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# Multidimensional sleep characteristics and arterial stiffness in older adults

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ABSTRACT

*Aim:* Sleep is a multidimensional behavior encompassing duration, quality, and behavioral patterns. The associations between sleep characteristics and arterial stiffness remain unclear. This study aimed to investigate the associations between multidimensional sleep characteristics and arterial stiffness in older adults.
*Methods:* This cross-sectional study included 243 community-dwelling adults aged ≥65 years. Arterial stiffness was assessed using the cardio-ankle vascular index (CAVI). Sleep parameters—including time in bed (TIB), sleep duration, sleep latency, sleep restfulness score (SRS), daytime sleepiness, and nocturia—were evaluated using standardized self-administered questionnaires. Multivariable linear regression analyses were performed to assess independent associations between sleep characteristics and CAVI after adjustment for potential confounders.
*Results:* The mean age was 76.4 years, and the mean CAVI was 8.90 ± 1.03. CAVI differed significantly across TIB, SRS, and nocturia categories, with higher values observed in participants with prolonged TIB (≥8 h), poorer sleep restfulness, and more frequent nocturia. In contrast, sleep duration, sleep latency, and daytime sleepiness were not associated with CAVI. Poorer sleep restfulness was associated with higher arterial stiffness, although this finding was sensitive to participants with very poor sleep restfulness. Nocturia was positively associated with CAVI in unadjusted and partially adjusted analyses, but the association was attenuated after full adjustment.
*Conclusions:* In older adults, poorer sleep restfulness and nocturia were associated with arterial stiffness, although these associations warrant cautious interpretation as an exploratory finding. CAVI also differed according to TIB category. These findings may provide additional information regarding vascular health.

## 1. Introduction

Inadequate sleep has been recognized as a risk factor for atherosclerosis. Several studies have reported that both short and long sleep duration are associated with atherosclerotic cardiovascular disease (ASCVD) and all-cause mortality [1–4]. In addition, non-restorative sleep has recently been shown to increase mortality risk in older adults [5]. Thus, sleep is commonly evaluated in terms of both duration and quality, both of which may be associated with ASCVD and mortality.

Previous studies have shown that short sleep duration [6–8], long sleep duration [9–11], or both [12] are associated with increased arterial stiffness. However, recent attention has focused on the clinical significance of time in bed (TIB), rather than sleep duration alone [13]. Prolonged TIB may increase health risks, potentially through mechanisms such as increased sleepiness, depression, and inflammation [14]. In addition, sleep quality has been implicated in arterial stiffness. Self-reported poor sleep quality has been associated with increased arterial stiffness [12,15,16], although some studies have reported no significant association [17]. Furthermore, nocturia, a common cause of sleep disruption, is associated with cardiovascular conditions such as hypertension and heart failure and has been linked to increased cardiovascular mortality [18,19].

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The cardio-ankle vascular index (CAVI) is a noninvasive and reproducible measure of arterial stiffness and has been shown to predict future ASCVD events [20–23]. However, only a limited number of studies have examined the association between sleep parameters and CAVI. While sleep duration has been evaluated in relation to CAVI [7, 24], sleep quality has been assessed in only one study [24].

Therefore, the present study aimed to investigate the associations between multidimensional sleep characteristics —including TIB, sleep duration, sleep latency, sleep restfulness score (SRS), the Japanese version of the Epworth Sleepiness Scale (JESS) [25], and nocturia —and CAVI in community-dwelling older adults.

## 2. Methods

### 2.1. Study population

This cross-sectional study included 252 older adults (≥65 years) who participated in a health promotion project conducted by Fukushima Medical University School of Health Sciences in August 2025. Measurements included CAVI, body mass index (BMI), blood pressure (BP), and heart rate (HR), along with assessment of sleep parameters (TIB, sleep duration, sleep latency, SRS, JESS, and nocturia). Participants with an ankle-brachial index (ABI) < 0.9 (n = 1) or unstable pulse waveforms that precluded accurate CAVI measurement (n = 8) were excluded. The final analysis included 243 participants. Participants completed a standardized self-administered questionnaire. Smoking exposure was assessed using the Brinkman index. Alcohol consumption was categorized as current drinker or non-drinker.

### 2.2. CAVI measurement

CAVI was measured automatically using VaSera VS-3000TE (Fukuda Denshi Co., Ltd., Tokyo, Japan) with participants in the supine position [21]. Cuffs were placed on both upper arms and ankles. Electrocardiographic electrodes were attached to both wrists, and a microphone was positioned on the sternum. BP obtained from the right arm was used for analysis.

### 2.3. Sleep assessment

TIB and sleep latency were assessed based on self-reported habitual sleep over the past month. TIB was calculated as the interval between usual bedtime and wake-up time. Sleep duration was estimated as TIB minus sleep latency. Although there are no established criteria for categorizing TIB in older adults, we categorized TIB into three groups: <7 h, 7–8 h, and ≥ 8 h, with reference to previous studies and recommending concerning sleep duration [13]. The 7–8 h category was used as the reference group. According to the consensus of the National Sleep Foundation in the United States, the recommended level of sleep duration for older adults is 7-8 h [13]. These recommendations refer to sleep duration rather than TIB. Participants rated their SRS over the past month on a 4-point scale (1 = not at all, 4 = completely) [26]. Daytime sleepiness was assessed using JESS, an 8-item self-administered questionnaire with scores ranging from 0 to 24, where higher scores indicate greater daytime sleepiness [25]. Nocturia was assessed as the number of nocturnal voids and categorized into three groups: 0, 1, ≥ 2 times per night, based on previous studies [27,28].

