Sleep Fragmentation, Awake Blood Pressure, and Sleep-Disordered Breathing in a Population-based Study

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Arousal from sleep produces transient increases in systemic blood pressure, leading to the suggestion that repeated arousals are associated with a sustained increase in daytime blood pressure. Using data from the Wisconsin Sleep Cohort Study, a population-based study, we tested the hypothesis that sleep fragmentation is associated with elevated awake blood pressure. Sleep, breathing, and seated blood pressure measurements from 1,021 participants (age 42 $\pm$ 8 yr; 590 males) were analyzed. Sleep fragmentation was defined as the total number of awakenings and shifts to Stage 1 sleep divided by the total sleep time (sleep fragmentation index: SFI). To reduce the confounding influence of sleep-disordered breathing, which is related to both increased daytime blood pressure and sleep fragmentation, all participants with an apnea–hypopnea index (AHI) $\geq$ 1 were analyzed separately. Accounting for the influences of sex, age, body mass index, and antihypertensive medication use, the SFI was significantly associated with higher levels of awake systolic blood pressure in people with an AHI $<$ 1; a 2 standard deviation increase in the SFI was associated with a 3.1 mm Hg rise in awake systolic blood pressure. In participants with an AHI $\geq$ 1, there was no independent association between the SFI and awake blood pressure after controlling for the influence of the AHI.

Arousal from sleep has been shown to produce abrupt increases in systemic blood pressure (1, 2). In patients with sleep-disordered breathing, the termination of each apnea–hypopnea is associated with an increase in systemic blood pressure, and typically with a lightening in sleep state and/or an arousal from sleep (3). In these patients, who may have hundreds of apneic episodes per night, the changes in arterial pressure can occur in the absence of airway occlusion (4) and hypoxemia (1). These findings have led to the suggestion that perturbations in conscious state are responsible for the acute increases in systemic blood pressure. Furthermore, there is evidence that sleep-disordered breathing is independently associated with elevated daytime blood pressure (5–8). Therefore, it is reasonable to suggest that repeated arousals from sleep may also be associated with elevated daytime blood pressure independent of sleep-disordered breathing or other physiological insults.

The aim of the present study was to investigate the relationship between arousals from sleep and daytime blood pressure in a population-based sample of middle-aged adults. We tested the hypothesis that the amount of sleep fragmentation was significantly associated with a measurement of awake systemic blood pressure independent of age, sex, body mass index, and occurrence of sleep-disordered breathing. A secondary hypothesis was that the higher levels of blood pressure recorded during wakefulness, which were related to sleep-disordered breathing (8), could be in part due to an increase in sleep fragmentation. To investigate these hypotheses we used data from the Wisconsin Sleep Cohort Study, an ongoing, prospective study of sleep-disordered breathing.

METHODS

The data used in this investigation were collected as part of the Wisconsin Sleep Cohort Study. A two-stage sampling procedure was used to construct the cohort. All men and women, aged 30–60 yr, employed at one of five state agencies in south-central Wisconsin were surveyed on sleep characteristics and sociodemographic factors. A probability sample was then drawn from survey respondents. Full details of the selection procedures for the Wisconsin Sleep Cohort Study are given elsewhere (9).

All participants underwent polysomnography conducted in a dedicated sleep research laboratory equipped with comfortable bedrooms. Each participant arrived at the sleep laboratory in the early evening and was interviewed; any use of antihypertensive medication was noted. Prior to the sleep study, height and weight (without shoes) were measured with standard procedures (10) and used to derive body mass index (BMI; kg/m^2). Two or three measurements of systemic blood pressure were taken with a sphygmomanometer according to the American Heart Association recommendations (11), following 15 min of rest in the seated position; the mean of these was taken as a single point measurement of the awake blood pressure. Transducers for polysomnography were then attached after which participants were free to relax (watch television, read, etc.) until they were ready to go to sleep. Participants were allowed to sleep for as long as they wished.

