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Association of Sleep Duration and Cardio-Ankle Vascular Index in Community-Dwelling Older Adults

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Aim: This study aims to investigate the association of the Cardio-Ankle Vascular Index (CAVI) with self-reported sleep duration and sleep quality in community-dwelling older adults aged ≥ 65 years.

Methods: The Tarumizu Study was a cohort of community-based health checkups conducted in the Tarumizu City, Japan, in 2018 and 2019. In total, 997 participants aged ≥ 65 years (median age, 74 years) were examined. We obtained the average sleep duration and sleep quality using self-reported questionnaires and classified them into three separate groups according to sleep duration (<6 h, 6–8 h, and ≥ 8 h) and sleep quality (good, medium, and poor). The arterial stiffness was measured using the CAVI.

Results: As per our findings, the CAVI was significantly higher in the ≥ 8 h sleep group (CAVI=9.6±1.3) than in the <6 h (CAVI=9.1±1.1) or 6–8 h (CAVI=9.1±1.2) groups ($p<0.001$). After adjustment for age, sex, systolic blood pressure, current smoking status, body mass index, frequency of exercise, educational background, frailty, sleep medication, sleep quality, and nap duration, multivariable regression analysis demonstrated that the CAVI was significantly higher in the ≥ 8 h group than in the 6–8 h group ($p=0.016$). In contrast, multivariable regression analysis showed that there was no significant association between sleep quality and CAVI.

Conclusions: A significant association was noted between long sleep duration (≥ 8 h) and elevated CAVI in community-dwelling older adults aged ≥ 65 years. We, therefore, suggest that long sleep duration, not sleep quality, is correlated with arterial stiffness in older adults.

Key words: Sleep duration, Arterial stiffness, Cardio-Ankle Vascular Index, Community-dwelling older, Sleep quality

Introduction

Aging has been identified as one of the main risk factors for atherosclerosis development. Age is associated with vascular wall remodeling, including luminal enlargement and intimal and medial thickening, and aging is known to increase vascular stiffness[1]. The brachial-ankle pulse wave velocity (baPWV) was used to evaluate the degree of atherosclerosis and arterial stiffness. The baPWV has been applied for the risk stratification of patients with atherosclerotic cardiovascular disease and/or its risk factors. Several cross-sectional studies have demonstrated a significant correlation between the baPWV and known risk factors for cardiovascular diseases such as age, diabetes mellitus, dyslipidemia, obesity, and smoking[2, 3]. Sleep is evaluated by sleep duration and quality, and long sleep duration and poor-sleep quality have been associated with increased arterial stiffness as measured by baPWV[4-10]. These

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previous studies on sleep duration and quality were analyzed in individuals aged <65 years, and the association between the baPWV and sleep duration or quality has not been evaluated in the general older population aged ≥ 65 years.

Although the baPWV is a simple but useful index for determining the arterial stiffness, the drawback is that the result is affected by changes in blood pressure (BP) during measurement of the pulse wave velocity (PWV)[11-13]. The Cardio-Ankle Vascular Index (CAVI), an atherosclerotic index, was developed by measuring the PWV and BP[13-15]. The CAVI is adjusted for BP based on the stiffness parameter β and measures the arterial stiffness independent of the BP. We have previously reported that the correlation of CAVI with systolic blood pressure (SBP) was weaker than that of baPWV with SBP, and CAVI was not affected by changes in BP during measurement[15]. The CAVI is high in patients with various atherosclerotic diseases, including coronary artery disease, stroke, and heart failure, and coronary risk factors are known to increase the CAVI[16-20]. To the best of our knowledge, no study has investigated the association between CAVI and sleep duration and quality. Furthermore, no report has analyzed the relationship between sleep and baPWV or CAVI in older individuals in whom notable quantitative and qualitative changes in sleep occur with age[21, 22].

Aim

This study has aimed to investigate the association between CAVI and self-reported sleep duration and sleep quality in community-dwelling older adults aged ≥ 65 years.

Methods

Study Population

This cross-sectional study obtained data from the Tarumizu Study (2018, 2019). The Tarumizu Study was a cohort of community-based health checkups conducted in the Tarumizu City, Kagoshima, Japan, from June to December 2018 (Tarumizu Study 2018) and from June to December 2019 (Tarumizu Study 2019). The participants were residents of Tarumizu City aged ≥ 65 years at the time of examination.

