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BRIEF REPORT

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A preliminary study of factors influencing the occurrence of post-arousal hypersynchrony

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Abstract

Background Post-arousal hypersynchrony (PAH) is a continuous delta wave occurring after arousal. We hypothesized that PAH would decrease with age because PAH is affected by sleep pressure, which decreases with age.

Methods We evaluated polysomnography (PSG) during daytime napping to determine whether age affected the incidence of PAH. Twenty healthy participants (10 females, 45.0 ± 14.8 years [mean ± standard deviation], and age range, 22-67 years) were assessed using PSG during 90-min naps. PAH was present in two participants in their 20 s, one in their 40 s, and two in their 60 s. We first investigated whether the incidence of PAHs decreases with age using correlation analysis. Secondly, correlations between PAH and sleep index were analyzed to evaluate the factors influencing PAH occurrence. Thirdly, we evaluated whether sleep pressure decreases with age. %N3 and slow-wave activity (SWA) were used to measure sleep pressure.

Results PAH occurrence was unchanged with age. PAH corrected with total sleep time (PAH/TST) increased with %N3, but not with SWA. PAH/arousal, which is PAH corrected by the number of arousals, was also increased with %N3 and SWA. These results indicate that PAH occurrence may be related to sleep pressure. Contrary to expectation, %N3 showed no change with age, but SWA decreased with age.

Conclusions PAH occurrence may be affected by sleep pressure. Contrary to our hypothesis, PAH was seen in older adults, and its occurrence was unchanged with age. This may be associated with the relatively high sleep pressure observed in older adults.

Keywords Arousal, Polysomnography, Sleep, Delta wave

Background

Post-arousal hypersynchrony (PAH) is a normal variant of sleep electroencephalography (EEG) and is accompanied by continuous delta waves after arousal (Kellaway and Fox 1952). PAH occurrence changes in sleep stages and timing in all-night sleep (Suzuki et al. 2021). For example, PAH appears during non-rapid eye movement (NREM) sleep, increasing in stage N3 and the first half of sleep, as does the %N3 index of sleep pressure (Suzuki et al. 2021). Moreover, its frequency power values are frontal-dominant (Suzuki et al. 2021), as is slow-wave activity (SWA) (Mander et al. 2017). Therefore, sleep pressure may influence PAH occurrence (Suzuki et al. 2021).

Factors affecting PAH other than sleep pressure are still unknown. However, measuring PAH and identifying the influencing factors may contribute to understanding sleep–wake control mechanisms in healthy adults and people with sleep disorders. There are similarities between arousal disorders and PAH. First, a sleep-wake

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dissociated state has been involved in the pathology of arousal disorders (Idir et al. 2022). PAH is also considered a mixed state of wakefulness and sleep. Usually, sleep is shallow after arousal; however, PAH is a paradoxical arousal in which sleep is seemingly deepened, as shown by delta bursts (Suzuki et al. 2021). Second, a previous clinical study examining sleepwalking revealed that the delta bursts and abnormal behavioral episodes were increased by a sleep deprivation load, or high sleep pressure (Pilon et al. 2006). Furthermore, delta bursts in sleepwalking only appear during NREM sleep (Pilon et al. 2006). PAH similarly increased at times of high sleep pressure and occurred during NREM sleep (Suzuki et al. 2021). Evaluating factors influencing PAH may elucidate the sleep–wake regulation system in healthy adults and the pathophysiology of arousal disorders.

A potential factor influencing PAH is age because sleep pressure decreases with age (Mander et al. 2017; Boulos et al. 2019; Dijk et al. 2010; Alger et al. 2018). PAH was found in children (Kellaway and Fox 1952) and also at a high rate (93.3%) in young adults (22.8 ± 2.0 years [mean ± standard deviation, SD]) (Suzuki et al. 2021). However, it remains unknown whether PAH is present in the middle-aged and elderly population. Thus, we hypothesized that the incidence of PAH may decrease with age.

