GMJ 2024;35:168-172

DOI: http://dx.doi.org/10.12996/gmj.2023.3787

A Speckle-Tracking Echocardiography Study: Cardiac Effect of Sleep Duration

Speckle-Tracking Ekokardiyografi Çalışması: Uyku Süresinin Kalp Üzerine Etkileri

Gülsüm Bingöl[1], Fulya Avcı Demir[2], Özge Özden[1], Emre Özmen[3], Serkan Ünlü[4]

[1]Memorial Bahçelievler Hospital, Clinic of Cardiology, İstanbul, Türkiye
[2]Medical Park Antalya Hospital, Clinic of Cardiology, Antalya, Türkiye
[3]Siirt Training and Research Hospital, Clinic of Cardiology, Siirt, Türkiye
[4]Gazi University Faculty of Medicine, Department of Cardiology, Ankara, Türkiye

ABSTRACT

Objective: Sleep is a complex event involving various physiological processes. Recent studies during the last few years have shed light on the effects of sleep duration on health. We investigated the cardiac effects of sleep duration on speckle-tracking echocardiography. We divided the patients into 2 groups who slept more or less than 7 hours and evaluated all of them with transthoracic echocardiography.

Methods: This study was a retrospective study was conducted to investigate the cardiac effects of sleep duration. Between June 2021 and February 2022, 129 individuals who were admitted to our hospital for routine check-up with no known chronic disease and with normal and good quality echocardiographic examination were included in the study. The patients who were eligible for the study design were called by the investigator to obtain information on average sleep duration, and two groups were defined by the self-reported sleep duration (≤7 hours, >7 hours per day) and were used in the statistical analyses.

Results: A total of 129 patients [≤7 hours (n=80), >7 hours (n=49) per day] were enrolled in this study. All echocardiographic parameters were compared with sleep duration. Among these parameters, left ventricular (LV) global longitudinal strain (GLS) was found to be greater in the group who slept less. (-21.5±2.6 vs. -20.3±2.2 p=0.005). However, being within normal limits, LV end diastolic diameter and right heart chambers were found to be more enlarged in those who slept 7 hours or less than 7 hours. It was also observed that the right atrial volume was higher in the less sleeper group

Conclusion: As a result, GLS, which is one of the parameters showing LV function, was found to be less in healthy people with a sleep time above a certain period, and we believe that this may be a hypothesis to explain the relationship of long sleep time with cardiovascular events.

Keywords: Echocardiography, sleep, speckle-tracking imaging

ÖZ

Amaç: Uyku, çeşitli fizyolojik süreçleri içeren karmaşık bir olaydır. Yakın zamanda yapılan çalışmalar, uyku süresinin sağlık üzerindeki etkilerine ışık tutmuştur. Biz de çalışmamızda uyku süresinin kalpte speckle-tracking ekokardiyografi bulguları üzerine etkisini araştırdık. Hastaları 7 saatten fazla uyuyan ve az uyuyan olarak 2 gruba ayırdık ve hepsini transtorasik ekokardiyografi ile değerlendirdik.

Yöntemler: Bu çalışma, uyku süresinin kardiyak etkilerini araştırmak amacıyla yapılan retrospektif bir çalışmadır. Hastanemize Haziran 2021-Şubat 2022 tarihleri arasında kontrol amaçlı başvuran, bilinen kronik bir hastalığı olmayan, normal ve görüntü kalitesi iyi ekokardiyografiye sahip 129 kişi çalışmaya alındı. Çalışmaya dahil edilmeye uygun hastalar, ortalama uyku süresi hakkında bilgi almak amacıyla bir araştırmacı tarafından arandı ve hastalar kendilerinin sözel olarak belirttiği uyku sürelerine istatistiksel analizlerde kullanılmak üzere iki gruba (günde ≤7 saat, >7 saat) ayrıldı.

Bulgular: Bu çalışmaya toplam 129 hasta [günde ≤7 saat (n=80), >7 saat (n= 49)] alındı. Tüm ekokardiyografik parametreler uyku süresi ile karşılaştırıldı. Bu parametreler arasında sol ventrikül (LV) global longitudinal strain (GLS) daha az uyuyan grupta daha fazla bulundu (-21,5±2,6 vs. -20,3±2,2 p=0,005) Ayrıca LV end diyastolik çapı ve sağ kalp boşluklarının çapları 7 saat veya 7 saatten az uyuyanlarda daha fazla genişlemiş olarak bulundu. Yine daha az uyuyan grupta sağ atriyal hacmin daha yüksek olduğu gözlendi.