In total, 1,056 older adults (≥ 65 years) were enrolled in the Tarumizu Study 2018, 2019. In patients who participated in both the Tarumizu Study 2018 and 2019, we have collected data from the Tarumizu Study 2019. Participants with atrial fibrillation (n=6) and ankle brachial pressure index (ABI) <0.9 (n=38) were excluded because the CAVI cannot be evaluated accurately in participants with atrial fibrillation or peripheral artery disease. There were missing data on CAVI in 13 patients and the sleep status in 2 patients. Ultimately, 997 participants were included in the analysis.

This study was conducted in compliance with the Declaration of Helsinki and Japanese Ethical Guidelines for Medical and Health Research Involving Human Subjects. This study was approved by the Ethics Committee of the Graduate School of Medical and Dental Sciences of Kagoshima University (Ref. No. 170351). Written informed consent was obtained from all participants.

Measurements

1. Assessment of Sleep

Information on the average daily sleep duration (<4 h, 4–6 h, 6–8 h, ≥ 8 h) was obtained and classified into the following three categories: <6 h, 6–8 h, and ≥ 8 h according to references[5, 8]. Sleep quality was obtained by asking the following question: "how was your sleep quality at night over?" The responses were good, medium, modestly poor, or poor and classified as "good," "medium," and "poor" (modestly poor and poor).

2. Measurements of CAVI, BP, and ABI

The CAVI was measured using Vasera VS-3000 (Fukuda Denshi, Tokyo, Japan), as reported previously[14, 15]. Briefly, cuffs were applied to the four extremities, and the electrocardiographic electrodes were attached to the upper arm. A microphone was placed at the sternal angle for the phonocardiography. The participants then rested in the supine position for 5 min, and the CAVI was measured. The PWV was calculated by dividing the distance from the aortic valve to the ankle artery by the sum of the time between the closing sound of the aortic valve and the notch of the brachial pulse wave and the ankle pulse wave. To minimize the cuff inflation effects on blood flow dynamics, the pulse waves were measured with the cuffs inflated to less than the diastolic BP (DBP) (50 mmHg). The CAVI was calculated using the following equation: $CAVI = a\ [\{2\rho \times 1/(SBP - DBP)\} \times \{\ln (SBP/DBP) \times PWV^2\}] + b$, where a and b are constants and ρ is the density of blood. The extremity BP was then measured using an oscillometric technique. Using the Vasera VS-3000, the ABI was also calculated. The SBP and DBP in the right brachial artery were used for individual BP measurements.

3. Data Collection

Anthropometric measurements, including the

body weight and height, were evaluated, and the body mass index (BMI) was calculated.

Each participant was interviewed by a trained research staff using a standardized questionnaire designed specifically for this present study. Cigarette smoking was classified as current or non-current. The educational background was classified as ≤ high school, who were graduated from high school or lower, and >high school, who were graduated from higher than high school. The frequency of exercise was classified as rarely or never and two times per week or more. Physical frailty was operationalized using the Fried's components: slowness, weakness, exhaustion, low physical activity, and weight loss[23-25]. Frailty was defined when three or more characteristics are present. Nap duration per day was classified as none, <60 min, or ≥ 60 min.

4. Statistical Analysis

Quantitative data were presented as mean ± standard deviation for data showing parametric distributions, whereas median and interquartile range (IQR) were utilized for data showing nonparametric distributions. The differences in the distribution of the variables according to the daily sleep duration and quality were tested for statistical significance using the one-way analysis of variance (ANOVA) for the parametrically distributed continuous variables, Kruskal–Wallis test for the non-parametrically distributed continuous variables, and chi-square test for categorical variables. Multivariable regression analyses were then performed to determine the relationship between daily sleep duration and CAVI or the relationship between sleep quality and CAVI in the following models: Crude model, without adjustment; Model I, adjusted for age, sex, and SBP; Model II, adjusted for Model I variables plus current smoking and BMI; Model III, adjusted for Model II variables plus the frequency of exercise, educational background, frailty, and sleep medication; and Model IV, adjusted for Model III variables plus sleep quality or duration, and nap duration.

Statistical significance was assumed at $p<0.05$. The statistical analyses were performed using the JMP version 15.0 software (SAS Institute, Cary, NC, USA).