### 2.4. Statistical analysis

Normality was assessed using the Shapiro–Wilk test. Continuous variables are expressed as mean ± standard deviation, and categorical variables as number (percentage). Group comparisons were performed using one-way ANOVA with Bonferroni post hoc tests or chi-square tests, as appropriate. Correlations between CAVI and sleep parameters were assessed using Pearson's or Spearman's correlation coefficients. Multivariable linear regression analyses were performed to evaluate associations between CAVI and multidimensional sleep characteristics, adjusting for relevant covariates (e.g., age, sex, BP, HR). In the primary analysis, overall population analysis (n = 243) was analyzed. Exploratory subgroup analyses were performed after stratification by TIB (<7 h, 7–8 h, and ≥8 h) and sleep duration (<7 h, 7–8 h, and ≥8 h) were considered. The assumptions of the linear regression models, including linearity, homoscedasticity, and normality of residuals were assessed using standardized residual plots and normal probability plots. Multicollinearity was assessed using variance inflation factors. A p-value < 0.05 was considered statistically significant. Analyses were conducted using SPSS version 29 (IBM Corp., Armonk, NY, USA).

### 2.5. Data availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

## 3. Results

The mean age of participants was 76.4 years, and 25.9% were male. The mean CAVI was 8.90 ± 1.03. CAVI of participants classified by TIB, sleep duration, sleep latency, SRS, JESS, and nocturia are presented in Fig. 1. CAVI differed significantly across TIB categories, with higher values observed in the ≥8 h group compared with the 7–8 h group (Fig. 1a, overall $p < 0.05$). Similarly, CAVI differed across SRS categories, with higher values in participants reporting “not at all” restful sleep (Fig. 1d, overall $p < 0.01$). CAVI differed significantly across nocturia categories, with higher values observed in participants with more frequent nocturia (Fig. 1f, overall $p < 0.01$). In contrast, no significant differences were observed for sleep duration, sleep latency, or JESS (Fig. 1b, c, e).

Table 1 shows the characteristics of the participants classified by TIB. CAVI, age, TIB and sleep duration were higher, and more frequent nocturia in the “≥ 8 h” group. No significant differences were observed in other characteristics or lifestyle such as sex, BMI, BP, brinkman index, and co-morbidities among the groups. In regression analyses (Supplementary Table 1), TIB ≥ 8 h (vs. 7−8h) was associated with higher CAVI in crude models; however, this association was attenuated after adjustment for all models. In addition, as shown in Table 2, CAVI was negatively correlated with SRS in the overall population ($\beta = -0.129$, $p < 0.05$). We further conducted exploratory subgroup analyses stratified by TIB. In the exploratory subgroup analysis, the β coefficient for SRS and CAVI was −0.195 (p = 0.047) in the TIB 7–8 h group and −0.234 (p = 0.014) in Model 3 after adjustment for relevant covariates (Table 2). Sleep latency was no longer associated with CAVI after adjustment. With respect to sleep duration, no significant difference in CAVI was observed (Fig. 1b–Supplementary Table 2), and long sleep duration was not significantly associated with CAVI in the regression analyses (Supplementary Table 3). In addition, the β coefficient for sleep parameters and CAVI were 0.264 (TIB: p = 0.013), 0.215 (Sleep duration: p = 0.046), 0.054 (Sleep latency: p = 0.617), −0.260 (SRS: p = 0.015), 0.232 (JESS: p = 0.030), 0.295 (Nocturia: p = 0.006) (Supplementary Table 4).

Table 3 shows the characteristics of the participants classified by SRS. The “not at all” group consisted of only two participants (n = 2) and had higher CAVI, older age, shorter sleep duration, longer sleep latency, and higher JESS scores, as well as a lower proportion of current alcohol drinkers and more frequent nocturia. No significant differences were observed in other characteristics or lifestyle such as sex, BMI, BP, brinkman index, TIB and other co-morbidities. In multivariable regression analyses, SRS was significantly associated with CAVI in the overall population (Table 2). After excluding the two participants in the “not at all” group, the difference in CAVI across SRS categories was no longer statistically significant, and the association between SRS and CAVI was no longer statistically significant in the sensitivity analysis. In contrast,