Sleep state was recorded using the standard placements of electroencephalogram (EEG: C3–A2, C4–A1), electrooculogram (EOG), and electromyogram (Chin EMG); measurements of breathing were obtained from nasal/oral temperature changes (thermistor, Breathsensor; Edentec, Täby, Sweden) and from ribcage plus abdominal movements (respiratory inductance plethysmography, Respitrace; Ambulatory Monitoring, Ardsley, NY). Arterial oxygen saturation was monitored using pulse oximetry (Biox 3700; Ohmeda, Louisville, CO); heart rate was calculated from the electrocardiogram (ECG).

Each sleep study was analyzed by a trained technician and reviewed by one of two sleep clinicians. Sleep stages were scored using conventional criteria (12). A sleep fragmentation index (SFI) was calculated as the total number of awakenings/shifts to Stage 1 (from deeper non-rapid eye movement [NREM] or REM sleep) divided by the total sleep time in hours. Breathing events with no airflow for at least 10 s were scored as apneas and events with at least a 40% reduction in tidal volume accompanied by a 4% decrease in oxygen saturation were scored as hypopneas. The total number of apneas and hypopneas, divided by the number of hours of sleep (apnea–hypopnea index, AHI), was used as a measure of the severity of sleep-disordered breathing.

SFI was calculated as the total number of awakenings/shifts to Stage 1 (from deeper NREM or REM sleep) divided by the total sleep time in hours. In addition, arousals of a short duration were scored in a subset of 25 sleep studies using standard criteria (16) modified to include any arousals lasting $>$ 2 s, as opposed to the standard criterion

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TABLE 1

CHARACTERISTICS OF THE SAMPLE (n = 1,021)

Characteristic	Value
Sex	
Women, n (%)	431 (42)
Men, n (%)	590 (58)
Age, yr, mean ± SD	45 ± 8
Body mass index, kg/m^2, mean ± SD	29 ± 6
Apnea–hypopnea index, events/h; median, min, max	1 (0, 97)
Systolic blood pressure, mm Hg, mean ± SD	126 ± 14
Diastolic blood pressure, mm Hg, mean ± SD	82 ± 10
Hypertensive* participants, n (%)	135 (13)
Participants using antihypertensive medication, n (%)	117 (11)

* Defined as systolic blood pressure ⩾ 140 or diastolic blood pressure ⩾ 90 or antihypertensive medication use.

of > 3 s. This modification was made to ensure that even short duration increases in EEG frequency were captured in this subanalysis. A microarousal index (micro-AI) was calculated as the total number of short duration arousals divided by the total sleep time in hours.

To assess the reproducibility of sleep patterns and SFI recorded in the sleep laboratory, 56 participants were studied twice within a short time span (the median time between the two measures was 2 wk). On the second night, blood pressure measurements were not taken. The reproducibility in the mean SFI between the first and the second sleep study was assessed.

Statistical Analysis

The data collected in this study were analyzed using SAS (13) for descriptive statistics (parametric and nonparametric data) and contingency tables (categorical data). Multiple linear regression was performed using SUDAAN (14) software, with analyses weighted to give unbiased estimates and appropriate standard errors in order to account for the stratified sampling of the sleep cohort.

The SFI was categorized into quartiles to initially examine its relationship with age, sex, body mass index, and blood pressure. Statistically significant differences across quartiles were assessed using Chi square tests, *F* tests, and Kruskal–Wallis tests as appropriate. To estimate the relationship between the SFI and the measurement of awake blood pressure, two multiple linear regression models were analyzed, stratified by the presence or absence of an AHI (AHI < 1, AHI ⩾ 1). This approach was taken to account for the possibility that sleep-disordered breathing may alter or mask any effect of sleep fragmentation on blood pressure. For individuals without sleep-disordered breathing (AHI < 1), the analysis was adjusted for the confounding factors of age, sex, body mass index, and antihypertensive medication use (i.e., all potentially vasoactive medications). For individuals with AHI ⩾ 1, the analysis was adjusted for the confounding factors mentioned above and additionally for the AHI. The significance of the multiple linear regression coefficients was assessed with *t* tests. Results with a p value < 0.05 were considered significant. Residual plots were examined to assess the compliance of the statistical model assumptions.