Results

Table 1 shows the characteristics of the 997 participants divided by sleep duration per day. The median age of the participants was 74 [70, 80] years, and 37.6% were men. The mean CAVI was 9.1 ± 1.2. The number of participants who slept for less than 6 h, 6–8 h, and ≥ 8 h was 449 (45%), 438 (44%), and 110 (11%), respectively. Furthermore, 84 adults (8.4%) took sleep medications. Age and CAVI in the ≥ 8 h group were significantly higher than those in the <6 h or 6–8 h groups ($p<0.001$). Significant differences were noted in terms of sex ($p=0.011$), sleep medication ($p=0.035$), and sleep quality ($p<0.0001$) among the groups. However, there were no significant differences in BMI, current smoking status, SBP, education, exercise, frailty, or nap duration among the groups.

We analyzed the relationship between sleep duration and CAVI in the participants using multivariable regression analysis with CAVI as the dependent variable **(Table 2)**. CAVI was significantly higher in the ≥ 8 h group compared in the 6–8 h group in the unadjusted crude model ($p<0.0001$). Moreover, Model I adjusted for age, sex, and SBP ($p=0.027$); Model II adjusted for Model I plus current smoker and BMI ($p=0.027$); Model III adjusted for Model II plus the frequency of exercise, educational background, frailty, and sleep medication ($p=0.020$); and Model IV adjusted for Model III plus sleep quality and nap ($p=0.016$) demonstrated a significantly higher CAVI in the ≥ 8 h group compared in the 6–8 h group.

Next, we analyzed the characteristics of the participants according to their sleep quality **(Table 3)**. There were 468 (47%) patients in the good-sleep group, 374 (38%) in the medium-sleep group, and 155 (15%) in the poor-sleep group. There were significant differences in terms of sex ($p=0.003$), exercise frequency ($p=0.044$), frailty ($p=0.001$), sleep medication ($p<0.0001$), nap duration ($p=0.010$), and sleep duration ($p<0.0001$) among the three groups. Patients in the good-sleep group were significantly older than those in the medium-sleep group ($p<0.001$). However, the ANOVA did not show any significant association between sleep quality and CAVI.

Moreover, we analyzed the relationship between sleep quality and CAVI in all the participants **(Table 4)**. The multivariable regression analysis using CAVI as a dependent variable demonstrated that the medium-sleep group showed a significantly lower CAVI than the good-sleep group in the unadjusted crude model ($p=0.028$), but there was no significant association between sleep quality and CAVI in Models I–IV adjusted for the factors described in **Table 4**.

Discussion

In this study, among the community residents aged ≥ 65 years, the CAVI was determined to be significantly higher in the ≥ 8 h sleep group than in

Table 1. The characteristics of the participants by sleep duration ($n = 997$)

Characteristics	Overall	Sleep duration per day			p value
		< 6h	6-8h	≥ 8h	
Number	997	449	438	110	
CAVI	9.1 ± 1.2	9.1 ± 1.1	9.1 ± 1.2	9.6 ± 1.3*	0.0001 [b]
Age (years)	74 [70, 80]	73 [70, 78]	74 [69, 80]	77 [72, 83]*	0.0001 [c]
Sex, Men, *n* (%)	375 (37.6)	152 (33.8)	169 (38.6)	54 (49.1)	0.011 [a]
BMI (kg/m^2)	23.2 ± 3.3	23.3 ± 3.3	23.2 ± 3.3	23.0 ± 3.6	0.570 [b]
Current smoker, *n* (%)	47 (4.7)	22 (4.9)	21 (4.8)	4 (3.6)	0.850 [b]
SBP (mmHg)	137.1 ± 17.3	136.1 ± 17.3	137.9 ± 17.3	137.4 ± 17.3	0.293 [b]
Educational background, *n* (%)					
> High school	527 (52.9)	242 (53.9)	238 (54.3)	47 (42.7)	0.078 [a]
≤ High school	470 (47.1)	207 (46.1)	200 (45.7)	63 (57.3)	
Frequency of exercise, *n* (%)					
Rarely or never	599 (60.0)	257 (57.2)	278 (63.5)	64 (58.2)	0.151 [a]
2 times/week or more	398 (40.0)	192 (42.8)	160 (36.5)	46 (41.8)	
Frailty, *n* (%)	78 (8.0)	34 (7.7)	30 (7.0)	14 (12.7)	0.135 [a]
Sleep medication, *n* (%)	84 (8.4)	49 (10.9)	29 (6.6)	6 (5.5)	0.035 [a]
Nap duration, /day, *n* (%)					
None	401 (40.2)	184 (41.0)	185 (42.2)	32 (29.1)	0.054 [a]
< 60min	540 (54.2)	241 (53.7)	232 (53.0)	67 (60.9)	
≥ 60min	56 (5.6)	24 (5.3)	21 (4.8)	11 (10.0)	
Sleep quality, *n* (%)					
Good	468 (46.9)	149 (33.2)	241 (55.0)	78 (70.9)	< 0.0001 [a]
Medium	374 (37.5)	183 (40.8)	165 (37.7)	26 (23.6)	
Poor	155 (15.5)	117 (26.1)	32 (7.3)	6 (5.5)	