The effect of sex on PAH in healthy participants has not been studied (Suzuki et al. 2021). A previous study showed sex differences in age-related sleep changes (Boulos et al. 2019; Dorffner et al. 2015); however, a previous meta-analysis showed no sex differences during all-night sleep among all ages (Boulos et al. 2019). Since sleep variables do not differ significantly between males and females, we hypothesized that sex does not affect PAH occurrence.

This study examined the factors influencing PAH occurrence. A preliminary study was conducted on daytime nap data to determine whether age and sex affect the PAH ratio.

Methods

Participants

This study was conducted in conjunction with "The effect of a rocking sheet on napping." The Ethics Committee of University of Tsukuba approved this study, and written consent was obtained from all participants. Twenty healthy adults were recruited: four participants in their 20 s, 30 s, 40 s, 50 s, and 60 s, and two male and two female participants in each age group. To examine the effects of rocking stimuli in "The effect of a rocking sheet on napping", we excluded the effects of confounding factors on sleep by having the same number of participants in each age group and sex. Then, to examine the effect of age on PAH, we considered the experimental paradigm appropriate, with equal numbers of participants in each age group from their 20 s to their 60 s. As the opportunity to investigate each age group in such a balanced way was difficult in our previous experiments (Suzuki et al. 2021; Fan et al. 2023), we decided to investigate PAH as an opportunity to investigate physiological factors of age and sex, albeit in a preliminary study. The inclusion criteria were as follows: no difficulty with the Japanese language, no claustrophobia, no hearing problems, and no history of sleep disorders. The exclusion criteria were as follows: irregular lifestyle (a regular lifestyle was defined as a regular bedtime between 21:00 and 01:00, a wake-up time between 06:00 and 09:00, and a sleep time of 7–9 h), body mass index (BMI) < 18.5 or ≥ 25 kg/m^2, those who worked a night shift (after 22:00) or traveled to a country with a time difference of more than 3 h within the past three months, habitual drinkers or smokers, those with a caffeine intake of ≥ 300 mg/day, Morningness-Eveningness Questionnaire score of ≤ 30 or ≥ 70 (Ishihara et al. 1986), Pittsburgh Sleep Quality Index ≥ 5.5 (Doi et al. 2000), Motion Sickness Susceptibility Questionnaire score ≥ 19, those who were possibly pregnant and breastfeeding, those with a potentially emergent disease or a history of such disease, those who received treatment for psychiatric disorders, and those who currently have symptoms related to treatment. The menstrual cycle was not uniform or standardized. Participants wore a Fitbit Charge 3 (Fitbit Inc., San Francisco, CA) for three days before polysomnography (PSG) measurement to measure their sleep–wake lifestyle. Fitbit is a wristwatch sleep measurement device that combines an activity meter with a photoplethysmography sensor to determine sleep stages. Fitbit has been validated by simultaneous measurement with PSG (Menghini et al. 2021; Chinoy et al. 2021; Suzuki et al. 2021). As Fitbit is more accurate than actigraphy in determining sleep–wake timing (Chinoy et al. 2021), we used Fitbit to control the sleep–wake lifestyle of the participants. Participants abstained from alcohol and caffeine consumption from 23:00 on the day before the experiment until the day of the experiment.

PSG measurement

PSG was measured during napping in 20 participants (10 females, 45.0 ± 14.8 years [mean ± SD]) in the rocking experiment using Polymate (MP6100, Miyuki-Giken, Tokyo, Japan). The effects of rocking stimulation using rocking seats will be presented elsewhere. The naps lasted 90 min from 14:30–16:00. The PSG measurement was obtained four times per person for 80 naps. EEG (electrode placements: F3, F4, C3, C4, O1, O2, Fpz, Fz, Cz, Pz, Oz, M1, and M2), electrooculography, chin electromyography, and electrocardiography results were recorded (Berry et al. 2018). PSG was sampled at 500 Hz

and recorded at a high frequency of 160 Hz and low frequency of the direct current.

