Examination of the First-Night Effect during the Sleep-Onset Period

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Study Objectives: The present study examined the first-night effect during the sleep-onset period using the 9 electroencephalogram stage scoring system.

Design: After a week of monitoring sleep-wake habits with sleep diaries and wrist actigraphy, polysomnography recording was made for 3 consecutive nights.

Setting: Participants slept in their own private, individual, temperature-controlled bedroom in a sleep laboratory at the university.

Participants: Eleven healthy student volunteers (5 women and 6 men, 21 to 25 years old, mean 22.7 years) who had no experience sleeping in a laboratory participated in the study.

Interventions: N/A

Measurements and Results: The electroencephalogram during the sleep-onset period was scored manually for every 5-second epoch into 9 electroencephalogram stages. Latencies of the electroencephalogram stages were delayed on the first night, especially during the alpha-wave intermittent stages. The average time of the alpha-wave train, intermittent (> 50%) and the electroencephalogram flattening stage increased on Night 1. Stage changes among these stages also increased on Night 1. In contrast, stage changes between the alpha-wave intermittent stage (< 50%) and the theta-wave stage increased on Night 3.

Conclusions: Alpha-wave activity increased on Night 1, demonstrating that the activity of the wake-promoting system during the sleep-onset period was enhanced on the first night. From the second to the third night, the alpha-wave intermittent stage jumped to the theta-wave stage, omitting electroencephalogram flattening, suggesting that the electroencephalogram flattening stage is unlikely to appear during stable sleep-onset period. This is the first study to demonstrate the detail of the first-night effect during the sleep-onset period.

Key Words: First-night effect, laboratory adaptation process, sleep-onset period, EEG stages, alpha wave activity, EEG flattening, wake-promoting system, sleep-promoting system

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INTRODUCTION

SLEEP STRUCTURE IS DISTORTED IN A LABORATORY, PARTICULARLY DURING THE FIRST NIGHT OF SLEEP. THIS PHENOMENON IS RECOGNIZED AS THE "FIRST-NIGHT EFFECT" (FNE).[1] The FNE has long been a puzzling issue in sleep research. The following characteristics of the FNE have been reported: reduced total sleep and rapid eye movement (REM) sleep time, lowered sleep efficiency, increased intermittent wake time and amount of sleep stage 1, and longer latency of sleep stage 1 and REM sleep.[1-7] Especially, prolonged sleep latency (latency to non-REM [NREM] sleep stage 1 or 2) and difficulty in falling asleep are the most frequently reported characteristics of the FNE.[1,3,5,6]

Difficulty in falling asleep has been studied not only in normal volunteers, but also in patients suffering from generalized anxiety disorders, sleep apnea-hypopnea syndrome, chronic fatigue syndrome, depression, and posttraumatic stress disorder.[8-14] The fact that the sleep latencies of the first and the following nights for these patients are much more prolonged than those of healthy participants shows that the transition process from wakefulness to sleep is not smooth and is unstable in these patients. As such, those who have less of an ability to adapt to a new environment would be apt to have sleep problems, especially in the sleep-initiation process, when they enter various types of facilities, such as in-hospital admission. Sleep management for these people has widely been a critical issue.

Several factors have been proposed as the cause of the FNE: the change in sleeping environment, discomfort caused by electrodes or being kept under watched by experimenters, or reactions to a novel or stressful situation.[1,3-5,15] These factors would increase arousal and inhibit a sleep-initiation process, eventually resulting in the FNE. However, the details of when and how the FNE affects the wake-sleep transition period are not fully understood.

The transition period from wakefulness to sleep is called the sleep-onset period (SOP).[16] The reciprocal antagonistic interaction between the 2 systems, the wake-promoting system (wake system) and the sleep-promoting system (sleep system) accounts for the alteration of waking and sleep.[17] Recently, as part of the wake system, the tuberomammillary nucleus in the lateral hypothalamus has been studied, which contains orexin/hypocretin neurons that are crucial for wakefulness.[18] As part of the sleep system, the ventrolateral preoptic nucleus has been studied, which contains GABAergic and galaninergic neurons that are active during sleep.[18] Also, Steriade et al[19] have clearly demonstrated that the hyperpolarization of the thalamocortical neurons contribute to sleep deepening.

The onset of the sleep spindle is frequently treated as the sleep onset. However, quantitative analysis of the electroencephalogram (EEG) has demonstrated that the inactivation of the wake system and the increasing activity of the sleep system start before this point.[20-29] Occipital-dominant alpha-band activity (8.0-12.0 Hz) and beta- and gamma-band activities (20.0-45.0 Hz) are highest during wake and decrease in parallel with the attenuation of the alpha waves.[20-27] On the contrary, EEG activity of the theta

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band (2.0-7.0 Hz) increases gradually during the SOP,[21-28] especially from the period when the vertex sharp waves appear.[23,24,26,28] Also, the sigma band (11.5-13.5 Hz) activity, which reflects the activity of the sleep spindles, increases rapidly during the SOP,[21,23,25-27,29] especially from the period when the vertex sharp waves appear.[23,26,29] Therefore, the effects of the FNE on the wake and sleep systems could be clarified by examining the SOP in more detail.