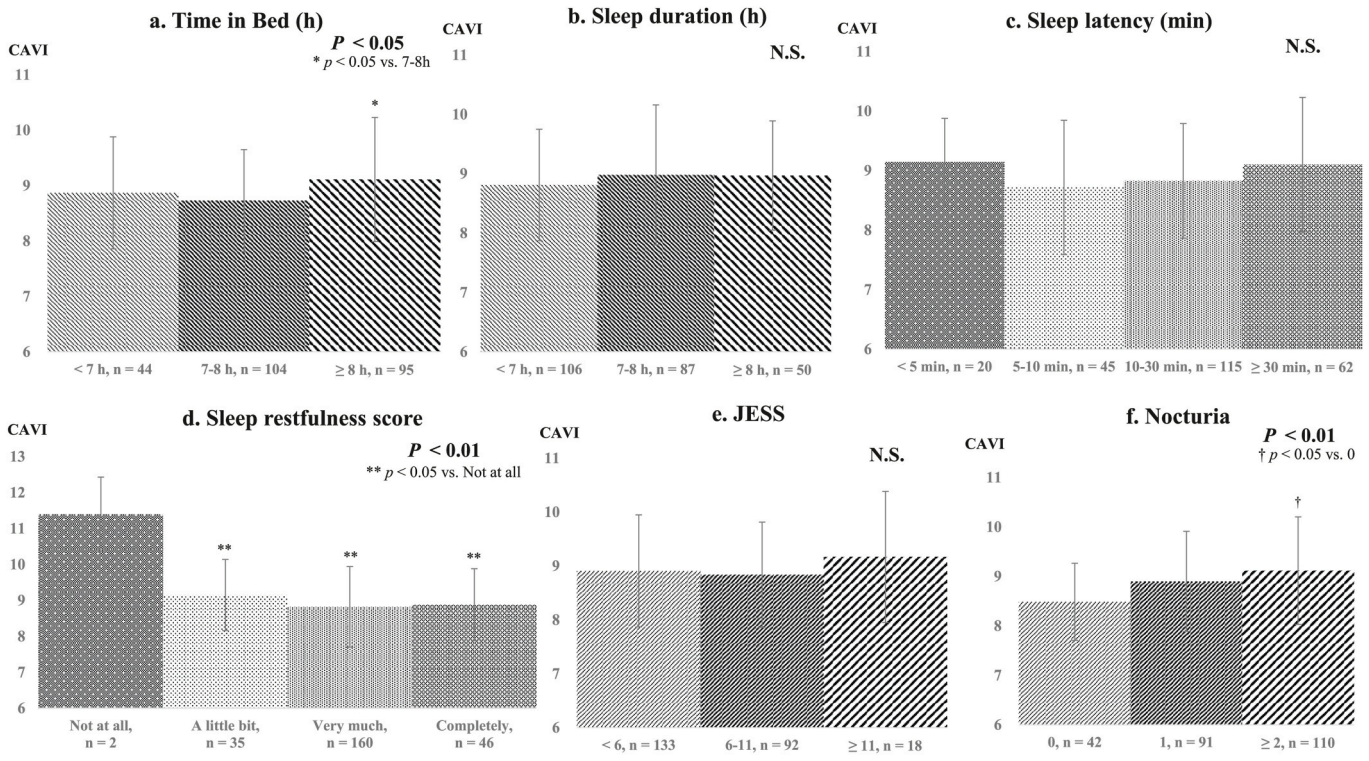



**Fig. 1. CAVI according to sleep characteristics**. CAVI are presented across categories of sleep parameters, including TIB, sleep duration, sleep latency, SRS, JESS, and nocturia.

**Table 1**
Cardio-ankle vascular index of participants classified by time in bed (n = 243).

| | Overall | Time in bed | | | *p* value |
|---|---|---|---|---|---|
| | | < 7 h | 7-8 h | ≥ 8 h | |
| **Number** | 243 | 44 | 104 | 95 | |
| **Cardio-ankle vascular index** | **8.90 ± 1.03** | **8.87 ± 1.01** | **8.73 ± 0.92** | **9.11 ± 1.12**** | **0.032** |
| **Characteristics** | | | | | |
| Age (years) | **76.4 ± 5.4** | **75.1 ± 5.0** | **75.0 ± 4.9** | **78.6 ± 5.5***, ** | **< 0.001** |
| Male, n (%) | 63 (25.9) | 10 (22.7) | 27 (26.0) | 26 (27.4) | 0.845 |
| Body mass index (kg/m$^2$) | 23.2 ± 3.2 | 23.2 ± 3.1 | 23.3 ± 3.5 | 23.1 ± 2.9 | 0.864 |
| Heart rate (bpm) | 66 ± 10 | 67 ± 10 | 65 ± 10 | 66 ± 10 | 0.369 |
| Systolic BP (mmHg) | 140.8 ± 17.8 | 141.6 ± 18.3 | 141.1 ± 17.3 | 140.0 ± 17.8 | 0.875 |
| Diastolic BP (mmHg) | 84.3 ± 9.5 | 84.6 ± 8.4 | 84.9 ± 10.2 | 83.3 ± 9.3 | 0.476 |
| Pulse pressure (mmHg) | 56.5 ± 13.1 | 56.9 ± 15.1 | 56.1 ± 12.0 | 56.7 ± 13.3 | 0.927 |
| Hypertension, n (%) | 99 (40.7) | 15 (34.1) | 40 (38.5) | 44 (46.3) | 0.324 |
| Heart failure, n (%) | 2 (0.8) | 0 (0) | 0 (0) | 2 (2.1) | 0.208 |
| Myocardial infarction, n (%) | 2 (0.8) | 0 (0) | 1 (1.0) | 1 (1.1) | 0.798 |
| Diabetes, n (%) | 30 (12.3) | 7 (15.9) | 10 (9.6) | 13 (13.7) | 0.499 |
| Dyslipidemia, n (%) | 61 (25.1) | 11 (25.0) | 24 (23.1) | 26 (27.4) | 0.784 |
| Kidney disease, n (%) | 7 (2.9) | 0 (0) | 2 (1.9) | 5 (5.3) | 0.168 |
| Sleep apnea syndrome, n (%) | 6 (2.5) | 2 (4.5) | 2 (1.9) | 2 (2.1) | 0.616 |
| Current alcohol drinker, n (%) | 78 (32.1) | 18 (40.9) | 32 (30.8) | 28 (29.5) | 0.377 |
| Brinkman index | 0 (0-0) | 0 (0-0) | 0 (0-6) | 0 (0-0) | 0.851 |
| **Sleep-related parameters** | | | | | |
| Time in bed (h) | **7.55 ± 0.99** | **6.17 ± 0.49** | **7.24 ± 0.25*** | **8.53 ± 0.63*, **** | **< 0.001** |
| Sleep duration (h) | **7.23 ± 0.99** | **5.93 ± 0.55** | **6.96 ± 0.39*** | **8.12 ± 0.73*, **** | **< 0.001** |
| Sleep latency (min) | 10 (5-30) | 10 (5-27.5) | 10 (5-20) | 15 (10-30) | 0.108 |
| Sleep restfulness score | | | | | 0.460 |
| answer1 = not at all, n (%) | 2 (0.8) | 0 (0) | 1 (1.0) | 1 (1.1) | |
| answer2 = a little bit, n (%) | 35 (14.4) | 8 (18.2) | 16 (15.4) | 11 (11.6) | |
| answer3 = very much, n (%) | 160 (65.8) | 28 (63.6) | 73 (70.2) | 59 (62.1) | |
| answer4 = completely, n (%) | 46 (18.9) | 8 (18.2) | 14 (13.5) | 24 (25.3) | |
| JESS | 5 (3-7) | 5 (2-7) | 6 (4-8) | 4 (2-7) | 0.260 |
| Nocturia (times per night) | | | | | **< 0.001** |
| 0, n (%) | **42 (17.3)** | **12 (27.3)** | **18 (17.3)** | **12 (12.6)** | |
| 1, n (%) | **91 (37.4)** | **24 (54.5)** | **43 (41.3)** | **24 (25.3)** | |
| ≥ 2, n (%) | **110 (45.3)** | **8 (18.2)** | **43 (41.3)** | **59 (62.1)** | |