The night-to-night variability of the SFI was assessed using a paired *t* test to look for consistent changes in the SFI between the first and the second sleep study. The correlation coefficient for these repeat measures was also computed. Comparability of the micro-AI and SFI was investigated by computing the correlation coefficient for these two measures and the significance of any differences was assessed using a paired *t* test.

RESULTS

One thousand and twenty-one people were studied; the characteristics of the sample are given in Table 1. Descriptive statistics for the total sleep time, percentage of time spent in each sleep stage, and the SFI are given in Table 2. The percentage of time spent in Stage 3/4 and REM sleep was similar to that recorded in other studies (9, 15).

Sleep Fragmentation Index (SFI)

For the participants who underwent sleep studies on two nights, the SFI data are shown in Figure 1. The paired *t* test showed that there was no significant difference between the SFI for the first and second nights (mean ± SD; 8.14 ± 4.18 events/h of sleep and 8.13 ± 4.31 events/h of sleep, respectively); the correlation coefficient for the paired measures was 0.73 ($p < 0.01$). These statistics indicate good test–retest reproducibility for SFI in this subgroup of participants, suggesting that the SFI recorded on a single night is representative of a usual night's sleep in the laboratory.

The relationship between the SFI and the micro-AI is shown in Figure 2. For all participants the micro-AI was greater than the number of overt changes in the sleep/wake state (mean ± SD micro-AI, 22.02 [± 9.85] events/h of sleep; SFI, 8.14 [± 4.18] events/h of sleep; $p > 0.01$). However, the two variables were significantly correlated ($r = 0.54$; $p = 0.005$) indicating that quantification of sleep disruption by SFI is related to another, more sensitive index of arousal.

Effects of Sleep Fragmentation on Our Measurement of Awake Systemic Blood Pressure

The details of participant characteristics and blood pressure data with respect to SFI quartiles are given in Table 3. Note that as sleep became progressively more fragmented, there was a concurrently higher level of both systolic and diastolic blood pressure, and an increasing number of people on antihypertensive medication. Sleep fragmentation was more prominent in males and was positively associated with both the AHI and ageing.

Multiple linear regression analysis of data from individuals without sleep-disordered breathing (AHI < 1) was used to

TABLE 2

SLEEP CHARACTERISTICS OF THE SAMPLE

Characteristic	Participants with AHI < 1 (*n = 483*)	Participants with AHI ⩾ 1 (*n = 538*)
Total sleep time, h, mean ± SD	6.1 ± 1.1	5.9 ± 1.0
Sleep fragmentation index,* event/h, mean ± SD	4.8 ± 2.2	5.9 ± 3.1
Stage 1 sleep†, %, mean ± SD	9.0 ± 5.7	10.8 ± 6.2
Stage 2 sleep†, %, mean ± SD	61.3 ± 9.8	62.1 ± 9.7
Stage 3/4 sleep†, %, mean ± SD	11.6 ± 8.4	9.9 ± 8.4
REM sleep†, %, mean ± SD	18.0 ± 6.0	17.1 ± 6.2

Definition of abbreviations: AHI = apnea–hypopnea index; REM = rapid eye movement.

* Total number of awakenings/shifts to Stage 1 (from deeper NREM or REM sleep) divided by the total sleep time in hours, minimum = 1.1/h.

† Sleep stages are expressed as a percentage of the total sleep time.