Under the standard criteria of sleep stages,[30] the SOP has been subsumed under sleep stage 1. This has caused difficulties in examining the delicate changes in the SOP. Hori et al[31] classified the EEG patterns during the SOP into 9 EEG stages: H1 (hypnagogic 1), alpha-wave train; H2, alpha-wave intermittent A (> 50%); H3, alpha-wave intermittent B (< 50%); H4, EEG flattening; H5, ripples; H6, vertex sharp-wave solitary; H7, vertex sharp-wave train or burst; H8, vertex sharp-wave and incomplete spindle; H9, spindles.

Availability and validity of the EEG stages are recognized by examining the subjective, behavioral, and physiologic changes during the SOP.[23,26,28,31-36] Tanaka et al[26,28] have found that the basic EEG activities during the SOP are alpha, theta, and sleep-spindle activities; the posterior dominant alpha-band activities decrease drastically during the alpha wave-disappearing stage (H3 to 4); and the theta, delta, alpha-3, and sigma-band activities start to increase rapidly when the vertex sharp wave appears (H6). They have also used coherence analysis to summarize the characteristics of the SOP.[26,28] Alpha-2 coherence decreased smoothly from H3 during the SOP. On the contrary, delta, theta, and alpha-3 coherence rose rapidly from H6, which indicates that these slow-wave cortical-coherence increases are associated with the onset of vertex sharp wave.[26,28,37] These results suggest that the activities of the wake system decrease around H3 and those of the sleep system increase rapidly after H6.

In this study, we examined the FNE on the SOP using the 9 EEG stage scoring system.[31] By using this scoring system, we were able to examine the FNE during the SOP in detail. Furthermore, potential underlying neurophysiologic mechanisms behind the FNE on the SOP are proposed.

METHODS

Participants

Eleven healthy student volunteers (5 women and 6 men, 21 to 25 years old, mean age 22.7 ± 0.42 years) participated in the study. They had no experience in sleeping under experimental situations. Prior to the experiment, they answered a questionnaire as to their sleep-wake habits. Usual sleep and wake time, phase of the circadian activity cycle, regularity of the sleep habits, nap-taking habits, sleep complaints, and regularity of lifestyle (eg, the time of meals) were checked by the questionnaire. In addition, they completed a morningness and eveningness questionnaire[38] and questionnaires as to their physical and psychiatric health and sleeping conditions.

Participants had regular sleep-wake cycles, slept 6 to 9 hours daily, were not in the habit of taking a nap or consuming alcoholic beverages before sleep, and were all nonsmokers. Any volunteer who had physical or psychiatric diseases, was currently receiving medical treatment, or was suspected of having a sleep disorder was excluded. None of the participants had taken any prescription medications within the month prior to the experiment. After receiving an explanation of the purpose and the procedure of the present study, they gave written informed consent. They were informed that they were free to discontinue the experiment whenever they wished.

Procedure

During the week prior to the experiment (preparation week), participants were instructed to maintain their sleep-wake habits, ie, their daily wake-sleep time and sleep duration. During the preparation week, they were also instructed to refrain from excessive alcohol consumption, unusual physical exercise, and the taking of naps. Their sleep-wake habits were monitored by a sleep log and wrist activities (Mini-Motionlogger Actigraph, Ambulatory Monitoring Inc., Ardsley, NY) to make sure the procedure was successful during this week.

After the preparation week, polysomnogram (PSG) recordings were made for 3 consecutive nights. Participants slept in their own private, individual, temperature-controlled bedroom in a sleep laboratory at the university. Taking into account the individual variations of the biologic rhythm, their retiring and waking times were set to their habitual sleep time instead of fixing to a uniform time. They reported to the laboratory 3 hours before their retiring time. After the electrodes for PSG recording were attached, they answered questionnaires about their daytime activities, sleepiness, and sleep-wake time. They went to bed at their habitual retiring time. The experimenter woke them at their habitual waking time. They acted freely during daytime except for refraining from alcohol consumption, unusual physical exercise, and taking a nap.

PSG Recordings

Electroencephalogram (EEG) was recorded at 19 scalp sites (Fp1, Fp2, F3, F4, C3, C4, P3, P4, O1, O2, F7, F8, T3, T4, T5, T6, Fz, Cz, Pz) according to the 10-20-electrode system using a digital polygraph (EEG1100, Nihon Kohden corp., Tokyo, Japan), referred to the left ear lobe (A1). Electrooculogram was recorded from 2 electrodes placed at the outer canthi of both eyes. System reference (the mean amplitude between C3 and C4) was used for recording. The data were re-referenced off line to the linked earlobes. The submental electromyogram was recorded bipolarly. Electrocardiogram and respiratory movement were recorded at the same time. Electrode impedance was kept below 5kΩ. The sampling rate was 1000 Hz. Time constants for each recording were 0.3 seconds for EEG, 5.0 seconds for electrooculogram, and 0.03 seconds for electromyogram. The high-cut filter was set at 120 Hz.