BP, blood pressure; JESS, Japanese version of the Epworth sleepiness scale.
*$p < 0.05$ vs. < 7 h, **$p < 0.05$ vs. 7-8 h.

**Table 2**
Regression analyses of sleep parameters and cardio-ankle vascular index by time in bed classification (n = 243).

| | | Simple | | Model 1 | | Model 2 | | Model 3 | |
|---|---|---|---|---|---|---|---|---|---|
| | | β | *p* value | β | *p* value | β | *p* value | β | *p* value |
| **Overall** (n = 243) | **Time in bed (h)** | 0.111 | 0.084 | 0.001 | 0.991 | 0.010 | 0.868 | 0.008 | 0.891 |
| | **Sleep duration (h)** | 0.090 | 0.162 | −0.009 | 0.891 | −0.001 | 0.981 | 0.009 | 0.886 |
| | **Sleep latency (min)** | 0.098 | 0.130 | 0.072 | 0.241 | 0.043 | 0.470 | 0.036 | 0.561 |
| | **Sleep restfulness score** | **−0.129** | **0.044** | **−0.139** | **0.020** | **−0.138** | **0.018** | **−0.136** | **0.021** |
| | **JESS** | 0.041 | 0.522 | 0.056 | 0.362 | 0.080 | 0.177 | 0.082 | 0.167 |
| | **Nocturia** | **0.190** | **0.003** | 0.117 | 0.058 | **0.119** | **0.046** | 0.106 | 0.085 |
| **< 7 h** (n = 44) | **Time in bed (h)** | 0.105 | 0.498 | 0.037 | 0.801 | 0.125 | 0.371 | 0.181 | 0.204 |
| | **Sleep duration (h)** | −0.004 | 0.980 | −0.047 | 0.745 | 0.046 | 0.744 | 0.112 | 0.438 |
| | **Sleep latency (min)** | 0.309 | 0.041 | 0.255 | 0.073 | 0.221 | 0.122 | 0.187 | 0.222 |
| | **Sleep restfulness score** | 0.045 | 0.771 | 0.021 | 0.882 | 0.072 | 0.630 | 0.069 | 0.660 |
| | **JESS** | −0.133 | 0.390 | −0.052 | 0.308 | −0.080 | 0.568 | −0.098 | 0.500 |
| | **Nocturia** | 0.192 | 0.211 | 0.098 | 0.515 | 0.062 | 0.683 | 0.023 | 0.884 |
| **7-8 h** (n = 104) | **Time in bed (h)** | 0.080 | 0.418 | 0.055 | 0.557 | 0.017 | 0.860 | −0.032 | 0.741 |
| | **Sleep duration (h)** | −0.112 | 0.257 | −0.161 | 0.095 | −0.141 | 0.140 | −0.141 | 0.141 |
| | **Sleep latency (min)** | 0.182 | 0.064 | 0.221 | 0.021 | 0.177 | 0.069 | 0.145 | 0.148 |
| | **Sleep restfulness score** | −0.195 | 0.047 | −0.213 | 0.021 | −0.208 | 0.024 | −0.234 | 0.014 |
| | **JESS** | 0.164 | 0.096 | 0.091 | 0.341 | 0.106 | 0.263 | 0.116 | 0.223 |
| | **Nocturia** | 0.103 | 0.298 | 0.069 | 0.463 | 0.073 | 0.433 | 0.077 | 0.427 |
| **≥ 8 h** (n = 95) | **Time in bed (h)** | −0.055 | 0.598 | −0.092 | 0.355 | −0.074 | 0.434 | −0.091 | 0.331 |
| | **Sleep duration (h)** | 0.024 | 0.815 | 0.005 | 0.961 | −0.014 | 0.880 | 0.005 | 0.956 |
| | **Sleep latency (min)** | −0.065 | 0.533 | −0.075 | 0.451 | −0.119 | 0.212 | −0.113 | 0.278 |
| | **Sleep restfulness score** | −0.197 | 0.056 | −0.159 | 0.109 | −0.143 | 0.130 | −0.125 | 0.187 |
| | **JESS** | 0.075 | 0.467 | 0.116 | 0.255 | 0.151 | 0.126 | 0.186 | 0.056 |
| | **Nocturia** | 0.224 | 0.029 | 0.187 | 0.063 | 0.195 | 0.043 | 0.172 | 0.085 |

JESS, Japanese version of the Epworth sleepiness scale.
Model 1: adjusted for age and sex.
Model 2: adjusted for Model 1 + body mass index, heart rate, systolic blood pressure.
Model 3: adjusted for Model 2 + hypertension, diabetes, dyslipidemia, current alcohol drink, brinkman index.