Sonuç: Sonuç olarak, LV fonksiyonunu gösteren parametrelerden biri olan GLS değerinin, uyku süresi belirli bir sürenin üzerinde olan sağlıklı kişilerde daha az olduğunu saptadık. Bunun uzun uyku süresinin kardiyovasküler olaylarla ilişkisini açıklamak için bir hipotez olabileceğini düşünmekteyiz.

Anahtar Sözcükler: Ekokardiyografi, uyku, speckle-tracking görüntüleme

Address for Correspondence/Yazışma Adresi: Gülsüm Bingöl MD, Memorial Bahcelievler Hospital, Clinic of Cardiology, İstanbul, Türkiye
E-mail / E-posta: bulut_gulsum@hotmail.com
ORCID ID: orcid.org/0000-0001-5879-7866

Received/Geliş Tarihi: 05.01.2023
Accepted/Kabul Tarihi: 04.04.2023

INTRODUCTION

Sleep is a complex event involving various physiological processes. Recent studies during the last few years have shed light on the effects of sleep duration on health (1). Currently, there is no evidence of harm and long-term consequences of sleeping between 6-8 hours a day, but there are data on the benefits of consistent sleep between 6 and 8 h per night (2). Many factors such as the variety of studies, how sleep duration is determined, the diversity in the studied populations, and the selection of parameters that are considered to be affected by sleep duration affect the results, which makes it challenging to obtain a clear result about healthy sleep duration and its effects (2). Over recent years, there has been a good amount of evidence suggesting that too little or too much sleep is related to adverse health outcomes such as mortality (3,4), cardiovascular disease (5-7), respiratory disorder (8), type 2 diabetes (9) hypertension (10,11), and obesity (12,13). Unlike the cardiovascular adverse effects of short sleep duration, there are no compelling hypotheses regarding the mechanism of the relationship between long sleep and illness. In addition, there is only little empirical data to make clear inferences. Myocardial strain imaging can measure left ventricular (LV) global longitudinal strain (GLS) and provides objective quantification of LV myocardial deformation (14). In our current study, we divided the patients into 2 groups who slept more or less than 7 h and evaluated all of them with speckle-tracking echocardiography (STE).

MATERIALS AND METHODS

This retrospective study was conducted to investigate the cardiac effects of sleep duration. Echocardiographic assessment was performed by two experienced echocardiography specialists. The study protocol was approved by the Local Ethics Committee of Memorial Bahçelievler Hospital (approval number: 34, date: 23.03.2022). Written and verbal consent was obtained from all the patients. Between June 2021 and February 2022, 160 individuals who were admitted to our hospital for routine check-up with no known chronic disease were scanned, and patients with normal and good quality echocardiographic examination were included in the study. Patients with poor image quality and whose images were unsuitable for strain examination on echocardiography, who did not answer our phone calls, who had a chronic disease such as asthma, coronary heart disease, hypertension, diabetes, or obstructive sleep apnea, and who were on regular medical treatment were excluded from the study. After all patients were evaluated according to the exclusion criteria, 129 patients were enrolled. The patients who were eligible for the study design were called by the investigator to obtain information on average sleep duration, and two groups were defined by the self-reported sleep duration (≤7 h, >7 h per day) and were used in the statistical analyses.

Echocardiographic Evaluation

Transthoracic echocardiography examinations were performed according to the recommendations of the American Society of Echocardiography. M-mode, two-dimensional imaging, conventional, tissue Doppler evaluation at the septal and lateral mitral annulus, and strain imaging were obtained in all patients at rest in the left decubitus position (15). The echocardiography machine that was used for all patients was a "Philips Epiq 7C." Patients without optimal image quality to perform strain analysis were excluded from the study. The echocardiographic images were analyzed by one experienced cardiologist using the workstation "QLAB version 13."

Data Analysis

STE was performed on four consecutive cardiac cycles of two-dimensional left ventricle (LV) images from the three standard apical views for LV and left atrium (LA); right ventricle (RV) focused apical 4 chamber according to the latest guidelines and a novel 2D strain analytical software (AutoStrain, Philips) was used for strain analysis (16). This software is able to define the region for tracking around a line automatically, and it allows manual changes afterwards. In segments with poor tracking, the border was re-adjusted manually until the best tracking was obtained. Furthermore, RV global longitudinal strain and RV free wall strain, components of left atrial strain (LAS) (atrial reservoir (LAS-r), conduit (LAS-cd), and contractile (LAS-ct) function) were examined according to the recommendations of the European Association of Cardiovascular Imaging (16).