Sleep Stage Scoring and Variables

According to Rechtschaffen and Kales' criteria,[30] EEGs were scored manually into sleep stages for every 30-second epoch from lights off to more than 2 minutes after the appearance of well-defined sleep spindles. EEG recordings at the C3 area were used for this scoring except for the waking stage; the O1 area was used for scoring the EEG alpha activities. If C3 or O1 recording was difficult to score due to artifact contamination, C4 or O2 recording was used instead. The scalp area used to score was unified within individuals during the 3 nights. The epochs contaminated by body movements for longer than half of the epoch were scored as movement time. According to the scored data, latencies to NREM sleep stage 1 and 2 from lights off were calculated.

EEG Stage Scoring and Variables

The above EEG recordings were rescored manually for every 5-second epoch according to the 9 EEG stage scoring system.[31] EEG recordings at the C3 area were used for this scoring, except for the EEG stages accompanied by alpha-wave activities (H1-3); the O1 area was used for scoring these stages. When any artifacts contaminated the tracings, C4 or O2 recording was used, as with the sleep stage scores.[30] The scalp area used to score was unified during the 3 nights, and the epochs contaminated by body movement were scored as movement time, as with the sleep-stage scoring by Rechtschaffen and Kales.[30] The definitions of the 9 EEG stages are listed below.[31,36]

H1. Alpha wave, train: epoch composed of a train of alpha activity with a minimum amplitude of 20 μV.
H2. Alpha wave, intermittent A: epoch composed of a train of at least 50% alpha activity with a minimum amplitude of 20 μV.
H3. Alpha wave, intermittent B: epoch composed of a train of less than 50% alpha activity with a minimum amplitude of 20 μV.
H4. EEG flattening: epoch composed of suppressed waves of < 20 μV.
H5. Ripples: epoch composed of low-voltage theta waves (20 μV < θ < 50 μV) with burst suppression.
H6. Vertex sharp wave, solitary: epoch contained 1 well-defined vertex sharp wave.
H7. Vertex sharp wave, train or burst: epoch contained at least 2 well-defined vertex sharp waves.
H8. Vertex sharp wave and incomplete spindle: epoch contained at least 1 well-defined vertex sharp wave and 1 incomplete spindle (duration < 0.5 seconds, amplitude < 20 μV and > 10 μV).
H9. Spindles: epoch contained at least 1 well-defined spindle of at least 0.5 seconds duration and 20 μV in amplitude.

By the use of this EEG-stage scoring system, we examined the following 3 issues: which EEG stage contributed to the delays in the sleep latencies reported in the previous studies about the FNE, the FNE on the durations of the EEG stages, and the FNE on the stability of the EEG stages. To examine the first issue, the latencies to the EEG stages from lights off were calculated. Latencies to the EEG stages from the previous stages were also calculated. In case the previous stage did not appear, the latency from the stage that appeared before the previous stage was calculated. To examine the second issue, the average time that each stage appeared and the distribution of the durations of each epoch were calculated. Durations were classified into 3 classes: 5-15 seconds (1-3 epochs), 20-30 seconds (4-6 epochs), and 35 seconds or more (more than 6 epochs). The frequency of each of the durations was calculated. To examine the last issue, the number of stage changes from each stage to the other stages was calculated. Based on Tanaka et al,[36] 10 x 10 (9 EEG stages and movement time) contingency tables for 3 nights were constructed. Mean frequencies were rounded off to 1 decimal place. Numbers of 0.5 or more were described.

Statistical Analysis

One-way repeated-measures analysis of variance (ANOVA) with factors of nights (Night 1-3) were performed on NREM sleep stage 1 and 2 latencies, EEG stage latencies, latencies from the previous EEG stages, average time, durations, and number of stage changes. To control the type 1 error, Huynh-Feldt ε correction was applied. Posthoc tests were performed by multiple comparisons using Shaffer modified sequentially rejective multiple test procedure.[39]

RESULTS

Sleep Latencies

Table 1 shows the latencies from lights off to NREM sleep stage 1 and 2 by the criterion of Rechtschaffen and Kales.[30] The ANOVA showed significant differences among the nights for both stages. Latencies of these sleep stages were delayed on the first night and shortened on the second and the third nights.

EEG Stage Latencies

Scoring of the EEG stages revealed that the participants did not always show all of the EEG stages. The following stages did not appear in 1 of the participants: H3 on Night 2, H4 on Night 2 and 3, H7 on Night 1 and 2, and H8 on Night 2 and 3. Figure 1 shows the latencies from lights off to 9 EEG stages. Latencies of almost all of the EEG stages were shortened from Night 1 to Nights 2

Table 1—Latencies of NREM sleep stage 1 and 2 (min).