PSG analysis

PSG analysis was performed using Sleepware G3 (version 3.9.4; Respironics, Murrysville, PA). In a blinded manner, a registered polysomnographic technologist manually scored the sleep stage using standard criteria (Berry et al. 2018). PAH was defined as ≥ 5 s of continuous high-voltage (≥ 75 μV) delta waves (0.5–4 Hz) in the frontal lead (F4–M1) after arousal (Suzuki et al. 2021). The following sleep parameters were calculated: total sleep time (TST), sleep latency (SL), sleep efficiency, wake after sleep onset, percentage of TST at each stage, stage R latency (RL), NREM sleep duration, and number of arousals (Berry et al. 2018). The PAH number was corrected for TST (PAH/TST) to eliminate the effect of sleep duration. The PAH number was corrected for arousal by ejecting the effect of the arousal (PAH/arousal).

EEG power spectral analysis

To scrutinize the relationship between PAH and sleep pressure, we analyzed SWA (delta band power, 0.5–4 Hz) (Mander et al. 2017; Dijk et al. 2010; Dijk 2009; Tarokh et al. 2021). EEG data were sampled at 500 Hz and filtered at 0.3 Hz (low-frequency filter) and 35 Hz (high-frequency filter). We used MATLAB 2020b (MathWorks, Natick, MA) for the SWA analysis. The EEG was visually observed, body movements were tagged as artifacts, and EEG epochs containing artifacts were excluded from the analysis. The 4-s EEG recordings were fast-Fourier transformed for F3, F4, C3, C4, O1, O2, Fpz, Fz, Cz, Pz, and Oz, and all channels were re-referenced to the mean mastoid (M1 and M2) after eliminating eye movement using independent component analysis using the EEGLAB toolbox in MATLAB. Ten power spectra of 4-s epochs with a 1-s overlap were assigned to a 30-s epoch. The SWA of N2 and N3 was calculated for each 4-s epoch and averaged over ten epochs. For each electrode, the power value of the SWA during N2 and N3 was corrected for total power (0.5–30 Hz) and multiplied by 100 to obtain the percentage of the relative SWA power (%).

Statistical analysis

Sleep variables, PAH numbers, and corrected PAHs per person were calculated as the mean of four naps. The Shapiro–Wilk test was used for normality. The Pearson correlation coefficient, Spearman rank correlation coefficient, independent *t*-test, and Mann–Whitney U test were used to perform statistical analyses. For relative SWA power, cluster-based permutation tests were used to correct *p*-values and control for multiple comparisons. We calculated the observed power of the sample size using Gpower 3.1.9.7 (http://www.gpower.hhu.de/) (Table 1). Binary logistic regression analysis was used to identify factors influencing PAH occurrence using PSG data. First, exploratory univariate logistic regression analysis was performed for all potential confounders, age, sex, BMI, questionnaires, sleep indices, stage R occurrence, and relative SWA power in each electrode. Then, multiple logistic regression analysis of those variables with significant differences was performed. The significance level was set at $p < 0.05$.

Results

PSG recordings in which participants continuously fell asleep for more than 1 min before lights out were excluded; 66 PSGs from 19 participants were used for analysis. The PAH incidence was low, at 5 out of 19 (26.3%), with a median of 0 (minimum–maximum: 0.0–1.0) and a mean of 0.17 ± 0.34 (SD) per participant. Contrary to our hypothesis, PAH occurred in middle-aged and elderly adults (two in their 20 s, one in their 40 s, and two in their 60 s; Fig. 1A, B). Furthermore, there was no correlation between PAH/TST or PAH/arousal and age ($r_s = 0.11$ and $r_s = 0.02$, respectively, both $p > 0.05$; Fig. 1A, B).

