distribution. A paired sample test was used when comparing dependent parameters and Student's t test was used when comparing nondependent parameters. The Mann–Whitney U test was used with both sexes. Results were recorded with 95% confidence interval and statistical significance was accepted if  $P < 0.05$ .

## 3. Results

There were 17 men and 17 women in the study. Mean age was  $28.9 \pm 3.5$  for men and  $26.2 \pm 3.4$  for women. Systolic blood pressure of participants increased significantly after SD compared to control values, which were recorded after normal sleep. There was no difference between the sexes in terms of SD ( $P = 0.35$ ). There was also a statistically significant difference in diastolic blood pressures ( $118.29 \pm 13.17$  vs.  $119.52 \pm 11.94$ ,  $P = 0.001$ ;  $70.70 \pm 12.04$  vs.  $71.08 \pm 9.51$ ,  $P = 0.029$ ; respectively).

There was a statistically significant decrease in distensibility after SD compared to basal values ( $4.80 \pm 2.88$  vs.  $4.42 \pm 2.49$ ,  $P = 0.042$ ; respectively). The association between acute SD and B-index and strain did not reach statistical significance ( $11.18 \pm 6.22$  vs.  $12.25 \pm 6.24$ ,  $P = 0.064$ ;  $46.44 \pm 43.71$  vs.  $48.17 \pm 35.09$ ,  $P = 0.07$ ; respectively) (Table).

## 4. Discussion

Working all day long gives rise to SD and triggers many metabolic and physiologic abnormalities. To our knowledge, this is the first study in which the effects of acute SD on aortic stiffness in healthy workers were investigated. Our findings suggest acute partial SD triggers a substantial increase in both systolic and diastolic blood pressures, and there was a significant association between acute partial SD and distensibility as a parameter of aortic stiffness. There was also a correlation between the other parameters of aortic stiffness, strain, and B-index. However, this association did not reach statistical significance.

Aortic stiffness is shown to be an independent predictor of mortality in many systemic diseases, particularly in cardiovascular diseases. Laurant et al. (8) revealed that aortic stiffness is an independent predictor

**Table.** Blood pressure and aortic elastic parameters before and after sleep deprivation.

	Sleep deprivation absent	Sleep deprivation present	P
Aortic strain (%)	11.18 ± 6.22	12.25 ± 6.24	0.070
Aortic B-index	46.44 ± 43.71	48.17 ± 35.09	0.064
Aortic distensibility (cm <sup>2</sup> dyn <sup>-1</sup> )	4.80 ± 2.88	4.42 ± 2.49	0.042
Systolic blood pressure (mmHg)	118.29 ± 13.17	119.52 ± 11.94	0.001
Diastolic blood pressure (mmHg)	70.70 ± 12.04	71.08 ± 9.51	0.029

of cardiovascular and all-cause mortality in hypertensive patients. European Society of Cardiology (ESC) clinical practice guidelines on hypertension strongly recommend measuring aortic stiffness in the evaluation of target organ damage (17). Şatıroğlu et al. (18) showed coronary atherosclerosis extension is correlated with impairment of aortic elasticity. In addition, aortic stiffness is also shown to predict cardiovascular events, stroke, and death even in healthy populations (19).

Epidemiological studies suggest an association between SD and cardiovascular events. The American Cancer Society (20) stated that sleeping less than 4 h a night is associated with increased risk of acute myocardial infarction (MI). Liu et al. (21) also showed a 2.3-fold increase in myocardial infarction risk in men sleeping less than 5 h compared to those who sleep 6–8 h a day. Ayas et al. (5) concluded that sleeping less than 8 h is associated with increased risk of cardiovascular events in the Nurse Health Study, which enrolled 71,617 women between 45 and 65 years of age. In our study, acute SD was significantly associated with aortic stiffness as a surrogate for cardiovascular events. We can conclude that professions that expose people frequently to sleep loss can be considered a high risk for cardiovascular events.

Soreca et al. (22) showed an increased prevalence of hypertension in patients older than 40 years exposed to SD. Fujikawa et al. (23), in their study that enrolled 331

healthy young adults, revealed an association between acute SD and increased blood pressure. Increased sympathetic tone is thought to mediate this association (24). Reduction in blood pressure at night (dipping) by depressed sympathetic tone is attenuated by SD, especially in healthy subjects (2). High frequency heart rate patterns seen in healthy subjects are replaced by low frequency heart rate patterns after SD, which indicates increased sympathetic system activation (1). In our study, there was a substantial increase in both systolic and diastolic blood pressure after SD compared to basal values. Furthermore, increase in blood pressure in these patients is primarily responsible for aortic stiffness, which is expected after frequent episodes of SD.

The main limitation of our study is the relatively low number of participants. This is the reason why the association between SD and B-index and strain did not reach statistical significance despite the statistical tendency. In addition, this was a cross-sectional study. The effect of acute SD on cardiovascular outcomes in the long term needs further validation with prospective studies.

In conclusion, acute SD triggers an increase in blood pressure and is associated with aortic stiffness, which is an independent predictor of cardiovascular events. Our study also suggests that professions that expose subjects frequently to SD should be accepted as a high risk for cardiovascular events.

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