Sleep †	Nights						ANOVA		
stages	1		2		3		*F* (2,20)	ε	*p*
1	6.3	(1.29) [a]	3.0	(0.53) [b]	3.0	(0.55) [b]	5.53	0.61	0.031
2	14.0	(2.40) [a]	7.3	(0.99) [b]	7.6	(0.77) [b]	9.83	0.54	0.009

Values in parentheses are standard errors.
[a,b] Means with different letters are significantly different (p <.05).
† Rechtschaffen and Kales' criterion.

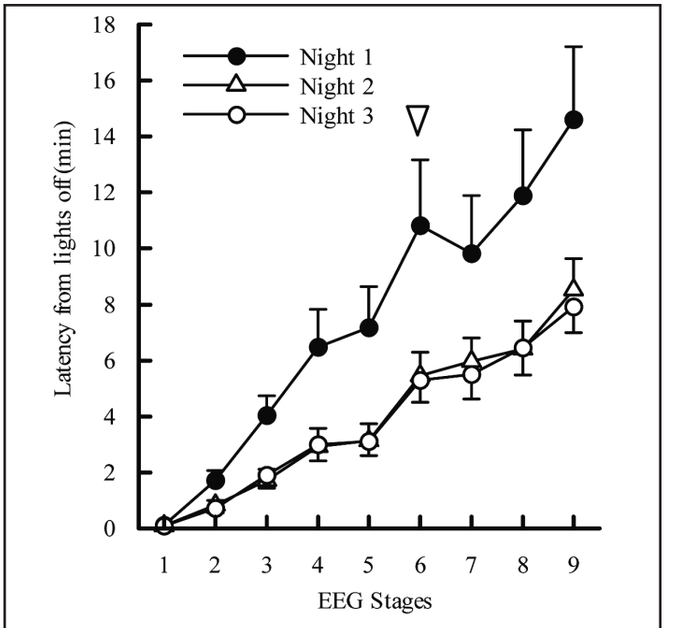

Figure 1—Latencies to the 9 electroencephalogram (EEG) stages of the sleep-onset period from lights off (N = 11). All the EEG stages except for stage 1 were delayed on Night 1 compared to the second and the third nights (P < .05). In addition, on the first night, the sequence of the latencies of EEG stage 6 and 7 were in inverse order; EEG stage 7 appeared before stage 6 appeared (indicated by an inverted triangle). Error bars show standard errors.

and 3. In addition, H7 appeared prior to H6 on the first night. This phenomenon was not seen on the subsequent nights. Latency to H9 was delayed for approximately 6 to 7 minutes on the first night compared to the subsequent nights (14.6, 8.5, and 7.9 minutes, at Night 1 to 3, respectively).

The ANOVA showed significant differences among the nights for all stages except for H1 [H2: $F_{2, 20} = 6.26$, $\varepsilon = .72$, $P = .017$; H3: $F_{2, 18} = 10.59$, $\varepsilon = 1.0$, $P = .001$; H4: $F(2, 16) = 5.89$, $\varepsilon = .67$, $P = .027$; H5: $F_{2, 20} = 9.83$, $\varepsilon = .64$, $P = .006$; H6: $F_{2, 20} = 6.06$, $\varepsilon = .53$, $P = .031$; H7: $F_{2, 16} = 5.11$, $\varepsilon = .58$, $P = .046$; H8: $F_{2, 16} = 6.15$, $\varepsilon = .58$, $P = .031$; H9: $F_{2, 20} = 8.43$, $\varepsilon = .65$, $P = .009$]. The posthoc comparisons showed that the latencies of EEG stages from H2 to 9 were delayed on Night 1 compared to Nights 2 and 3 (P values < .05). No significant difference was found between Nights 2 and 3.

Latencies from the Previous EEG Stages

Table 2 shows the latencies of EEG stages from the previous stages. On the whole, stage latencies were delayed on the first night compared to subsequent nights. The ANOVA showed significant differences for H2 and a marginally significant difference for H3. The posthoc comparisons showed that the latency of H2 was significantly delayed on Night 1 compared to Nights 2 and 3. In addition, H3 was significantly delayed on Night 1 compared to Night 2.

Average Time

Table 3 shows the average time that each stage appeared. Although H1, 2, and 5 appeared over 1 minute for 3 nights, the other stages (H3, 4, 6-9) were less than 1 minute. The ANOVA showed significant differences among the nights for H1, 2, and 4. The posthoc comparisons showed that H1 and 2 were longer for Night 1 compared to Nights 2 and 3. H4 was longer on Night 1 compared to Night 3.

Distribution of Stage Durations

Table 4 shows the distribution of the durations of H1, 2, and 4, in which the significant differences of the average time among the nights were observed. Although there were 20 to 30 seconds or at least 35 seconds in duration for H1 and 2, most of the durations were less than 15 seconds for H4.