Since we found no decline of PAH with age, we first examined whether PAH was related to sleep variables and then evaluated the effects of age on sleep variables. First, PAH/TST was significantly positively correlated with %N3, RL, and NREM sleep duration ($r_s = 0.53$, $r_s = 0.62$, and $r_s = 0.48$, respectively, all $p < 0.05$, Fig. 1C-E). Other sleep variables showed no correlation with PAH/TST (all $p > 0.05$). PAH/arousal was also significantly correlated with %N3 ($r_s = 0.57$, $p < 0.05$; Fig. 1F). We found no correlation between other sleep variables and PAH/arousal (all $p > 0.05$). Second, we evaluated whether age affected sleep variables and found that %N1, arousal, SL, and age were significantly correlated ($r_s = -0.64$, $r_s = 0.49$, $r = 0.73$, all $p < 0.05$; Fig. 1G-I). There was no correlation between age and %N3 (Fig. 1J) and other sleep parameters (all $p > 0.05$).

For relative SWA power analysis, Spearman's correlation coefficients of each electrode with PAH/TST, PAH/arousal, and age are summarized in the topographies (Fig. 1K-M). PAH/TST showed no significant correlation with relative SWA power (all $p > 0.05$, corrected by cluster-based permutation tests; Fig. 1K). PAH/arousal was significantly correlated with relative SWA power in F3, F4, C4, O1, O2, Fz, Cz, Pz, and Oz ($r_s = 0.47$, $r_s = 0.49$, $r_s = 0.50$, $r_s = 0.57$, $r_s = 0.62$, $r_s = 0.53$, $r_s = 0.54$, $r_s = 0.50$, $r_s = 0.64$, respectively, all $p < 0.05$, corrected by cluster-based permutation tests; Fig. 1L). Relative SWA power was significantly negatively correlated with age in F3, F4, C3, C4, Fpz, and Pz ($r_s = -0.50$, $r_s = -0.49$,

Table 1 The observed power of sample size of each sleep parameter

Parameter	Correlation with age (years)	Between sex	Correlation with PAH/TST (/min)	Correlation with PAH/arousal
PAH/TST (/min)	0.07	0.05	NA	NA
PAH/arousal	0.05	0.07	NA	NA
TST (min)	0.15	0.49	0.27	0.22
SE (%)	0.15	0.49	0.27	0.22
SL (min)	0.93	0.27	0.25	0.16
WASO (min)	0.05	0.30	0.33	0.30
%N1 (%)	0.64	0.16	0.10	0.15
%N2 (%)	0.25	0.11	0.17	0.17
%N3 (%)	0.08	0.11	0.74	0.81
%R (%)	0.10	0.06	0.10	0.10
RL (min)	0.35	0.46	0.69	0.59
NREM sleep duration (min)	0.45	0.51	0.61	0.52
Number of arousals	0.99	0.34	0.21	0.14
Relative SWA power[a]				
F3 (%)	0.65	0.30	0.47	0.58
F4 (%)	0.63	0.29	0.52	0.64
C3 (%)	0.83	0.15	0.39	0.52
C4 (%)	0.75	0.14	0.54	0.66
O1 (%)	0.54	0.09	0.70	0.81
O2 (%)	0.43	0.13	0.80	0.90
Fpz (%)	0.59	0.40	0.39	0.45
Fz (%)	0.52	0.31	0.63	0.73
Cz (%)	0.54	0.14	0.60	0.74
Pz (%)	0.73	0.10	0.52	0.66
Oz (%)	0.61	0.09	0.85	0.93

NA not applicable, *NREM* non-rapid eye movement, *PAH* post-arousal hypersynchrony, *RL* stage R latency, *SE* sleep efficiency, *SL* sleep latency, *SWA* slow-wave activity, *TST* total sleep time, *WASO* wake after sleep onset

[a] The relative SWA power were calculated using Spearman's correlation coefficient for all to evaluate topography and cluster-based permutation tests

$r_s = -0.58$, $r_s = -0.54$, $r_s = -0.47$, $r_s = -0.53$ respectively, all $p < 0.05$, corrected by cluster-based permutation tests; Fig. 1M).

There were no sex differences in PAH (PAH/TST: $Z = -0.42$, PAH/arousal: $Z = -0.63$, both $p > 0.05$). The sleep variables and SWA did not differ by sex (all $p > 0.05$).