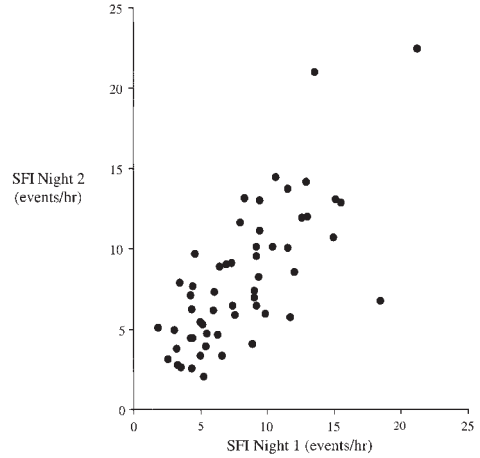

Figure 1. For each participant the sleep fragmentation index (SFI: total number of awakenings/shifts to Stage 1, from deeper NREM or REM sleep, divided by the total sleep time in hours) obtained on the first night of study in the sleep laboratory (Night 1) is plotted against the SFI obtained on a subsequent night (Night 2).

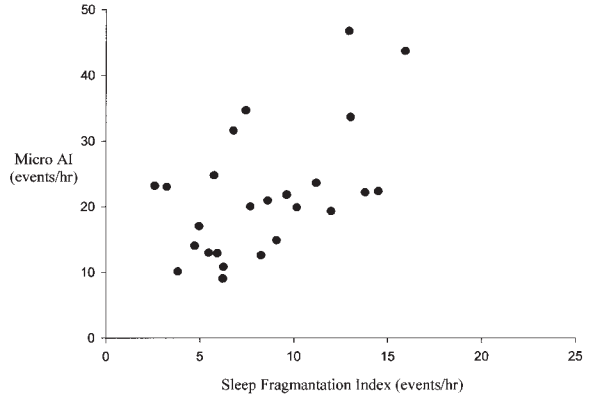

Figure 2. For each participant the sleep fragmentation index (SFI) is plotted against the corresponding arousal index (micro-AI: number of microarousals per hour of sleep), where each index was calculated from the same record.

evaluate the independent effect of SFI on systolic and diastolic blood pressure adjusted for sex, age, BMI, and antihypertensive medication use; the results are shown in Table 4. There was a significant association between the SFI and the awake measurement of systolic but not diastolic blood pressure. A 1 event increase in the SFI/h was associated with a higher level (0.62 mm Hg) of awake systolic blood pressure; thus an individual with 5 SFI units (i.e., 2 standard deviations) more per night would on average have a 3.1 mm Hg higher systolic BP.

For individuals with an AHI $\geqslant$ 1, the results of the same multiple linear regression analysis as described above, but adjusted for the AHI, are shown in Table 5. For these data, we found no relationship between the SFI and the measurement of awake systolic or diastolic blood pressure.

DISCUSSION

We have investigated the relationship between sleep fragmentation and a measurement of awake systemic blood pressure in a population-based sample of middle-aged adults. The severity of sleep-disordered breathing in the population was variable; typically it was mild. We found that in adults without overt sleep-disordered breathing (AHI $<$ 1), the amount of fragmented sleep was independently associated with significant higher levels of awake systolic blood pressure. This effect of SFI was not discernible in adults with an AHI $>$ 1.

In the present study, we used an index of sleep fragmentation based on conventional scoring of 30-s epochs of EEG; thus only changes in sleep state lasting greater than 15 s were recorded in this index. By ignoring more transient arousals, it is possible that the SFI was too crude to allow the relationship between changes in sleep state and systemic blood pressure to be fully explored. Indeed inspection of Table 3 shows that participants with a relatively high AHI could have had a low SFI. However, our comparison of the SFI and micro-AI (based on 2-s epochs of EEG) showed that there was a significant relationship between the two measurements, indicating that although the SFI underestimated the number of microarousals, it did reflect the degree of sleep disruption as measured by a more sensitive index. To our knowledge a direct comparison of arousal detection using ASDA criteria (16) and standard 30-s epoch criteria (12) has not been previously reported.