For H1, the ANOVA showed significant differences among the nights for the durations of 5 to 15 seconds and at least 35 seconds. The duration of 20 to 30 seconds was marginally significant. The posthoc comparisons showed that frequencies in the durations of 5 to 15 seconds and at least 35 seconds significantly decreased from Nights 1 to 2 and from Nights 1 to 3. The frequency in the duration of 20 to 30 seconds almost significantly

Table 2—Latencies of EEG stages from the previous stages (min).

EEG † stages	Nights 1		Nights 2		Nights 3		ANOVA $F(2,20)$	ANOVA ε	ANOVA p
H 2	1.6	(0.36) [a]	0.7	(0.17) [b]	0.6	(0.16) [b]	5.86	0.71	0.010
H 3	2.3	(0.58) [a]	0.9	(0.26) [b]	1.2	(0.43)	3.38	0.87	0.054
H 4	2.4	(0.85)	1.0	(0.39)	1.0	(0.27)	3.03	0.60	n.s.
H 5	0.7	(0.31)	0.4	(0.16)	0.2	(0.36)	0.90	1.00	n.s.
H 6	3.7	(1.23)	2.3	(0.39)	2.2	(0.48)	1.23	0.54	n.s.
H 7	0.3	(0.36)	0.3	(0.24)	0.2	(0.38)	0.01	0.83	n.s.
H 8	0.8	(0.39)	0.6	(0.20)	0.7	(0.28)	0.08	0.71	n.s.
H 9	2.7	(0.97)	2.1	(0.58)	1.7	(0.33)	0.56	0.69	n.s.

Values in parentheses are standard errors.
[a,b] Means with different letters are significantly different (p <.05).
† Hori' s 9 stages criterion.

Table 3—Average time of EEG stages during the SOP (min).

EEG † stages	Nights 1		Nights 2		Nights 3		ANOVA $F(2,20)$	ANOVA ε	ANOVA p
H 1	3.9	(0.81) [a]	1.4	(0.34) [b]	1.3	(0.34) [b]	12.33	0.59	0.003
H 2	2.5	(0.68) [a]	1.0	(0.21) [b]	1.1	(0.23) [b]	5.69	0.60	0.011
H 3	1.2	(0.47)	0.6	(0.16)	0.8	(0.15)	2.43	0.52	n.s.
H 4	0.5	(0.11) [a]	0.3	(0.06)	0.3	(0.04) [b]	4.52	0.76	0.024
H 5	3.9	(0.56)	3.4	(0.41)	2.9	(0.41)	2.91	0.92	n.s.
H 6	0.3	(0.06)	0.3	(0.08)	0.4	(0.07)	0.13	0.99	n.s.
H 7	0.4	(0.06)	0.4	(0.10)	0.4	(0.11)	0.21	1.00	n.s.
H 8	0.6	(0.15)	0.6	(0.17)	0.4	(0.10)	0.72	1.00	n.s.
H 9	0.2	(0.11)	0.1	(0.01)	0.1	(0.00)	1.00	0.51	n.s.
MT	1.1	(0.42)	0.3	(0.11)	0.3	(0.07)	3.58	0.55	n.s.
Total	14.6	(3.43) [a]	8.5	(1.64) [b]	7.9	(1.53) [b]	8.43	0.65	0.002

Values in parentheses are standard errors.
[a,b] Means with different letters are significantly different (p <.05). † Hori' s 9 stages criterion.

decreased from Nights 1 to 2. For H2, the ANOVA showed significant differences among the nights for the durations of 5 to 15 seconds and 20 to 30 seconds. The posthoc comparisons showed significant differences between Nights 1 and 2 and between Nights 1 and 3. For H4, an almost significant difference was shown for the duration of 5 to 15 seconds. The posthoc comparisons showed a significant difference between Nights 1 and 3.

Number of Stage Changes

Table 5 shows the number of stage changes from each EEG stage to the other stages. Among the 3 nights, the total number of stage changes was the greatest on Night 1. The ANOVA revealed significant differences among the nights. The posthoc comparisons showed that the number of stage changes of Night 1 was significantly larger than that of Nights 2 and 3.

The larger number of total stage changes of Night 1 was mainly due to those of H1, 2, and 4. The ANOVA showed significant differences among the nights at H1, 2, and 4. The posthoc comparisons showed that the number of stage changes was significantly larger on Night 1 compared to the subsequent nights for H1 and 2 and on Night 1 compared to Night 3 for H4.

Distribution of Stage changes

Table 6 shows the contingency table of 3 nights. Throughout the nights, EEG stages from H1 to H4 mainly changed to the adjacent stages. For example, the number of the stage changes from H2 to H3 was 5.4 on Night 1, 2.5 on Night 2, and 3.7 on Night 3. The number of the stage changes from H2 to H4 was 0.5 on Nights 1 and 2, and less than 0.5 on Night 3, which was less than that from H2 to H3. In contrast, H5 to H9 often jumped the adjacent stages, eg, from H5 to H7 or H8, from H6 to H8, or turning back from H7 or H8 to H5. Preceding and subsequent stages of H5 were widespread, ranging from H3 to H9.