Statistical Analysis

Continuous variables are presented as mean ± standard deviation, and categorical data are presented as percentages or frequencies. Continuous variables were examined by the Kolmogorov-Smirnov test to check for normality of distribution. The patient population was categorized on the basis of sleep duration. Baseline characteristics were compared between groups using Student's t-test or the χ^2 test. A two-tailed p-value of ≤0.05 was considered statistically significant. All data were analyzed using SPSS version 23.0.

RESULTS

We included 129 individuals (71 male and 58 female). The basal clinical characteristics of the patients are presented in Table 1. The mean age of participants who slept 7 hours or less than 7 hours was 36.2±9.2, while the mean age of those who slept for more than 7 hours were 33±11.

All echocardiographic parameters were compared with sleep duration. Among these parameters, LV-GLS was found to be greater in the group who slept less. (-21.5±2.6 vs. -20.3±2.2 p=0.005). However, being within normal limits, left ventricular end diastolic diameter (LVEDD) and right heart chambers were found to be more enlarged in those who slept 7 hours or less than 7 hours. It was also observed that the right atrial volume was higher in the less sleeper group. The analysis of standart echocardiographic parameters and STE findings according to sleep duration is summarized in Table 2, 3 respectively.

DISCUSSION

In the current study, we evaluated the effects of sleep duration on cardiac functions with STE. The main result of our study was that the LV-GLS value was lower in people who slept more than 7 h than in those who slept for 7 h or less. According to the statistical results, although LV-GLS values were within normal limits in both groups, they were significantly lower in those who slept more than 7 h.

Table 1. Clinical and basic biochemical features of the patients

	Sleep duration <7 hours (n=80)	Sleep duration ≥7 hours (n=49)	p
Age (years)	36.2±9.2	33±11	0.078
Height (cm)	169.2±9.3	166.1±9.8	0.074
Weight (kg)	72.5±12.7	69.8±12.7	0.230
Haemoglobin (gr/dL)	14.2±1.9	13.6±2.3	0.097
Haematocrit (%)	43±5	42±4.1	0.243
Creatinine (mg/dL)	0.8±0.2	0.8±0.2	0.689
ALT (U/L)	19±11.5	18.8±21.1	0.931
AST (U/L)	19.3±12.9	23±34.2	0.388
Serum fasting glucose (mg/dL)	97.4±20	100±13.7	0.419
TSH (mU/L)	2±1.2	2.2±1.1	0.478
Estimated GFR (mL/min)	109.9±19.1	107.9±18.6	0.566

ALT: Alanine aminotransferase, AST: Aspartate transaminase, TSH: Thyroid stimulant hormone, GFR: Glomerular filtration rate.

Table 2. Standard echocardiographic parameters according to sleep duration

	Sleep duration <7 hour	Sleep duration ≥7 hour	p
LVEF (%)	65.1±4.1	65.5±3.9	0.677
RVEF (%)	58.7±6.7	58.9±6.9	0.870
LVEDD (cm)	4.6±0.4	4.5±0.4	**0.039**
LVESD (cm)	2.7±0.4	2.5±0.4	0.056
LVEDV (mL)	79.2±20.1	73.2±17.9	0.112
LVESV (mL)	27.5±7.9	25.6±6.8	0.206
LA volume (mL)	45±18.9	41.9±16.3	0.375
RA volume (mL)	39.1±12.1	33.6±14.9	0.035
LA diameter (cm)	3.3±0.5	3.1±0.4	0.099
RVEDD (cm)	3.4±0.5	3.2±0.5	**0.018**
RA diameter (cm)	3.5±0.5	3.1±0.6	**0.001**
TAPSE (cm)	2.3±0.4	2.3±0.4	0.228
MAPSE (cm)	1.6±0.3	1.6±0.3	0.601
Mitral inflow E wave (m/sec)	0.7±0.2	0.8±0.2	0.052
Mitral inflow A wave (m/sec)	0.6±0.1	0.6±0.2	0.104
Septal e' (m/sec)	10.3±2.1	10.7±2.6	0.404
Septal a' (m/sec)	7.5±2	7.1±1.8	0.351
Septal s' (m/sec)	7.1±1.2	7±1.1	0.961
Lateral e' (m/sec)	14.1±3.8	14.8±3.9	0.365
Lateral a' (m/sec)	7.5±1.9	7.8±2.3	0.521
Lateral s' (m/sec)	9.8±2.3	9.5±2.3	0.404
IVRT (msec)	88.9±20.7	84.3±21.3	0.268
DT (msec)	128.5±37.5	136.8±36.1	0.273
TR maximum velocity (m/sec)	2.3±0.2	2.2±0.3	0.067
Estimated PAPs (mmHg)	25.8±4.4	24.4±3.9	0.107