Our decision to use the SFI in the present study was based on a number of considerations. Practically, we were aware that

TABLE 3

CHARACTERISTICS OF THE SAMPLE (n = 1,021) ACCORDING TO SLEEP FRAGMENTATION INDEX QUARTILE

	Sleep Fragmentation Index* Quartiles			
	1 (1.1–3.6)	2 (3.6–4.8)	3 (4.8–6.5)	4 (6.5–26.0)
Males,† n (%)	111 (44)	130 (50)	163 (64)	186 (72)
Hypertensive‡ participants,† n (%)	65 (26)	78 (30)	71 (28)	101 (39)
Participants using antihypertensive medication,† n (%)	26 (10)	27 (10)	24 (9)	40 (16)
Age,§ yr, mean (SD)	43.5 (0.5)	45.5 (0.5)	44.9 (0.5)	47.2 (0.5)
Body mass index, kg/m^2, mean (SD)	28.8 (0.4)	29.3 (0.4)	29.7 (0.4)	29.9 (0.4)
Systolic blood pressure,§ mm Hg, mean (SD)	122.8 (0.9)	125.0 (0.9)	125.9 (0.9)	128.2 (0.9)
Diastolic blood pressure,§ mm Hg, mean (SD)	81.2 (0.6)	82.8 (0.6)	82.7 (0.6)	83.8 (0.6)
Apnea–hypopnea index, events/h,‖ median (range)	0.5 (0, 70.8)	1.0 (0, 66.9)	1.4 (0, 97.5)	2.4 (0, 87.8)

* Total number of awakenings/shifts to Stage 1 (from deeper NREM or REM sleep) divided by total sleep time in hours.
† Chi square $p < 0.05$.
‡ Hypertensive, defined as systolic blood pressure $\geqslant$ 140 or diastolic blood pressure $\geqslant$ 90 or antihypertensive medication use.
§ F-test $p < 0.05$ test.
‖ Kruskal–Wallis.

TABLE 4

MULTIPLE LINEAR REGRESSION MODEL* FOR THE SLEEP FRAGMENTATION INDEX AND BLOOD PRESSURE IN INDIVIDUALS WITH AHI < 1 (n = 483)

Independent Variable	Systolic Blood Pressure β-coeff ± SE	Diastolic Blood Pressure β-coeff ± SE
Sex, male	6.39 ± 1.20	3.97 ± 0.84
Age, 1 yr	0.25 ± 0.09	0.18 ± 0.07
Body mass index, 1 kg/m^2	0.71 ± 0.13	0.42 ± 0.14
Sleep fragmentation index, 1 SF/h	0.62 ± 0.26	0.28 ± 0.20

* Adjusted for the use of antihypertention medication.

TABLE 5

MULTIPLE LINEAR REGRESSION MODEL* FOR SFI AND BLOOD PRESSURE IN INDIVIDUALS WITH AHI ≥ 1 (n = 538)

Independent Variable	Systolic Blood Pressure β-coeff ± SE	Diastolic Blood Pressure β-coeff ± SE
Sex, male	4.60 ± 1.20	4.13 ± 0.96
Age, 1 yr	0.40 ± 0.08	0.07 ± 0.06
Body mass index, 1 kg/m^2	0.60 ± 0.13	0.31 ± 0.10
Sleep fragmentation index, 1 SF/h	−0.03 ± 0.22	−0.13 ± 0.16

* Adjusted for the use of antihypertention medication and the apnea–hypopnea index.

counting the number of arousals based on 2- or 3-s epochs, in records lasting approximately 8 h, from over a thousand people would have taken more than one person. Thus, had we attempted to use a more sensitive measure of arousal, it may have been less specific because the interscorer agreement for identifying arousals using 3-s epochs of EEG is relatively low (17). In addition, there is a lack of agreement in what constitutes a microarousal; definitions have been based on changes in EEG frequencies ranging from 1.5 s to > 10 s, and may or may not include EMG criteria (2, 15, 16, 18). There is no such lack of agreement when an arousal is defined using the established rule of greater than half an epoch (12). Finally, the SFI showed better construct validity as evident by the correlation's found with age, leg kick indices, apnea–hypopnea indices, and sleep staging. Taken together, these considerations led us to the use of the SFI, rather than a more subtle measure of arousal.