ANOVA revealed significant differences at the segments from H1 to H2 [$F_{2, 20} = 4.88$, $\varepsilon = .79$, $P = .029$] and H2 to H1 [$F_{2, 20} = 5.47$, $\varepsilon = .75$, $P = .023$]. These stage changes were significantly larger on Night 1 than on Nights 2 and 3 (P values $<.05$). Significant differences were also found at the segments from H3 to H5 [$F_{2, 20} = 6.42$, $\varepsilon = .89$, $P = .010$], and H5 to H3 [$F_{2, 20} = 4.51$, $\varepsilon = .75$, $P = .038$]. These changes significantly increased on Night 3 (3.2 times from H3 to H5 and 1.8 times from H5 to H3) compared to Night 2 (1.1 times from H3 to H5 and 0.5 times from H5 to H3). There was an almost significant difference at the segments from H4 to H5 [$F_{2, 20} = 3.96$, $\varepsilon = .62$, $P = .063$]. The number of changes significantly decreased on Night 3 (1.2 times) compared to Night 1 (3.1 times) ($P < .05$).

Table 4—Frequency distributions of durations in EEG stages 1, 2 and 4 (min).

EEG† stages	Duration (s)	Nights 1		2		3		ANOVA F (2,20)	ε	p
H 1	5-15	9.5	(2.47) [a]	3.6	(0.96) [b]	3.5	(1.18) [b]	5.40	0.73	0.025
	20-30	2.1	(0.58) [a]	0.7	(0.30) [b]	1.0	(0.38)	3.45	1.00	0.052
	≥ 35	1.9	(0.49) [a]	0.8	(0.23) [b]	0.5	(0.21) [b]	6.50	0.67	0.017
H 2	5-15	14.5	(3.91) [a]	6.1	(1.10) [b]	6.5	(1.72) [b]	5.42	0.66	0.029
	20-30	1.6	(0.51) [a]	0.6	(0.31) [b]	0.5	(0.25) [b]	5.52	1.00	0.012
	≥ 35	0.3	(0.19)	0.0	(0.00)	0.2	(0.12)	1.19	0.87	n.s.
H 4	5-15	4.6	(1.15) [a]	3.1	(0.51)	2.2	(0.40) [b]	3.87	0.67	0.060
	≥ 20	0.1	(0.09)	0.0	(0.00)	0.0	(0.00)	-	-	-

Values in parentheses are standard errors.
[a,b] Means with different letters are significantly different ($p < .05$).
† Hori's 9 stages criterion.

Table 5—Number of stage changes from each EEG stage to the other stages.

EEG † stages	Nights 1		2		3		ANOVA F (2,20)	ε	p
H 1	13.5	(3.15) [a]	5.2	(1.17) [b]	5.0	(1.36) [b]	7.15	0.67	0.005
H 2	16.5	(4.37) [a]	6.7	(1.24) [b]	7.3	(1.75) [b]	5.89	0.62	0.010
H 3	9.4	(3.37)	4.4	(0.83)	6.8	(1.53)	2.53	0.60	n.s.
H 4	4.7	(1.12) [a]	3.1	(0.51)	2.2	(0.40) [b]	4.25	0.68	0.029
H 5	13.4	(2.24)	10.6	(1.59)	10.9	(1.37)	0.97	0.87	n.s.
H 6	3.9	(0.77)	3.5	(0.78)	4.2	(0.66)	0.27	0.99	n.s.
H 7	3.5	(0.65)	3.7	(0.93)	3.0	(0.67)	0.35	0.85	n.s.
H 8	4.6	(1.27)	4.5	(1.02)	3.5	(0.81)	0.34	1.00	n.s.
H 9	1.2	(1.09)	0.2	(0.12)	0.0	(0.00)	1.00	0.51	n.s.
MT	6.2	(2.41)	2.5	(0.84)	2.5	(0.59)	2.88	0.60	n.s.
Total	76.9	(20.44) [a]	44.5	(9.04) [b]	45.3	(9.16) [b]	5.81	0.69	0.010

Values in parentheses are standard errors.
[a,b] Means with different letters are significantly different ($p < .05$).
† Hori's 9 stages criterion.

DISCUSSION

In accordance with the previous studies,[1,3,5,6] latencies to NREM sleep stage 1 and 2 were delayed on Night 1 compared to the subsequent nights. In addition, no significant difference was found between Nights 2 and 3. When rescored by the sleep-onset 9 EEG stages, it was found that not all of the EEG stages changed in the same way. H2 and H3, in which alpha waves appeared, were delayed significantly on the first night. The average time of H1 (alpha-wave train), H2 (alpha-wave intermittent) and H4 (EEG flattening) increased on Night 1. In addition, the number of stage changes between these stages was greater on Night 1.