Finally, we evaluated factors influencing PAH occurrence. Univariate logistic regression analysis revealed that participants with PAH were associated with %N3, with an odds ratio of 1.06 (95% confidence interval: 1.01–1.11, $p < 0.05$), and with relative SWA power in O2 (SWA_{O2}) with an odds ratio of 222.39 (95% confidence interval: 1.14–43350.13, $p < 0.05$). Multiple logistic regression analysis showed no significant associations between PAH occurrence, %N3, and SWA_{O2} (all $p > 0.05$). However, %N3 and SWA_{O2} were significantly correlated ($r_s = 0.58$, $p < 0.05$).

Discussion

The incidence of PAH in daytime naps was low at 26.3% (mean, 0.17 times) compared with data from all-night sleep in a previous study (Suzuki et al. 2021). Sleep pressure during naps was lower than during nocturnal sleep (Tarokh et al. 2021), which may have contributed to the lower PAH incidence.

We hypothesized that the incidence of PAH might decrease with age because PAH could be affected by sleep pressure (Suzuki et al. 2021), and sleep pressure might be reduced with aging (Mander et al. 2017; Boulos et al. 2019; Dijk et al. 2010; Alger et al. 2018). Firstly, PAH occurrence increased with %N3 (Fig. 1C, F), suggesting that PAH appears in individuals with higher sleep pressure. Secondly, PAH/arousal increased with SWA (Fig. 1L). The results of the present study were consistent with a previous study, which reported that PAH was related to sleep pressure (Suzuki et al. 2021).