LVEF: Left ventricular ejection fraction, RVEF: Right ventricular ejection fraction, LVEDD: Left ventricular end-diastolic diameter, LVESD: Left ventricular end-systolic diameter, LA: Left atrium, RA: Right atrium, RVEDD: Right ventricular end-diastolic diameter, TAPSE: Tricuspid anular plane systolic excursion, MAPSE: Mitral anular plane systolic excursion, IVRT: Isovolumetric relaxation time, DT: Deceleration time, TR: Tricuspid regurgitation, PAPs: Systolic pulmonary artery pressure.

Table 3. STE components according to sleep duration

	Sleep duration <7 hour	Sleep duration ≥7 hour	p
LV-GLS (%)	-21.5±2.6	-20.3±2.2	**0.005**
LA-r (%)	49.4±15.3	50.1±16.4	0.822
LA-cd (%)	-34.8±11.7	-33.3±12.1	0.488
LA-ct (%)	-14.5±11	-16.8±9.6	0.242
RV-FWSL (%)	-29.3±5.5	-27.8±5.7	0.121
RV-GLS (%)	-25±7.3	-24.7±5	0.780

STE: Speckle-tracking echocardiography, RV-FWSL: Right ventricular free wall longitudinal strain, RV-GLS: Right ventricular global longitudinal strain, LV-GLS: Left ventricular global longitudinal strain, LAS-R: Left atrial reservoir strain, LAS-cd: Left atrial conduit strain, LAS-ct: Left atrial contractile strain.

Although various studies have shown the effects of long sleep duration on cardiovascular health, to the best of our knowledge, there is no study in the literature showing its relationship with LV-GLS. LV-GLS is a measure of LV myocardial shortening, which is a good marker for LV dysfunction and determining cardiovascular events in patients with various diseases and healthy people (17).

Sleep is an indispensable physiological activity for people to maintain their lives optimally. Many studies conducted to date have shown that sleep duration is associated with morbidity and mortality (9,18).

In our study, we found that LV-GLS was lower in people who slept more than 7 hours compared to the people who slept 7 hours or less. In a multicenter study by Wang et al. (18), the relationship between sleep duration, death, and major cardiovascular events was investigated. As a result of this study, it was observed that the risk of death and major cardiovascular events was increased in people who slept 6 hours or less and people who slept for more than 8 hours. In this study, it was observed that the risk increase in people who slept 6 h or less was less than that in those who slept 9-10 hours, and this risk was even higher in people who slept 10 hours or more [<6 hours hazard ratio (HR): 1.09, 9-10 hours HR: 1.17, >10 hours HR: 1.41] (18). In a meta-analysis, Cappuccio et al. (9) investigated the relationship between sleep duration and coronary heart disease, cardiovascular causes, and stroke-related death. Because of this meta-analysis, the risk of death from coronary artery disease and stroke was found to be high in both groups, but a significant increase in cardiovascular mortality was not found in the group with short sleep duration, unlike long sleep duration (9). In another meta-analysis, Yin et al. (19) compared people who slept 7 h or less with those who slept more than 7 hours. As a result of this analysis, it was shown that the risk of coronary heart disease, cardiovascular disease, stroke, and total mortality increased with each hour of decrease and/or increase in sleep time. They also found that each hour of oversleeping increased the risk of these events by more than one hour of less sleep (19).

As seen in the above-mentioned studies and meta-analyses, long sleep duration increases the risk of cardiovascular morbidity and mortality. Due to this situation, a clear mechanism has not yet been revealed. One of the most prominent hypotheses at the moment is that long sleep duration may be a marker of one or more underlying diseases. In a study by Patel et al. (20), it was observed that in conditions such as systemic lupus erythematosus and various psychiatric pathologies such as depression, excessive alcohol intake, and obesity, the rate of sleep for 7 hours or more was higher. Similarly, in another study, Stranges et al. (21) showed that people who slept for 8 hours or more had less physical activity and worse results in the SF-36 questionnaire, which indicates physical health. Krueger and Friedman (22), on the other hand, compared people who sleep 9 hours or more per day with those who sleep 7 hours. Compared with the other group, the prevalence of diabetes, cardiovascular diseases, and smoking history was higher in people who slept a lot (22). As seen in the aforementioned studies, it should be kept in mind that long sleep duration may not be a cause of adverse cardiovascular events in the studies but a reflection of the comorbidities that cause these events.