The greatest changes of the EEG stages among the nights were observed in H1 to H3 in which alpha waves appeared. On the first night, latencies from H1 to H2 and H2 to H3 were prolonged. On that night, the average time of the alpha-dominating stages (H1 and H2) increased and the number of stage changes increased. Latencies to NREM sleep stage 1 or 2 are known to be delayed on the first night.[1,3,5,6] However, it was not clarified as to how the FNE affects the process of the sleep-onset state. The present study demonstrated that the prolonged latencies of sleep stages 1 and 2 by the FNE would contribute to the increased activity of the wake system. Alpha activity was slow to attenuate on the first night and it reappeared repeatedly even after it had disappeared. In addition to the difficulty in the attenuation of alpha activity, frequent emergence of alpha activity seems to be the main characteristic of the SOP in an unfamiliar situation.

Tanaka et al[26,28] quantitatively studied EEG changes during the SOP. They found that posterior—dominant alpha-band activities decreased drastically during the alpha wave-disappearing stages (H3 to 4). The posterior-dominant alpha-band activities are treat-

Table 6—Distribution of the stage changes of Nights.

Precedent EEG stages (*This stage...*) †

Subsequent EEG stages (...is followed by this stage)

	MT	H 1	H 2	H 3	H 4	H 5	H 6	H 7	H 8	H 9
MT		1.9 (0.56) 0.5 (0.31) 0.5 (0.31)	1.5 (0.84) 0.6 (0.28) - -	1.2 (0.99) - - 0.5 (0.31)	- - - - - -	1.1 (0.37) 1.0 (0.45) 1.0 (0.36)	- - - - - -	- - - - - -	- - - - - -	- - - - - -
H 1	3.0 (0.98) 0.9 (0.41) 0.6 (0.36)		8.5 (2.10) 3.0 (0.80) 3.0 (1.10)	1.0 (0.30) - - - -	- - - - - -	- - - - - -	- - - - - -	- - - - - -	- - - - - -	- - - - - -
H 2	1.2 (0.60) 0.5 (0.25) - -	10.5 (2.60) 4.4 (1.10) 4.3 (4.30)		4.1 (2.02) 1.5 (0.51) 2.0 (0.74)	- - - - - -	0.5 (0.21) - - 0.5 (0.21)	- - - - - -	- - - - - -	- - - - - -	- - - - - -
H 3	0.6 (0.54) - - 0.5 (0.25)	1.2 (0.52) - - - -	5.4 (1.96) 2.5 (0.45) 3.7 (0.94)		1.2 (0.48) 0.8 (0.30) 0.6 (0.20)	1.0 (0.38) 0.5 (0.21) 1.8 (0.42)	- - - - - -	- - - - - -	- - - - - -	- - - - - -
H 4	- - - - - -	- - - - - -	0.5 (0.21) 0.5 (0.16) - -	1.7 (0.45) 1.2 (0.33) 0.9 (0.28)		1.8 (0.40) 1.4 (0.41) 1.0 (0.27)	- - - - - -	- - - - - -	- - - - - -	- - - - - -
H 5	1.1 (0.59) 0.8 (0.33) 0.9 (0.37)	- - - - - -	0.6 (0.20) - - - -	1.4 (0.41) 1.1 (0.31) 3.2 (0.60)	3.1 (0.70) 1.9 (0.40) 1.2 (0.40)		2.4 (0.41) 2.3 (0.69) 2.7 (0.71)	1.8 (0.40) 1.8 (0.52) 1.0 (0.23)	2.5 (0.68) 2.5 (0.59) 1.5 (0.41)	0.5 (0.5) - - - -
H 6	- - - - - -	- - - - - -	- - - - - -	- - - - - -	- - - - - -	2.9 (0.65) 2.3 (0.56) 2.3 (0.60)		0.5 (0.21) 0.5 (0.21) 1.1 (0.34)	0.5 (0.21) 0.5 (0.21) 0.7 (0.38)	- - - - - -
H 7	- - - - - -	- - - - - -	- - - - - -	- - - - - -	- - - - - -	2.2 (0.48) 2.1 (0.67) 1.5 (0.31)	0.7 (0.27) 0.5 (0.21) 0.9 (0.34)		0.5 (0.16) 1.2 (0.40) 0.5 (0.37)	- - - - - -
H 8	- - - - - -	- - - - - -	- - - - - -	- - - - - -	- - - - - -	2.9 (0.90) 2.6 (0.80) 2.3 (0.54)	0.5 (0.37) 0.7 (0.24) - -	0.7 (0.19) 1.1 (0.34) 0.9 (0.46)		- - - - - -
H 9	- - - - - -	- - - - - -	- - - - - -	- - - - - -	- - - - - -	0.9 (0.37) 0.6 (0.15) - -	- - - - - -	- - - - - -	0.9 (0.62) - - 0.5 (0.16)	