**Table 3**
Cardio-ankle vascular index of participants classified by sleep restfulness score (n = 243).

| | Overall | Sleep restfulness score | | | | *p* value |
|---|---|---|---|---|---|---|
| | | Not at all | A little bit | Very much | Completely | |
| **Number** | 243 | 2 | 35 | 160 | 46 | |
| **Cardio-ankle vascular index** | **8.90 ± 1.03** | **11.40 ± 0.99** | **9.15 ± 1.12*** | **8.82 ± 1.00*** | **8.88 ± 0.92*** | **0.002** |
| **Characteristics** | | | | | | |
| Age (years) | **76.4 ± 5.4** | **89.0 ± 5.7** | **74.9 ± 5.2*** | **76.4 ± 5.0*** | **76.7 ± 6.3*** | **0.003** |
| Male, n (%) | 63 (25.9) | 0 (0) | 7 (20.0) | 45 (28.1) | 11 (23.9) | 0.606 |
| Body mass index (kg/m$^2$) | 23.2 ± 3.2 | 20.8 ± 4.3 | 23.2 ± 3.0 | 23.1 ± 3.3 | 23.5 ± 2.9 | 0.655 |
| Heart rate (bpm) | 66 ± 10 | 66 ± 10 | 64 ± 10 | 66 ± 10 | 67 ± 10 | 0.733 |
| Systolic BP (mmHg) | 140.8 ± 17.8 | 142.5 ± 6.4 | 143.7 ± 20.4 | 140.3 ± 17.2 | 140.2 ± 18.3 | 0.779 |
| Diastolic BP (mmHg) | 84.3 ± 9.5 | 81.0 ± 2.8 | 85.1 ± 7.8 | 84.3 ± 9.9 | 83.7 ± 9.7 | 0.827 |
| Pulse pressure (mmHg) | 56.5 ± 13.1 | 61.5 ± 3.5 | 58.6 ± 15.5 | 56.0 ± 12.6 | 56.5 ± 13.1 | 0.713 |
| Hypertension, n (%) | 99 (40.7) | 0 (0) | 14 (40.0) | 62 (38.8) | 23 (50.0) | 0.351 |
| Heart failure, n (%) | 2 (0.8) | 0 (0) | 1 (2.9) | 1 (0.6) | 0 (0) | 0.522 |
| Myocardial infarction, n (%) | **2 (0.8)** | **1 (50)** | **0 (0)** | **1 (0.6)** | **0 (0)** | **< 0.001** |
| Diabetes, n (%) | 30 (12.3) | 0 (0) | 6 (17.1) | 18 (11.3) | 6 (13.0) | 0.747 |
| Dyslipidemia, n (%) | 61 (25.1) | 0 (0) | 9 (25.7) | 38 (23.8) | 14 (30.4) | 0.676 |
| Kidney disease, n (%) | 7 (2.9) | 0 (0) | 3 (8.6) | 3 (1.9) | 1 (2.2) | 0.189 |
| Sleep apnea syndrome, n (%) | 6 (2.5) | 0 (0) | 3 (8.6) | 3 (1.9) | 0 (0) | 0.076 |
| Current alcohol drinker, n (%) | **78 (32.1)** | **0 (0)** | **5 (14.3)** | **60 (37.5)** | **13 (28.3)** | **0.037** |
| Brinkman index | 0 (0-0) | 0 (0-0) | 0 (0-0) | 0 (0-0) | 0 (0-0) | 0.345 |
| **Sleep-related parameters** | | | | | | |
| Time in bed (h) | 7.55 ± 0.99 | 7.50 ± 0.71 | 7.53 ± 1.20 | 7.51 ± 0.92 | 7.70 ± 1.07 | 0.728 |
| Sleep duration (h) | **7.23 ± 0.99** | **6.75 ± 1.06** | **6.86 ± 1.14** | **7.23 ± 0.90** | **7.52 ± 1.07**** | **0.024** |
| Sleep latency (min) | **10 (5-30)** | **37.5 (30-45)** | **17.5 (10-43)** | **10 (10-20)**** | **10 (5-16)**** | **<0.001** |
| JESS | **5 (3-7)** | **7 (0-14)** | **6 (4-10)** | **5 (3-7)** | **3 (1-6)** | **0.001** |
| Nocturia (times per night) | | | | | | **0.037** |
| 0, n (%) | **42 (17.3)** | **0 (0)** | **2 (5.7)** | **28 (17.5)** | **12 (26.1)** | |
| 1, n (%) | **91 (37.4)** | **0 (0)** | **10 (28.6)** | **67 (41.9)** | **14 (30.4)** | |
| ≥ 2, n (%) | **110 (45.3)** | **2 (100.0)** | **23 (65.7)** | **65 (40.6)** | **20 (43.5)** | |

BP, blood pressure; JESS, Japanese version of the Epworth sleepiness scale answer1 = not at all, answer2 = a little bit, answer3 = very much, answer4 = completely.
*$p < 0.05$ vs. not at all, **$p < 0.05$ vs. a little bit, †$p < 0.05$ vs. very much.

robust regression analysis using MM-estimation showed statistically significant associations after adjustment (Supplementary Table 5–7).