The data are presented as mean ± SD (standard deviation), median [quartile], or number (percentage). The categorical variables were analyzed using the chi-square test (a), and continuous variables were analyzed using the one-way analysis of variance (b) or Kruskal-Wallis test (c). CAVI, cardio-ankle vascular index; BMI, body mass index; SBP, systolic blood pressure.
*: $p < 0.001$ vs. < 6h or 6-8h by Bonferroni correction.

Table 2. Relationship of sleep duration and CAVI in the participants ($n = 997$)

Sleep duration	CAVI	Crude model		Model I		Model II		Model III		Model IV	
		β (95%CI)	p value	β (95%CI)	p value	β (95%CI)	p value	β (95%CI)	p value	β (95%CI)	p value
< 6h ($n = 449$)	9.1 ± 1.1	-0.02 (-0.10, 0.06)	0.625	0.01 (-0.06, 0.08)	0.752	0.02 (-0.05, 0.09)	0.627	0.02 (-0.05, 0.09)	0.543	0.02 (-0.05, 0.10)	0.533
6-8h ($n = 438$)	9.1 ± 1.2	Ref.		Ref.		Ref.		Ref.		Ref.	
≥ 8h ($n = 110$)	9.6 ± 1.3	0.25 (0.13, 0.38)	< 0.0001	0.13 (0.01, 0.24)	0.027	0.13 (0.01, 0.24)	0.027	0.14 (0.02, 0.25)	0.020	0.14 (0.03, 0.26)	0.016

CAVI data are presented as the mean ± SD (standard deviation).
Model I: adjusted for Age, Sex and Systolic blood pressure.
Model II: adjusted for all the variables in Model I plus current smokers and body mass index.
Model III: adjusted for all the variables in Model II plus frequency of exercise, educational background, frailty, and sleep medication.
Model IV: adjusted for all the variables in Model III plus sleep quality and nap duration.
CAVI, cardio-ankle vascular index; CI, confidence interval.

the < 6 h or 6–8 h groups. After adjustment for various factors, multivariable regression analysis demonstrated that the CAVI was significantly higher in the ≥ 8 h group than in the 6–8 h group. The multivariable regression analysis showed no significant association between sleep quality and CAVI in the adjusted models. To the best of our knowledge, this study is the first to clarify a significant association between sleep duration and CAVI in community-dwelling older adults aged ≥ 65 years.

Previous studies have reported an association between sleep duration and increased arterial stiffness using baPWV, which is a known arterial stiffness index. Among 4268 Japanese local government

Table 3. The characteristics of the participants by sleep quality ($n=997$)

