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Time Course of Reaction Time and EEG While Performing a Vigilance Task During Total Sleep Deprivation

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Summary: Nine young adult male (23–30 years old) paid volunteers were subjected to total sleep deprivation (TSD), after two consecutive nights in the laboratory, for 40 hours (from 0800 hours on the first day to 2400 hours on the following day). Oral temperature (OT), reaction time (RT) in a visual vigilance task, and electroencephalogram (EEG; C3, C4, T3, and T4) while performing the task were recorded every 2 hours during TSD and after recovery sleep. One second of EEG, before target and non-target stimuli for every subject and condition was visually inspected, and artifact-free epochs were Fourier transformed. Absolute power (AP) was calculated for 4–20 Hz (full band) and for theta, alpha1, alpha2, and beta1. Analyses of variance (ANOVAs), with TSD and time-of-day as factors, showed the following significant results: TSD induced an increase in RT and AP of the full band at C3 and C4, of all bands at C3, of theta at T3, and of beta1 at T4 ($p < 0.009$ for all comparisons). No time-of-day effects nor interactions were found. OT was not affected by TSD. All variables returned to baseline values after recovery sleep. RT and EEG power showed a linear increase with accumulating hours of wakefulness. The increment in RT also correlated with the increase in EEG power. The results demonstrate that the increment in RT is associated with the increase in AP, particularly in the left central cortex; that the EEG may be used to identify sleepiness; and that EEG during task performance is more sensitive to TSD than during relaxed wakefulness. **Key Words:** EEG power—Sleep deprivation—Reaction time—Oral temperature.

In three independent studies we have demonstrated that the effect of total sleep deprivation (TSD) is reflected on the waking electroencephalogram (EEG), recorded during relaxed wakefulness with eyes open and closed (1–3). Absolute power (AP) of the whole spectrum with eyes open, and of theta and beta with eyes closed, is higher after TSD, increasing linearly as hours of wakefulness accumulate and returning to pre-deprivation values after recovery sleep.

These changes in AP correspond with the increase of delta and theta, together with sleepiness, in train drivers as reported by Torsvall and Akerstedt (4), and with the increase in alpha and theta power that precedes incidents of dozing off, reported by the same authors (5).

On the other hand, TSD is known to cause various degrees of monotonic decrement in performance of a very broad range of variables, including vigilance, reaction time, arithmetic computations, short- and long-term memory, psychomotor tasks, and logical reasoning tasks. The longer the time period of previous wakefulness, the greater the decrement [see Babkoff et al. (6), Horne (7), and Horne (8) for review].

In one of the above-cited studies (3) we measured the reaction time (RT) and the number of errors in a vigilance task. We observed that the increase in RT was positively correlated with the number of hours of prior wakefulness and with the increase in theta AP during relaxed wakefulness.

The above findings, taken together, suggest that the increase in RT with TSD may also be related to the EEG during performance of the vigilance task. If so, it may be possible to observe a correlation between RT and AP during task performance. Thus, exposure to TSD should induce an increment of RT and AP during performance of the task. In contrast, recovery sleep should induce a decrement in both variables. In short, RT should increase parallel to the increase in AP and decrease after recovery sleep.

A second question relates to the diurnal peak of

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power at 1800 hours that was observed in the resting EEG (3) and the possibility that it may also be present during task performance. With these purposes in mind we analyzed the EEG activity of the same subjects during the performance of a vigilance task every 2 hours over a period of 40 hours of TSD and after recovery sleep.

METHODS

Subjects were recruited through an announcement in the university community soliciting healthy volunteers to participate in a study of sleep deprivation. Potential subjects were interviewed and asked to fill out a questionnaire on sleep habits and health. Nine paid male volunteers between 22 and 30 years old were selected on the basis of their history of regular sleep habits (bedtime between 2300 and 2400 hours and wake time between 0700 and 0800 hours). All were right-handed, as assessed by Annet's test (9), and free from sleep disorders, neurological problems, and medications. They were required to refrain from alcohol and caffeine intake during the study.

They slept ad libitum in the laboratory during 2 nights, the first for habituation and the second for control. Sleep was recorded and analyzed according to standard criteria (10), and the results were reported previously (3). Sleep stages 3 and 4 were combined to form stage 3+4. After spontaneous awakening from the second night's sleep (between 0700 and 0800 hours), the subjects remained at the laboratory under continuous surveillance and were not allowed to sleep for the following 40 hours (until 2400 hours of the next day). During most of this time, subjects were allowed to carry out their own preferred activities, such as reading, writing, playing games, or watching videos. Lying down, sleeping, and vigorous physical activity were not allowed. Subjects were tested with a vigilance task, and oral temperature (OT) and EEG were recorded every 2 hours, during deprivation (from 0800 hours of the first day to 2400 hours of the second day, for a total of 21 tests) and after recovery sleep. The recording session took about 25 minutes. The sampling rate of 2 hours allowed the observation of circadian as well as hemicircadian oscillations. OT was preferred over rectal temperature because it is less invasive and provides information accurate enough to monitor circadian variations.

At 2400 hours, on the second day, recovery sleep was allowed. It was divided into three blocks of sleep (R1, R2, and R3), separated by an enforced awakening, during which subjects were taken to the next room to perform the vigilance task. Each block of sleep consisted of 3 hours of sleep or two full sleep cycles. If the subjects were in paradoxical sleep, they were not awakened until it ended spontaneously. The test began 10 minutes after awakening to provide similar conditions for all subjects.