Table 4 shows the characteristics of the participants classified by nocturia. The "≥ 2" group presented higher CAVI, older, more likely to be male, lower prevalence of dyslipidemia, had longer TIB and sleep duration, lower SRS, and higher JESS. In contrast, no significant

**Table 4**
Cardio-ankle vascular index of participants classified by nocturia (n = 243).

| | Overall | Nocturia | | | *p* value |
|---|---|---|---|---|---|
| | | 0 | 1 | ≥ 2 | |
| **Number** | 243 | 42 | 91 | 110 | |
| **Cardio-ankle vascular index** | **8.90 ± 1.03** | **8.49 ± 0.78** | **8.90 ± 1.01** | **9.06 ± 1.09**** | **0.008** |
| **Characteristics** | | | | | |
| Age (years) | **76.4 ± 5.4** | **75.1 ± 5.5** | **75.5 ± 5.2** | **77.6 ± 5.4*, **** | **0.005** |
| Male, n (%) | **63 (25.9)** | **8 (19.0)** | **17 (18.7)** | **38 (34.5)** | **0.020** |
| Body mass index (kg/m$^2$) | 23.2 ± 3.2 | 22.8 ± 2.5 | 23.7 ± 3.4 | 22.9 ± 3.2 | 0.116 |
| Heart rate (bpm) | 65.9 ± 10.3 | 66.6 ± 9.3 | 67.2 ± 10.7 | 64.5 ± 10.2 | 0.159 |
| Systolic BP (mmHg) | 140.8 ± 17.8 | 137.4 ± 16.1 | 142.5 ± 17.2 | 140.6 ± 18.8 | 0.309 |
| Diastolic BP (mmHg) | 84.3 ± 9.5 | 83.8 ± 10.1 | 85.6 ± 8.8 | 83.3 ± 9.9 | 0.249 |
| Pulse pressure (mmHg) | 56.5 ± 13.1 | 53.6 ± 11.4 | 56.9 ± 12.9 | 57.3 ± 13.8 | 0.270 |
| Hypertension, n (%) | 99 (40.7) | 16 (38.1) | 32 (35.2) | 51 (46.4) | 0.255 |
| Heart failure, n (%) | 2 (0.8) | 0 (0) | 1 (1.1) | 1 (0.9) | 0.801 |
| Myocardial infarction, n (%) | 2 (0.8) | 0 (0) | 0 (0) | 2 (1.8) | 0.295 |
| Diabetes, n (%) | 30 (12.3) | 5 (11.9) | 7 (7.7) | 18 (16.4) | 0.176 |
| Dyslipidemia, n (%) | **61 (25.1)** | **18 (42.9)** | **23 (25.3)** | **20 (18.2)** | **0.007** |
| Kidney disease, n (%) | 7 (2.9) | 1 (2.4) | 1 (1.1) | 5 (4.5) | 0.340 |
| Sleep apnea syndrome, n (%) | 6 (2.5) | 3 (7.1) | 2 (2.2) | 1 (0.9) | 0.084 |
| Current alcohol drinker, n (%) | 78 (32.1) | 17 (40.5) | 29 (31.9) | 32 (29.1) | 0.404 |
| Brinkman index | 0 (0-0) | 0 (0-0) | 0 (0-0) | 0 (0-85) | 0.494 |
| **Sleep-related parameters** | | | | | |
| Time in bed (h) | **7.55 ± 0.99** | **7.17 ± 0.96** | **7.32 ± 1.00** | **7.88 ± 0.90*, **** | **<0.001** |
| Sleep duration (h) | **7.23 ± 0.99** | **6.95 ± 0.99** | **7.04 ± 1.00** | **7.49 ± 0.92*, **** | **<0.001** |
| Sleep latency (min) | **10 (5-30)** | **10 (5-16)** | **10 (10-20)** | **10 (5-30) *** | **0.039** |
| Sleep restfulness score | | | | | **0.037** |
| answer1 = not at all, n (%) | **2 (0.8)** | **0 (0)** | **0 (0)** | **2 (0.8)** | |
| answer2 = a little bit, n (%) | **35 (14.4)** | **2 (4.8)** | **10 (11.0)** | **23 (20.9)** | |
| answer3 = very much, n (%) | **160 (65.8)** | **28 (66.7)** | **67 (73.6)** | **65 (59.1)** | |
| answer4 = completely, n (%) | **46 (18.9)** | **12 (28.6)** | **14 (15.4)** | **20 (18.2)** | |
| JESS | **5 (3-7)** | **4 (3-6)** | **5 (3-7)** | **6 (2-8)** | **0.035** |

BP, blood pressure; JESS, Japanese version of the Epworth sleepiness scale.
Brinkman index = number of cigarettes per day × years of smoking * $p < 0.05$ vs. 0, **$p < 0.05$ vs. 1.

differences were observed in other characteristics or lifestyle such as BMI, BP, brinkman index, and other co-morbidities. Nocturia showed a significant positive association with CAVI in the unadjusted model and Model 2 in the overall population (Table 2). In the exploratory subgroup analysis, the β coefficient for nocturia was 0.224 ($p = 0.029$) in the TIB ≥8 h group (Table 2). Furthermore, regression analyses between nocturia and sleep parameters showed significant associations for multiple sleep parameters including TIB, sleep duration, sleep latency, and SRS across all models (Table 5).

## 4. Discussion

To our knowledge, this study is the first to suggest that sleep restfulness and nocturia may be associated with arterial stiffness in community-dwelling older adults. CAVI was also higher among participants with prolonged TIB in the unadjusted categorical analysis, although this association was not independent after multivariable adjustment. In contrast, sleep duration, sleep latency, and daytime sleepiness were not significantly associated with CAVI. These findings suggest that multidimensional sleep characteristics may provide information beyond sleep duration alone when considering vascular health.