The mean SFI measured in our study was close to that reported by Mathur and coworkers in a similar but smaller sample of normal healthy individuals (19); we noted a similar age-related association with this index. In addition, we established good test–retest reproducibility of this index in our laboratory. Together these observations indicate that the SFI is a robust measure of sleep disturbance.

A further consideration in the present study was our use of "arbitrary" cut-off criteria to define (*1*) the presence or absence of an apnea–hypopnea (4% desaturation) and (*2*) the presence or absence of sleep-disordered breathing (AHI < 1 event/h). Our 4% desaturation definition is the conventional marker for a sleep-disordered breathing event, and it is likely to have resulted in the misclassification of some participants in whom mild sleep-disordered breathing/upper airway resistance events occurred that did not meet the desatuartion cut-off, but were terminated with an arousal. Although we found a strong correlation (r = 0.99) between the AHI obtained using a desaturation definition of 4 versus 3%, the participants with less severe breathing events are likely to have had airflow limitation and a one or two percentage fall in oxygen saturation, factors that have been shown to contribute to systemic hypertension (*see below*). The inclusion of such people in the group with AHI < 1 event/h may have confounded the relationship between arousal and awake blood pressure recorded in our study.

A previous study using data from the Wisconsin Sleep Cohort study has shown that in 40 participants there was no significant difference in the AHI calculated for two different study nights (9); there was moderate test–retest reproducibility. Importantly the AHI varied randomly across nights. This indicates that any misclassification of participants in the present study, either due to measurement error or physiological changes (e.g., differences in sleep position, which was not controlled) would not have introduced a systematic bias into our results and would if anything have weakened the statistical association we found.

We have shown that sleep fragmentation is associated with elevated systemic blood pressure in people with an AHI < 1 event/h. Our findings are consistent with another study in which sleep fragmentation, resulting from periodic limb movements, produced acute changes in blood pressure (20). In both these studies, arousal-triggered reflexes may have produced the acute increases in blood pressure that have an independent influence on daytime blood pressure. Such findings have implications for the elderly in whom sleep is more fragmented compared to younger subjects (21). In addition, our findings may have public health implications for people who live in noisy environments that would produce more sleep fragmentation.

Sleep Fragmentation, Blood Pressure, and Sleep-disordered Breathing

The determinants of increased blood pressure in patients with sleep disordered breathing are unclear. There are at least three possible factors which could cause such increases, namely arousal, hypoxemia and greater intrathoracic pressure. Previous studies have shown that arousals from sleep are associated with acute increases in systemic blood pressure (1–2, 22). However, the occurrence of hypoxemia in patients with sleep-disordered breathing is also an important consideration in any discussion relating to the likely mechanisms of hypertension. When arousal from sleep is associated with an apnea, increases in blood pressure may be secondary to a corresponding hypoxemia leading to peripheral vasoconstriction (3, 23). Interestingly, sleep-disordered breathing is still associated with acute increases in blood pressure when hypoxemia is prevented with supplemental oxygen (1, 24) suggesting that hypoxemia alone does not account for this phenomenon. Changes in intrathoracic pressure may influence blood pressure by affecting venous return. Thus it has been suggested that with sleep-related upper airway obstruction, a more negative intrathoracic pressure leads to an increased thoracic blood volume and a resultant rise in stroke volume (and blood pressure) when intrathoracic pressure returns to normal following arousal (25). More recent findings have shown that reductions in stroke volume and cardiac output, together with an increase in mean arterial pressure, occur at the termination of apnea; this casts doubt on the idea that the changes in intrathoracic pressure are the sole cause of the apnea-related acute changes in blood pressure (26). Taken together these studies support the idea that arousal is a likely trigger for the acute transient increases in blood pressure which occur in patients with sleep-disordered breathing. It would have been interesting to investigate the relationship between changes in daytime blood pressure and the peak intrathoracic pressure produced during apneas by comparing participants with obstructive versus central sleep

apnea; unfortunately in our middle-aged adults, there were too few purely central events to investigate this thoroughly.