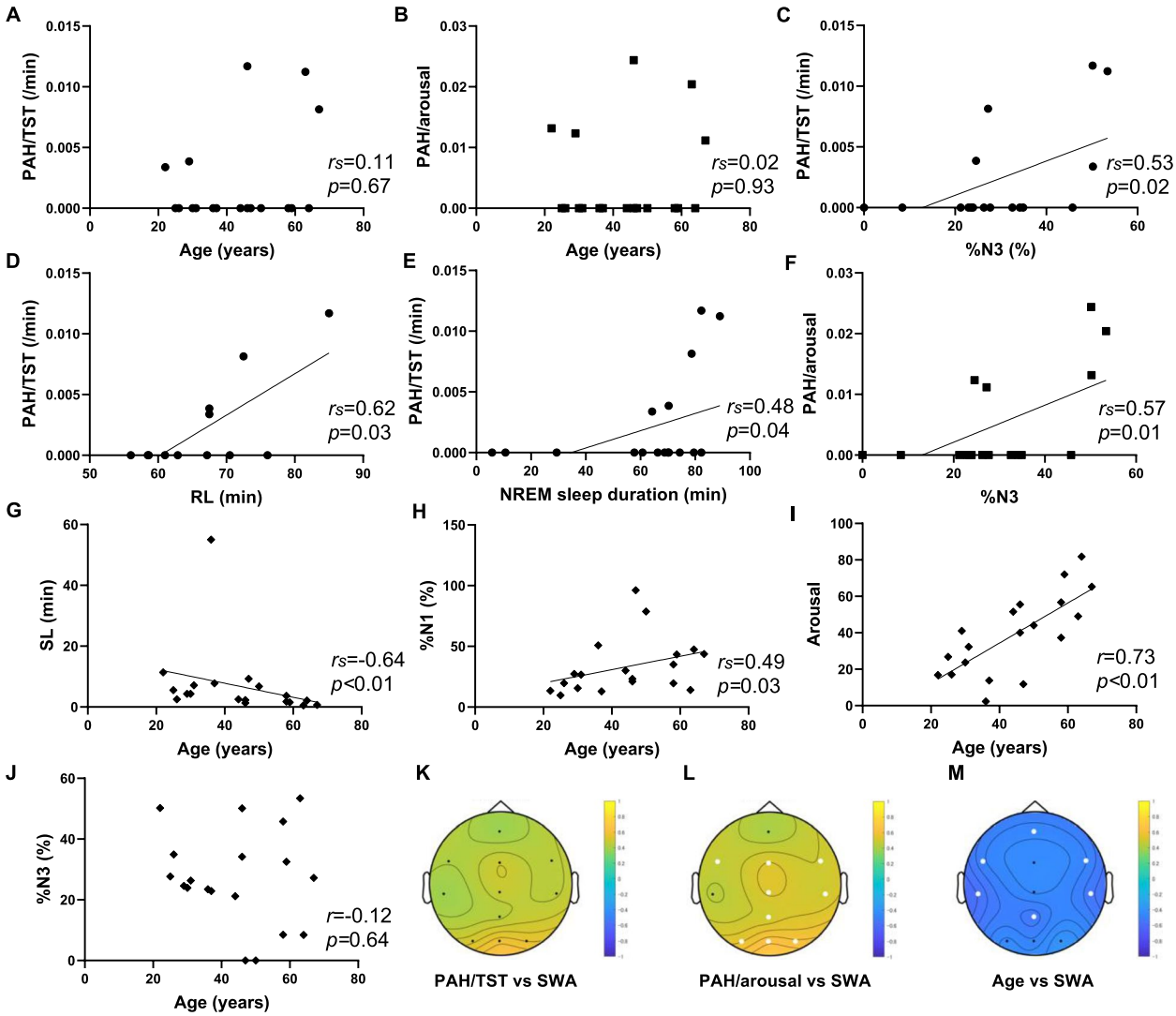

Fig. 1 Distribution of PAH, sleep index, slow-wave activity (SWA), and age. **A** PAH corrected for TST (PAH/TST) at all ages. **B** PAH corrected for arousal (PAH/arousal) at all ages. **C** PAH/TST and %N3. **D** PAH/TST and stage R latency (RL). **E** PAH/TST and non-rapid eye movement (NREM) sleep duration. **F** PAH/arousal and %N3. **G** Sleep latency (SL) at all ages. **H** %N1 at all ages. **I** Number of arousal number at all ages. **J** %N3 at all ages. **K** Topography of the correlation PAH/TST and between relative slow-wave activity (SWA) power. Color bar shows Spearman's rho. Black circle shows electrode location and non-significant correlation between PAH/TST and SWA. *P* values are corrected by cluster-based permutation tests. **L** Topography of the correlation between PAH/arousal and relative SWA power. Color bar shows Spearman's rho. Black circle shows non-significant correlation between PAH/arousal and SWA. White circle shows significant correlation between PAH/arousal and SWA. *P* values are corrected by cluster-based permutation tests. **M** Topography of the correlation between age and relative SWA power. Color bar shows Spearman's rho. Black circle shows non-significant correlation between age and SWA. White circle shows significant correlation between age and SWA. *P* values are corrected by cluster-based permutation tests. NREM: non-rapid eye movement, PAH: post-arousal hypersynchrony, RL: stage R latency, SL: sleep latency, TST: total sleep time, SWA: slow-wave activity

On the other hand, PAH/TST was not significantly correlated with SWA (Fig. 1K), possibly because PAH/arousal reflected sleep pressure better than PAH corrected for sleep duration in the previous study (Suzuki et al. 2021). Similarly, PAH showed positive correlations with RL and NREM sleep duration (Fig. 1D, E), suggesting that PAH is more likely to occur during stable, long NREM sleep. Thirdly, in previous studies, SWA and slow-wave sleep (N3) are often used as indices of sleep pressure (Mander et al. 2017; Dijk et al. 2010; Alger et al. 2018; Dijk 2009; Tarokh et al. 2021; Dinges 1986; Knowles et al. 1986), while other sleep indices also change in response to sleep pressure. Galli et al. applied a sleep restriction load to increase sleep

pressure and reported shortened SL, fewer awakenings after sleep onset, and increased %N3 (Galli et al. 2022). SL could have been affected by sleep pressure in the study. However, SL was not correlated with PAH occurrence. It may be because SL is related to the waking state before sleep onset.