Sleep is commonly assessed in terms of duration and quality, both of which have been reported to be significantly associated with arterial stiffness. Short sleep duration [6–8], as well as long sleep duration [9–11,29] have been associated with increased arterial stiffness in several cohorts. Previous studies have suggested a U-shaped association between sleep duration and arterial stiffness [12,30]. However, in this study, such a U-shaped pattern was observed for TIB but not for sleep duration, suggesting that TIB may capture aspects of sleep behavior that are not fully reflected by sleep duration in older adults. Long TIB has been recently reported to be more strongly associated with mortality than sleep duration in older adults [5]. These findings suggest that both TIB and sleep restfulness may be important for vascular health, although the exploratory subgroup findings should be interpreted cautiously. In the present study, when TIB was categorized, the β coefficient for SRS and CAVI was −0.195 (simple: p = 0.047) and −0.234 (model 3: p = 0.014). However, these findings should be interpreted with caution because multiple sleep-related variables and regression models were

**Table 5**
Regression analyses of sleep parameters and nocturia (n = 243).

| | | Simple | | Model 1 | | Model 2 | | Model 3 | |
|---|---|---|---|---|---|---|---|---|---|
| | | β | *p* value | β | *p* value | β | *p* value | β | *p* value |
| **Overall** | **Time in bed (h)** | **0.296** | **<0.001** | **0.260** | **<0.001** | **0.266** | **<0.001** | **0.264** | **<0.001** |
| | **Sleep duration (h)** | **0.226** | **<0.001** | **0.180** | **0.005** | **0.185** | **0.004** | **0.195** | **0.002** |
| | **Sleep latency (min)** | **0.163** | **0.011** | **0.171** | **0.007** | **0.175** | **0.007** | **0.176** | **0.007** |
| | **Sleep restfulness score** | **−0.174** | **0.006** | **−0.183** | **0.003** | **−0.172** | **0.006** | **−0.164** | **0.009** |
| | **JESS** | 0.091 | 0.159 | 0.083 | 0.192 | 0.085 | 0.187 | 0.083 | 0.189 |

JESS, Japanese version of the Epworth sleepiness scale.
Model 1: adjusted for age and sex.
Model 2: adjusted for Model 1 + body mass index, heart rate, systolic blood pressure.
Model 3: adjusted for Model 2 + hypertension, diabetes, dyslipidemia, current alcohol drink, brinkman index.

evaluated, which may increase the possibility of chance findings. These findings might suggest that sleep restfulness may be relevant to vascular health (i.e. lower CAVI), although the exploratory subgroup findings should be interpreted cautiously. Because no established cutoff values for optimal TIB in older adults are currently available, the TIB categories used in this study were based on sleep-duration literature, and their appropriateness for TIB itself remains uncertain. The association between SRS and CAVI was observed in the 7–8 h TIB group; however, this finding should be interpreted cautiously because the subgroup analysis was exploratory and did not remain statistically significant after correction for multiple comparisons. Additionally, the categorization of TIB used in the present study should be interpreted with caution. Because no established cutoff values for optimal TIB in older adults are currently available, the TIB categories used in this study were based on a previous study that categorized sleep duration into <7 h, 7−8 h, and ≥8 h [13]. Although this approach allowed us to examine TIB using previously established categories, whether these cutoff values are appropriate for TIB itself remains uncertain. Further studies are needed to determine clinically meaningful TIB ranges in older adults.

Several mechanisms have been proposed to explain the association between long sleep duration and increased cardiovascular risk. Long sleep duration (≥8 h) accompanied by high-sensitivity C-reactive protein levels has been shown to be significantly associated with increased arterial stiffness in older subjects of high risk of ASCVD [11]. Furthermore, prolonged sleep duration caused by sleep fragmentation and nocturnal awakenings may activate the sympathetic nervous system [31,32]. Sleep fragmentation and sympathetic overactivity have been associated with development of ASCVD [33,34]. The mechanism linking sleep quality to arterial stiffness remains unclear. However, several studies have reported an association between poor sleep quality and increased arterial stiffness. Self-reported poor sleep quality has been associated with higher arterial stiffness in middle-aged healthy adults and in patients with type 2 diabetes [12,15], whereas findings in hypertensive patients were less consistent [17]. A prospective study reported that poor sleep quality was related to the progression of arterial stiffness over a 3-year period, independent of cardiovascular risk factors such as BP, apnea-hypopnea index, and heart rate variability [16]. Aging is associated with not only arteriosclerosis but also chronobiological disruptions and changes in sleep duration and quality, which may predispose older individuals to sleep deprivation [35]. In addition, long sleep duration has been associated with reduced sleep quality and high stress levels [31] Previous studies, including the Sleep Heart Health Study, have reported that sleep restfulness is associated with objective sleep stability [36–38]. Although there is no single measure that adequately reflects the degree to which sleep is physiologically restorative, the sense of restfulness following sleep may play an important role in determining whether sleep confers health benefits [39–41]. Notably, a previous study reported that when sleep restfulness was considered simultaneously, individuals who felt unrested despite objective long TIB had an increased risk of mortality [5]. Therefore, differences in sleep restfulness might be associated with health outcomes even among individuals with similar sleep duration or TIB [5].

Nocturia has been associated with increased overall mortality risk, particularly through ASCVD [18,19,42], and has also been linked to increased arterial stiffness as assessed by pulse wave velocity (PWV) [28,43]. In a study of men with lower urinary tract symptoms or nocturia, those with nocturia had higher brachial-ankle PWV (baPWV), particularly among men aged <70 years, and the coexistence of nocturia ≥ 3 times per night and elevated baPWV (>1800 cm/s) was associated with an increased risk of ASCVD [43]. In addition, a community-based study demonstrated a linear positive association between nocturnal urination frequency and baPWV, which remained significant after adjustment for major covariates, including office and sleep BP [28]. Similarly, our study showed that higher nocturia frequency was significantly associated with increased CAVI.