Characteristics	Sleep quality			p value
	Good	Medium	Poor	
Number	468	374	155	
CAVI	9.2 ± 1.3	9.0 ± 1.1	9.1 ± 1.2	0.090[b]
Age (years)	75 [70, 81]*	72 [69, 78]	74 [69, 80]	0.0008[c]
Sex, Men, *n* (%)	202 (43.2)	119 (31.8)	54 (34.8)	0.003[a]
BMI (kg/m^2)	23.2 ± 3.4	23.3 ± 3.2	23.0 ± 3.5	0.705[b]
Current smoker, *n* (%)	26 (5.6)	14 (3.7)	7 (4.5)	0.464[a]
SBP (mmHg)	137.7 ± 17.4	136.8 ± 17.0	135.8 ± 17.6	0.498[b]
Educational background, *n* (%)				
> High school	253 (54.1)	197 (52.7)	77 (49.7)	0.636[a]
≤ High school	215 (45.9)	177 (47.3)	78 (50.3)	
Frequency of exercise, *n* (%)				
Rarely or never	281 (60.0)	212 (56.7)	106 (68.4)	0.044[a]
2 times/week or more	187 (40.0)	162 (43.3)	49 (31.6)	
Frailty, *n* (%)	45 (9.8)	14 (3.8)	19 (12.5)	0.001[a]
Sleep medication, *n* (%)	19 (4.1)	28 (7.5)	37 (23.9)	<0.0001[a]
Nap duration/day, *n* (%)				
None	171 (36.5)	158 (42.3)	72 (46.5)	0.010[a]
<60min	263 (56.2)	205 (54.8)	72 (46.5)	
≥ 60min	34 (7.3)	11 (2.9)	11 (7.0)	
Sleep duration, *n* (%)				
<6h	149 (31.8)	183 (48.9)	117 (75.5)	<0.0001[a]
6-8h	241 (51.5)	165 (44.1)	32 (20.7)	
≥ 8h	78 (16.7)	26 (7.0)	6 (3.9)	

The data are presented as mean ± SD (standard deviation), median [quartile], or number (percentage).
The categorical variables were analyzed using the chi-squared test (a), and continuous variables were analyzed using the One-way analysis of variance (b) or Kruskal-Wallis test (c). CAVI: cardio-ankle vascular index.
SBP: systolic blood pressure, BMI: body mass index.
*$p<0.001$ vs. medium by Bonferroni correction.

Table 4. Relationship of sleep quality and CAVI in the participants ($n=997$)

Sleep quality	CAVI	Crude model		Model I		Model II		Model III		Model IV	
		β (95%CI)	p value	β (95%CI)	p value	β (95%CI)	p value	β (95%CI)	p value	β (95%CI)	p value
Good ($n=468$)	9.2 ± 1.3	Ref.		Ref.		Ref.		Ref.		Ref.	
Medium ($n=374$)	9.0 ± 1.1	-0.09 (-0.17, -0.01)	0.028	0.01 (-0.07, 0.08)	0.861	0.01 (-0.06, 0.08)	0.798	0.002 (-0.07, 0.08)	0.955	0.006 (-0.07, 0.08)	0.880
poor ($n=155$)	9.1 ± 1.2	-0.04 (-0.15, 0.07)	0.444	0.02 (-0.08, 0.12)	0.700	-0.002 (-0.10, 0.10)	0.969	-0.01 (-0.11, 0.09)	0.846	-0.004 (-0.11, 0.10)	0.945

The CAVI data are presented as mean ± SD (standard deviation).
Model I: adjusted for Age, Sex and Systolic blood pressure.
Model II: adjusted for all the variables in Model I plus current smokers and body mass index.
Model III: adjusted for all the variables in Model II plus frequency of exercise, educational background, frailty, and sleep medication.
Model IV: adjusted for all the variables in Model III plus sleep duration and nap duration.
CAVI, cardio-ankle vascular index; CI, confidence interval.

employees aged 35–62 years, subjects with ≥ 9 h sleep showed significantly elevated baPWV compared with the reference group with 7 h sleep[7]. In 18,106 healthy Koreans with an average age of 45.8 years, a U-shaped association between sleep duration and baPWV was demonstrated by comparing ≤ 5, 6, 8, and ≥ 9 h duration of sleep with 7 h duration of sleep[9]. Among the 2,095 Taiwanese men with a mean aged of 45.7 years who underwent health examination, those with long sleep duration (>8 h), but not those with short sleep duration (<6 h), had a high risk of increased arterial stiffness determined by baPWV ≥ 1400 cm/s

than those with normal sleep duration (6–8 h)[8]. In 2,304 Japanese patients with a high risk for cardiovascular diseases and a mean age of 64.7 years, long sleep duration (>8 h) was found to be significantly associated with baPWV when the patients with 6–8 h of sleep duration were defined as the reference group[5]. However, previous studies using baPWV targeted participants aged <65 years and did not focus on older individuals. To the best of our knowledge, no study has analyzed the effects of sleep duration on CAVI, which is not affected by BP at measuring time. Our study using CAVI in residents aged ≥ 65 years was consistent with previous studies using baPWV and showed significantly higher CAVI values in the long sleep duration group (≥ 8 h), but not in the short sleep duration group (<6 h), compared with the 6–8 h of sleep duration group.