Contrary to our hypothesis, the incidence of PAH was not affected by age (Fig. 1A, B). The reason may be that the middle-aged and elderly participants also showed high sleep pressure as young adults, as demonstrated by the non-significant correlation between age and %N3 (Fig. 1J). This result was inconsistent with previous studies, which reported that %N3 decreases with age (Dijk et al. 2010; Alger et al. 2018). However, meta-analyses of all-night sleep reported a trend but not a significant decrease in %N3 with age (Boulos et al. 2019). Additionally, a previous study reported that %N3 did not significantly decrease with age in females compared with males (Dorffner et al. 2015); half of the participants in the present study were females, which may have resulted in no changes in %N3 with age. Although previous studies reported that SL was prolonged or unchanged with age (Mander et al. 2017; Boulos et al. 2019; Dijk et al. 2010; Alger et al. 2018; Dorffner et al. 2015), SL shortened with age in this study (Fig. 1G). This may suggest that older participants were sleepier than younger participants. Similar to %N3, this result may also support the higher sleep pressure in older participants in the present study. Contrary to macro-sleep structure in the sleep index, SWA decreased with age (Fig. 1M). This may suggest sleep pressure decreases with age in micro-sleep structure. This result was similar to previous studies (Mander et al. 2017; Dijk et al. 2010). Moreover, in sleep indices unrelated to sleep pressure, such as %N1 and arousal, this study showed age-related increases (Fig. 1H, I) as in previous studies (Mander et al. 2017; Boulos et al. 2019; Alger et al. 2018; Dorffner et al. 2015). These may reflect sleep instability, and factors associated with aging other than sleep pressure may also need to be considered. It is necessary to scrutinize the relationship between PAH and age, increasing the number of participants in future studies.

Exploratory univariate logistic regression analysis revealed that %N3 and SWA_{O2} were shown to be factors attributable to PAH. This analysis also supports the association between PAH occurrence and sleep pressure. However, multiple logistic regression analysis showed no association between PAH and %N3 or SWA_{O2}. This may be because SWA in %N3 and SWA_{O2} were associated.

As hypothesized, there was no sex difference in PAH because there was no sex difference in the sleep indices. However, further scrutiny of age, sex, and other factors in all-night PSG is warranted.

This study had several limitations. First, the menstrual cycle was not standardized. Second, the sample size was small. In examining sex differences, the small observed power (all < 0.8) (Cohen 1992) was pointed out (Table 1). There is a need to increase the sample size in future studies, particularly that of female participants, because PAH can be influenced by the sex cycle and menopause. In a previous study, there were changes in aging sleep variables by sex; males showed a significant decrease in %N3 with age, while females showed no change (Dorffner et al. 2015). PAH could also be more frequent in females in their 60 s than in males in their 60 s and older in a large-scale study. Third, this study proposes that rocking stimuli may have affected PAH; hence, such stimuli should be investigated using napping and overnight sleep and compared to conditions without any stimulus in the future. As PAH was found at a higher rate in young adults in a previous study (Suzuki et al. 2021), the incidence of PAHs may be higher in nocturnal sleep, and the effects of age or sex may be more apparent. The rate of PAH could decrease with age in full-night PSG, as we hypothesized. For the first time, it has been shown that a certain percentage of PAHs also occur in older people, and we would like to see further investigations in the future with full-night PSG.

In conclusion, the occurrence of PAH may be related to sleep pressure. PAH was observed in older adults and its occurrence did not change with age. It may be associated with the relatively high sleep pressure observed in older adults.

Abbreviations

EEG	Electroencephalography
NREM	Non-rapid eye movement
PAH	Post-arousal hypersynchrony
PSG	Polysomnography
RL stage	R latency
SD	Standard deviation
SL	Sleep latency
TST	Total sleep time
SWA	Slow-wave activity

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Authors' contributions
All authors contributed to the study conception and design. Data collection and analysis were performed by YS and ZF. The first draft of the manuscript was written by YS and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

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Availability of data and materials
The datasets used and analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
The Ethics Committee of the Institute of Systems and Information Engineering, University of Tsukuba approved this study (Permission Number: 2021R495). All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. A verbal explanation was provided to all participants, and written consent was obtained.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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