Another result of our study was that LVEDD and the diameters of the right heart chambers were larger in the group with a short sleep duration. Similar to our study, Lee et al. (23) demonstrated that LVEDD was significantly higher in people who slept for 7 h or less compared with other groups, yet no evaluation of right heart chambers was performed in this study. Although there is no clear explanation for the short sleep duration and the presence of enlargement of the heart chambers, various hypotheses can be put forward. It was first shown in studies that short sleep duration was associated with increased adrenergic tone (24,25) that may have increased the release of renin in the macula densa in the kidney via beta receptors, leading to sodium retention and consequently an increase in intravascular volume. This may explain the increase in the right and left ventricles, especially because of increased preload. This can also be explained by the increase in adrenergic tone, which may cause an increase in LV end-diastolic volume because of increased blood pressure in patients and a consequent increase in afterload. Therefore, we believe that more studies are needed on this subject.

Study Limitations

Our study has several limitations. The first limitation is that the study was a single-center study and the number of patients included was small. Second, verbal learning of sleep duration and not using polysomnography may have affected the results of the study. some sleep disorders and sleep quality, particularly those detected by polysomnography, affect cardiovascular health.

CONCLUSION

As a result, GLS, which is one of the parameters showing LV function, was found to be less in healthy people with a sleep time above a certain period, and we think that this may be a hypothesis to explain

the relationship of long sleep time with cardiovascular events. In addition, there may be significant changes in the geometry of the left and right heart chambers due to increased adrenergic tone during short sleep. We think that further studies are needed to reflect this situation in the clinic.

Ethics

Ethics Committee Approval: The study protocol was approved by the Local Ethics Committee of Memorial Bahçelievler Hospital (approval number: 34, date: 23.03.2022).

Informed Consent: Written and verbal consent was obtained from all the patients.

Authorship Contributions

Concept: G.B., Ö.Ö., Design: G.B., Ö.Ö., Supervision: G.B., Resources: G.B., Ö.Ö., Materials: G.B., Ö.Ö., Data Collection and/or Processing: G.B., Ö.Ö., Analysis and/or Interpretation: G.B., F.A.D., E.Ö., S.Ü., Literature Search: G.B., F.A.D., E.Ö., S.Ü., Writing: G.B., Ö.Ö., E.Ö., Critical review: F.A.D., E.Ö., S.Ü.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