The mechanism underlying the association between long sleep duration and arterial stiffness is yet to be fully understood. A meta-regression analyses using 5,134,036 participants from 137 prospective cohort studies reported statistically significant linear associations between longer sleep duration and increased mortality or cardiovascular diseases[26]. Long sleep (≥ 8 h) duration and high C-reactive protein (hs-CRP) levels were significantly associated with increased baPWV in 2,304 older Japanese patients at high risk for cardiovascular diseases[5]. A systematic review and meta-analysis of cohort studies reported that extreme long sleep duration, but not short sleep duration, was associated with increased markers of systemic inflammation, such as the CRP and interleukin-6 (IL-6)[27]. In addition, prolonged sleep duration due to sleep fragmentation and nocturnal wakefulness may activate the sympathetic nervous system[28, 29]. Sleep fragmentation and sympathetic overactivity were associated with diurnal and 24 h SBP and an increased risk of hypertension in 780 old volunteers, leading to atherosclerosis[30]. Increased fragmentation and sympathetic overactivity due to long sleep have been the proposed underlying mechanisms of the increased mortality and cardiovascular diseases. However, the mechanisms and interactions underlying long sleep durations and arterial stiffness remain to be poorly understood. Further mechanistic studies are required to confirm this finding.

In contrast, Morita *et al.* reported an inverse correlation between sleep duration and CAVI in children[31]. They discussed that short sleep duration could be related to higher sympathetic nervous activity, which may have concomitantly mediated alterations in arterial function in children with habitual short sleep durations[31]. Moreover, we suggest that sleep induces growth hormone release, which, in turn, protects against atherosclerosis and carotid intima-media thickness in children[32]. Therefore, long sleep may reduce CAVI in children. Level and effect of growth hormone are often reduced in older adults[33]. However, growth hormone level is high, and its effects play important role in children.

The precise mechanism underlying the correlation between sleep quality and arterial stiffness remains unclear. Previous studies have investigated the association between baPWV and sleep quality and duration. In 18,106 healthy Koreans who underwent a health checkup examination with a mean age of 45.1 years, poor-sleep quality was associated with higher baPWV than good-sleep quality[9]. In Japanese patients with type II diabetes with a mean age of 57.8 years, poor subjective sleep quality (n=77) was associated with a higher baPWV than average (n=185) or good (n=462) sleep quality[4]. This prospective cohort study included 306 patients with cardiovascular risk factors and demonstrated that low sleep quality was associated with the progression of arterial stiffness measured by baPWV over a 3-year period[10]. Among the 14,485 patients with hypertension, the patients with poor or medium-sleep quality were determined to have a nonsignificant higher prevalence of arterial stiffness (baPWV ≥ 1800 cm/s) than the patients with good-sleep quality[6]. The results of our study are consistent with these results. Although no study has analyzed the correlation between sleep quality and CAVI, this study is the first to report a nonsignificant association between sleep quality and CAVI in patients aged ≥ 65 years. Further studies using objective sleep quality and CAVI are needed to elucidate the correlation between sleep quality and CAVI.

This present study has several limitations. First, our study was a cross-sectional design, and the causality was not determined. Therefore, it is necessary to examine whether long sleep duration causes arterial stiffness by conducting further longitudinal studies. Second, information on sleep duration and quality was self-reported by the participants. Although previous studies have reported that subjective sleep duration correlated with objectively measured sleep duration[34, 35], further studies are needed to analyze the relationship between the CAVI and sleep duration or sleep quality using objective devices. Third, the self-reported sleep surveys in this present study did not consider sleep apnea because it was difficult to obtain accurate information on sleep apnea using self-reported questionnaires. Sleep apnea has been reported to affect sleep quality and atherosclerosis, which may have influenced the results of our study. Therefore, sleep apnea should be evaluated using an objective

device in further studies.

Conclusion

In conclusion, ≥ 8 h sleep duration was significantly associated with elevated CAVI as a marker of arterial stiffness in older individuals aged ≥ 65 years. However, there was no significant association between sleep quality and the CAVI. Further studies are needed to determine whether an appropriate sleep duration can prevent an increase in arterial stiffness.

Conflicts of Interests

The authors have no potential conflicts of interest to disclose.

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