† Hori' s 9 stages criterion.
Three figures lined up vertically in the each cell are the mean frequencies of Nights 1, 2, and 3 from the top, respectively.
Values in parentheses are standard errors.
Mean frequencies were truncated to one decimal place, and numbers below 0.5 were rounded down.
Segments with a box-shaped frame are stages in which ANOVAs showed significant differences among the nights ($p < .05$).

ed as the marker of wakefulness, as in the studies of microarousals (eg, Atlas Task Force of the American Sleep Disorders Association[40]; Smith and Trinder[41]). These studies show that the activities of the wake system decrease in parallel with the alpha-wave attenuation. The wake and sleep system are in an antagonistically opposed relationship.[17,18] Generally, during the SOP, the wake system makes a concession to the sleep system, though a change in sleeping environment, discomfort caused by electrode attachment, or being kept under watched by experimenters[1,3-5,15] would activate the wake system—as has been shown by the enhanced alpha waves in the results. The present study demonstrates that the FNE on the SOP is the result of the hyperactivation of the wake system and the difficulty in the exchange of the superiority of the wake and sleep system.

The FNE was also observed in H4 (flattening EEG stage). Although the duration of H4 was short throughout the nights (Table 4), the average time was shortened from the first to the third night (Table 3). The number of stage changes from H4 to the other stages decreased (Table 5), especially to H5 (ripples) (Table 6). In contrast, stage changes from H3 (alpha-wave intermittent) to H5 (ripples) increased from Night 1 to 3 (Table 6). It has been pointed out that the flattening EEG stage (H4) is a transitional and unstable stage.[36] The present results demonstrated that this stage was not only unstable by nature, but also diminished through the process of laboratory adaptation. After the attenuating of the alpha waves, the flattening EEG would jump to the ripples stage (H5).

Quantitative analysis of the EEG in the previous studies demonstrated that the wake system diminished prominently in accordance with the disappearance of alpha waves, whereas the sleep system was remarkably enhanced after theta waves or vertex sharp waves appeared.[23,24,26,28,29] The predominance of these systems would be altered at these sleep-onset stages. From this point of view, the predominance of these would not be smoothly altered in unfamiliar situations, and, thus, theta waves would not readily appear, even after the alpha waves had vanished. As for the results, the average time of the flattening EEG stage would act as an effective marker for the responses of environmental adaptation or psychological stress.

The sequences of the stage changes became complex from H5, in which theta waves appeared, as in the previous study.[36] In addition, H7 appeared prior to H6 on the first night in the present study. This phenomenon has not been previously reported. This demonstrates that the FNE affected not only the first half, but also the last half, of the SOP. A possible explanation for this phenomenon is that the sleep system, which has been inhibited by the activation of the wake system, is discharged at this point. There are several reports that suggest that the sleep system is rapidly activated at the time of the occurrence of the vertex sharp wave.[23,24,26,28,29] The activation level of the sleep system was not sufficient when theta waves appeared. When vertex sharp waves appear, the activation level of the sleep system increases, and then the wake-system can be suppressed. The FNE may remain even after the vertex sharp waves have occurred. To examine the possibility, further quantitative analysis of the EEG activity during the SOP would be required.

There have been several discussions about the duration of the FNE.[4-7] In the present study, with regard to stage latencies, diminution of alpha waves was more smooth in Night 1 than in Night 2 and there was no difference between Night 2 and 3. On the other hand, the FNE on the average time and stage changes of H3 (alpha-wave intermittent), H4 (flattening EEG), and H 5 (ripples) remained on Night 2 and even on Night 3. The participants were in good health, physically and mentally. These results indicate that the SOP stabilized over the 3 nights even for those who adapted easily to the novel environment. In addition, it is suggested that there are 2 processes of adaptation to the novel environment during the SOP. First, a smooth diminishing of alpha activities occurred on Night 2. Second, a smooth development of theta activities occurred on Nights 2 and 3, and then H5 frequently appeared skipping over the H4. So, care should be taken, especially when examining the qualitative aspects of the SOP by allowing 2 nights for adaptation.

It has been reported that the impact or the duration of the FNE differs by age.[5,6,42] Also, individual differences in the FNE have been studied in the field, such as sleep apnea-hypopnea syndrome,[8,11] chronic fatigue syndrome,[9] generalized anxiety disorder,[10] posttraumatic stress disorder,[14] depression,[12,13] or insomnia.[43-45] In these patients, sleep latencies of the first and subsequent nights were prolonged compared to healthy subjects. However, the FNE on the SOP was not examined in detail. Parameters used in the present study will be an effective tool in these clinical situations. When, how much, and how long the SOP is distorted by the FNE in those who have less of an ability to adapt to a new environment can be assessed by using the results of this study as a control. On the contrary, it could be even possible to assess the level or ability of the adaptation by the degree or the type of the distortion in the near future. This study presents significant findings as to sleep management.

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