REFERENCES

1. Montano N, Fiorelli E, Tobaldini E. Sleep Duration and the Heart: I Sleep, Therefore I Beat. J Am Coll Cardiol 2019; 74: 1315-6.
2. Cappuccio FP, D'Elia L, Strazzullo P, Miller MA. Sleep duration and all-cause mortality: a systematic review and meta-analysis of prospective studies. Sleep 2010; 33: 585-92.
3. Ferrie JE, Shipley MJ, Cappuccio FP, Brunner E, Miller MA, Kumari M, et al. A prospective study of change in sleep duration: associations with mortality in the Whitehall II cohort. Sleep 2007; 30: 1659-66.
4. Kripke DF, Garfinkel L, Wingard DL, Klauber MR, Marler MR. Mortality associated with sleep duration and insomnia. Arch Gen Psychiatry 2002; 59: 131-6.
5. Meisinger C, Heier M, Lowel H, Schneider A, Doring A. Sleep duration and sleep complaints and risk of myocardial infarction in middle-aged men and women from the general population: the MONICA/KORA Augsburg Cohort Study. Sleep 2007; 30: 1121-7.
6. Ikehara S, Iso H, Date C, Kikuchi S, Watanabe Y, Wada Y, et al. Association of sleep duration with mortality from cardiovascular disease and other causes for Japanese men and women: the JACC study. Sleep 2009; 32: 295-301.
7. Stone KL, Ewing SK, Ancoli-Israel S, Ensrud KE, Redline S, Bauer DC, et al. Self-reported sleep and nap habits and risk of mortality in a large cohort of older women. J Am Geriatr Soc 2009; 57: 604-11.
8. Bliwise DL. Sleep-related respiratory disturbances. J Gerontol 1984; 39: 255.
9. Cappuccio FP, D'Elia L, Strazzullo P, Miller MA. Quantity and quality of sleep and incidence of type 2 diabetes: a systematic review and meta- analysis. Diabetes Care 2010; 33: 414-20.
10. Gangwisch JE, Heymsfield SB, Boden-Albala B, Buijs RM, Kreier F, Pickering TG, et al. Short sleep duration as a risk factor for hypertension: analyses of the first National Health and Nutrition Examination Survey. Hypertension 2006; 47: 833-9.
11. Cappuccio FP, Stranges S, Kandala NB, Miller MA, Taggart FM, Kumari M, et al. Gender-specific associations of short sleep duration with prevalent and incident hypertension: the Whitehall II Study. Hypertension 2007; 50: 693-700.
12. Cappuccio FP, Taggart FM, Kandala NB, Currie A, Peile E, Stranges S, et al. Meta-analysis of short sleep duration and obesity in children and adults. Sleep 2008; 31: 619-26.
13. Stranges S, Cappuccio FP, Kandala NB, Miller MA, Taggart FM, Kumari M, et al. Cross-sectional versus prospective associations of sleep duration with changes in relative weight and body fat distribution: the Whitehall II Study. Am J Epidemiol 2008; 167: 321-9.
14. Collier P, Phelan D, Klein A. A Test in Context: Myocardial Strain Measured by Speckle-Tracking Echocardiography. J Am Coll Cardiol 2017; 69: 1043-56.
15. Mitchell C, Rahko PS, Blauwet LA, Canaday B, Finstuen JA, Foster MC, et al. Guidelines for Performing a Comprehensive Transthoracic Echocardiographic Examination in Adults: Recommendations from the American Society of Echocardiography. J Am Soc Echocardiogr 2019; 32: 1-64.
16. Badano LP, Kolias TJ, Muraru D, Abraham TP, Aurigemma G, Edvardsen T, et al. Standardization of left atrial, right ventricular, and right atrial deformation imaging using two-dimensional speckle tracking echocardiography: a consensus document of the EACVI/ASE/Industry Task Force to standardize deformation imaging. Eur Heart J Cardiovasc Imaging 2018; 19: 591-600.
17. Russo C, Jin Z, Elkind MS, Rundek T, Homma S, Sacco RL, et al. Prevalence and prognostic value of subclinical left ventricular systolic dysfunction by global longitudinal strain in a community-based cohort. Eur J Heart Fail 2014; 16: 1301-9.
18. Wang C, Bangdiwala SI, Rangarajan S, Lear SA, AlHabib KF, Mohan V, et al. Association of estimated sleep duration and naps with mortality and cardiovascular events: a study of 116 632 people from 21 countries. Eur Heart J 2019; 40: 1620-9.
19. Yin J, Jin X, Shan Z, Li S, Huang H, Li P, et al. Relationship of Sleep Duration with All-Cause Mortality and Cardiovascular Events: A Systematic Review and Dose-Response Meta-Analysis of Prospective Cohort Studies. J Am Heart Assoc 2017; 6: e005947.
20. Patel SR, Malhotra A, Gottlieb DJ, White DP, Hu FB. Correlates of long sleep duration. Sleep 2006; 29: 881-9.
21. Stranges S, Dorn JM, Shipley MJ, Kandala NB, Trevisan M, Miller MA, et al. Correlates of short and long sleep duration: a cross-cultural comparison between the United Kingdom and the United States: the Whitehall II Study and the Western New York Health Study. Am J Epidemiol 2008; 168: 1353-64.
22. Krueger PM, Friedman EM. Sleep duration in the United States: a cross-sectional population-based study. Am J Epidemiol 2009; 169: 1052-63.
23. Lee JH, Park SK, Ryoo JH, Oh CM, Kang JG, Mansur RB, et al. Sleep Duration and Quality as Related to Left Ventricular Structure and Function. Psychosom Med 2018; 80: 78-86.
24. Zhong X, Hilton HJ, Gates GJ, Jelic S, Stern Y, Bartels MN, et al. Increased sympathetic and decreased parasympathetic cardiovascular modulation in normal humans with acute sleep deprivation. J Appl Physiol 2005; 98: 2024-32.
25. Tochikubo O, Ikeda A, Miyajima E, Ishii M. Effects of Insufficient Sleep on Blood Pressure Monitored by a New Multibiomedical Recorder. Hypertension 1996; 27: 1318-24.