In humans, the link between arousal-related transient increases in blood pressure and systemic daytime hypertension is speculative. In our study, it is unlikely that the higher levels of systolic blood pressure that we observed in the group with an AHI < 1 could be due to hypoxemia, as this group did not experience episodic oxygen desaturation $> 4\%$ characteristic of overt sleep-disordered breathing. However, in this group, it is possible that changes in intrathoracic pressure, which were not classified as a hypopnea (i.e., upper airway resistance syndrome), might have contributed to the arousal-related effect on blood pressure. Furthermore, we may also have studied a small number of people who had hypoxemia associated with other chronic respiratory diseases. In our population 8.9% of people reported some form of asthma and 0.48% reported emphysema or obstructive lung disorder, 0.16% of the participants with lung disease also reported asthma. In these people daytime blood pressure may have been influenced by the hypoxia, but this is unlikely to have affected our analysis of episodic desaturation.

Morgan and coworkers provided evidence for a more direct influence of arousal from sleep on sympathetic outflow (27). In a previous study, these researchers have also shown that their measure of sympathetic outflow remained elevated for a substantial period of time even after a hypoxic stimulus was removed (28). These data provide a possible link between repetitive, transient events and sustained elevated sympathetic activity, albeit secondary to hypoxia. In patients with sleep apnea, sympathetic nerve activity (in resting muscle) has been shown to be elevated compared with controls (29). Of interest here would be a measurement of sympathetic nerve activity in patients with chronically fragmented sleep of a nonrespiratory cause, e.g., periodic limb movements.

In the present study we found no independent effect of an increase in SFI on a measurement of awake blood pressure in people with an AHI > 1. In another study, the influence of spontaneously occurring EEG arousals (scored using 3-s epochs) on acute changes in blood pressure was investigated (30). It was found that the specific effect of spontaneous arousals on blood pressure decreased as the duration of an apnea increased. We speculate that in our population, the significance of the independent contribution of the arousal on transient (and hence chronic) changes in blood pressure decreased as AHI increased. Here there is an assumption that as AHI increases so to does the length of the apnea, which, in turn, will be linked with greater physiological insult (i.e., hypoxemia and greater negative intrathoracic pressure). We have not directly tested this assumption and in our population the median AHI was relatively low although the range was large (*see* Table 3). Nevertheless, our analysis does not support the idea that arousal is the sole contributor to sustained higher levels of awake blood pressure in people who have a relatively high frequency of sleep-disordered breathing events. A further thought is that sleep disruption may in part modulate blood pressure via an arousal-related mechanism but that statistically the blood pressure association is more strongly predicted by the AHI because of interactions between all of the physiological insults associated with a sleep-disordered breathing event.

Finally, a series of studies aimed at investigating the relative roles of arousal and hypoxia on the cardiovascular system have been carried out using chronically instrumented dogs and rats. In these studies, repeated nocturnal arousal induced by an auditory stimuli did *not* result in a sustained increase in daytime blood pressure (31, 32). Whereas in the same animals intermittent airway occlusion (31) and repeated hypoxic exposure (33) did result in significant sustained hypertension. Apart from the obvious methodological differences, it is difficult to account for the difference between the findings in animals and those of the present study. One possible explanation is that the frequency of the apneas induced in the animals was relatively severe compared with those in the present study.

Conclusions. It has previously been shown that sleep fragmentation is associated with acute increases in systemic blood pressure. Until now, the relationship between arousal and awake systemic blood pressure has not been explored. Using data from a large population-based study, we found that sleep fragmentation was associated with sustained higher levels in awake systolic blood pressure in people without overt sleep-disordered breathing (AHI < 1). However, in people with an AHI > 1, the independent effect of changes in sleep state on blood pressure was not evident. Our results could have implications for conditions where the level of sleep fragmentation is highly independent of the presence of sleep-disordered breathing, such as periodic limb movements, certain neurological diseases, and aging.

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