An observational study [44] reported reduced blood perfusion of the bladder neck in patients with lower urinary tract symptoms, which was inversely associated with nocturnal urination frequency. Experimental evidence has shown that bladder ischemia due to atherosclerosis induces structural and functional changes, including reduced compliance and hyper-reflexia [45]. Some studies have suggested that nocturia may reflect, rather than cause, underlying arterial stiffness [28]. Furthermore, Nocturia may impair sleep quality [42,46], leading to activation of the sympathetic nervous system and increased cardiovascular stress. However, as most previous studies were cross-sectional in design, it is difficult to infer causal relationships among sleep disturbance, nocturia, and arterial stiffness.

In particular, these findings regarding SRS and nocturia may provide exploratory insights into the relationship between sleep characteristics and arterial stiffness, although they should be interpreted with caution.

### 4.1. Study limitations

This study has several limitations. First, this study had a cross-sectional design, and causal relationships between sleep characteristics and arterial stiffness could not be determined. Second, sleep parameters were assessed using self-reported questionnaires rather than objective measures (i.e. actigraphy or polysomnography). In addition, other sleep patterns, such as daytime resting behaviors including bed rest or daytime napping or social jet lag, were not evaluated in this study. Although previous studies have reported that subjective sleep duration correlates with objectively measured sleep duration [47,48], objective assessment methods may provide more precise evaluation [49–51]. Third, group sizes were uneven, particularly for SRS categories, with only two participants classified as “not at all”. Because age differed across SRS categories, particularly in the “not at all” category, age was considered an important potential confounder. Sleep restfulness was associated with CAVI after adjustment for age and other cardiovascular risk factors. However, in a sensitivity analysis excluding the two participants in the “not at all” category, the association was no longer statistically significant. Therefore, the findings regarding sleep restfulness should be considered exploratory and hypothesis-generating, particularly given the very small sample size of the “not at all” category. Fourth, although several potential confounding variables were adjusted for in the multivariable analyses, the possibility of residual or unmeasured confounding cannot be completely ruled out. Potential confounders not assessed in this study included urological disease, depression, and medication use. Thus, future longitudinal studies with larger sample size using objective measurements are needed to clarify whether TIB, SRS and other sleep-related factors are associated with arterial stiffness and future ASCVD.

## 5. Conclusion

In older adults, poorer sleep restfulness and more frequent nocturia were associated with higher arterial stiffness, although this finding was sensitive to the inclusion of two participants with very poor sleep restfulness. More frequent nocturia was associated with higher CAVI in unadjusted and partially adjusted analyses, but this association was attenuated after full multivariable adjustment. Sleep duration, sleep latency, and daytime sleepiness were not significantly associated with arterial stiffness. CAVI also differed according to TIB category, although this association was not independent after multivariable adjustment. These exploratory findings may provide additional information regarding vascular health.

## CRediT authorship contribution statement

**Takumu Watanabe:** Writing – original draft, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Yuko Horikoshi:** Writing – review & editing, Writing – original draft, Visualization, Supervision, Software, Resources, Project administration,

Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Rie Sakuraba-Hirata:** Writing – review & editing, Supervision, Methodology, Investigation, Funding acquisition, Data curation, Conceptualization. **Noa Goto:** Writing – review & editing, Methodology, Investigation, Formal analysis, Data curation. **Kenshin Sakawaki:** Writing – review & editing, Methodology, Investigation, Formal analysis, Data curation. **Miho Noguchi:** Writing – review & editing, Methodology, Investigation, Formal analysis, Data curation. **Riku Yamaguchi:** Writing – review & editing, Methodology, Investigation, Formal analysis, Data curation. **Niina Suzuki:** Writing – review & editing, Methodology, Investigation, Formal analysis, Data curation. **Toa Kotani:** Writing – review & editing, Methodology, Investigation, Formal analysis, Data curation. **Maki Ogasawara:** Writing – review & editing, Supervision, Project administration, Methodology, Investigation, Data curation, Conceptualization. **Toshimi Sato:** Writing – review & editing, Supervision, Project administration, Methodology, Investigation, Data curation, Conceptualization. **Tatsuya Nakanowatari:** Writing – review & editing, Supervision, Project administration, Methodology, Investigation, Data curation, Conceptualization. **Toshimasa Sone:** Writing – review & editing, Supervision, Resources, Project administration, Methodology, Investigation, Funding acquisition, Data curation, Conceptualization. **Yoshitaka Shiba:** Writing – review & editing, Supervision, Resources, Project administration, Methodology, Investigation, Funding acquisition, Data curation, Conceptualization. **Yasuchika Takeishi:** Writing – review & editing, Supervision, Resources, Conceptualization. **Akiomi Yoshihisa:** Writing – review & editing, Supervision, Resources, Methodology, Investigation, Funding acquisition, Data curation, Conceptualization.

## Ethics declaration

Written informed consent to take part in the study and to publish the article has been obtained from all participants or their legal representatives. The privacy rights of participants have been observed.

This study was performed in compliance with relevant laws, regulatory frameworks and guidelines where the research took place. This study was approved by the Ethics Committee of Fukushima Medical University. (Approval No. 2022-123)

## Declaration of generative AI and AI-assisted technologies in the manuscript preparation process

During the preparation of this work, the authors used ChatGPT (Open AI) for English language editing and proofreading of the manuscript. The authors reviewed and edited the output as needed and take full responsibility for the content of the published article.

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## Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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## Appendix A. Supplementary data

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