Vigilance task

Although frequent testing can induce boredom, boring tasks (11) and long-lasting tasks (12) are more sensitive to TSD. Therefore a simple 15-minute task was chosen.

A total of 150 visual stimuli were displayed, one at a time, in a random sequence on a computer monitor. To avoid laterality effects within the visual field, stimuli were always displayed at the center of the screen. The stimulus consisted of one of four patterns: a white square (3.7 × 3.3 cm) missing one of its four corners. The duration of the stimulus on the screen was 50 milliseconds, and the interstimulus interval ranged randomly between 5 and 7 seconds. Before starting the test, one of the four patterns was selected as a target stimulus, and it was randomly alternated with the other three (non-target stimuli) in a ratio of 1:3. Subjects had to press the ENTER key every time that the preselected target stimulus appeared. They were instructed to remain as quiet as possible during the task, to pay attention to the monitor, and to respond as quickly as possible to the selected stimulus.

The program triggered the capture of EEG activity 1 second before the stimulus was displayed, and it computed RT and two types of incorrect responses: failures to respond to the target stimulus, defined as omissions; and incorrect responses, when subjects pressed the ENTER key for non-target stimuli, defined as errors. The total duration of the test was about 15 minutes. The variation in the total duration was due to the random interstimulus interval. The test was applied on the second night before going to sleep in order to make sure that the subjects understood the instructions.

Electroencephalographic activity

EEG activity was recorded with golden cup electrodes at C3, C4, T3, and T4, referred to the ispilateral earlobes according to the 10–20 International System. A Grass model 8–16E polygraph was used, with a time constant of 0.1 second and a low pass filter with a cutoff frequency at 35 Hz. Electrode impedance was kept below 10 kOhm. Monopolar rather than bipolar recording was preferred to maintain recording conditions comparable to those in previous studies (1–3). One second preceding every target stimulus and preceding one type of non-target stimulus of the same pattern was stored in a personal computer at a sampling rate of 128 Hz through an analog-to-digital converter with 12-bit resolution for off-line analysis.

Electroencephalographic signals were carefully inspected off-line, and those segments showing signs of eye movements or muscular artifacts were discarded. The remaining segments (at least 20 target and 20 non-target stimuli per subject per condition) were digitally filtered using a square window by means of a Fast-Fourier transform into the traditional broad bands: delta, theta, alpha1, alpha2, beta1, and beta2 (13). Only power from 4 to 20 Hz, defined as full band power (FB), and therefore power of theta (4–7.5 Hz), alpha1 (7.5–9.5 Hz), alpha2 (10–12.5 Hz), and beta1 (13–20 Hz) bands, was considered for statistical analysis. Although the present sampling rate allows resolution from 0.5 to 64 Hz, it was decided to discard the delta and beta2 bands from statistical analysis. This was done because the visuo-motor nature of the performance task could induce eye movements that would be reflected on the delta band and because high EEG frequencies can be confounded with muscular activity (14). For each frequency interval, we integrated the power density across the conventional bands and the full band, and AP values were log transformed (15).

Three kinds of analyses were performed: 1) Comparisons between pre- and post-deprivation day. To assess the effects of sleep deprivation and time of day, as well as their interaction on performance, OT, RT, and EEG AP and relative power (RP) during normal waking hours [data collected at 1000, 1200, 1400, 1600, 1800, 2000, and 2200 hours on the day prior to sleep deprivation (pre-deprivation) and on the day after the night of sleep deprivation (post-deprivation)] were submitted to two-way analyses of variances (ANOVAs) for repeated measurements. Omissions and errors were submitted to the Friedman test. Deprivation was one factor and time of day was the second factor. The level of significance was set at $p < 0.009$ to reduce type I error. Tukey's Student t tests and Wilcoxon tests were used for post-hoc pairwise comparisons. Because there was no equivalent control for data from 2400 hours to 0600 hours, they were not considered in these analyses. The first testing session (0800 hours) was also omitted to maintain equivalent circadian times, and only data from 1000 to 2200 hours were taken into account. 2) Pearson product-moment correlations between hours of sleep deprivation and performance, OT, and AP, with all of the data collected during the 40-hour period of sleep deprivation (21 measurements), were performed. 3) One- and two-way ANOVAs were performed for the recovery period.

RESULTS

No significant differences were found between the EEG activity from target and non-target stimuli; therefore, the following tests were carried out on the EEG from target stimuli.

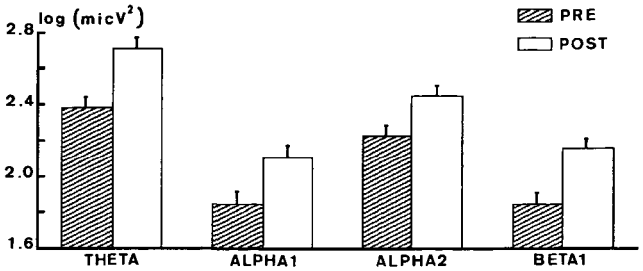

FIG. 1. Mean and standard deviation of absolute power, log transformed, at C3 during the control day before deprivation (PRE) and during the deprivation day (POST). The main effect of sleep condition was pooled with time of day (ANOVA: sleep condition×time of day).

Comparison between pre- and post-deprivation day

First, EEG power and performance in the vigilance task were submitted to two-way ANOVAs to determine the main effects of sleep deprivation conditions (pre-deprivation and post-deprivation days) and time of day (1000, 1200, 1400, 1600, 1800, 2000, and 2200 hours), as well as their interaction.

The main effect of deprivation was a significant increase in RT [F (1,144) = 49.81; $p < 0.001$] with TSD. However, the main effect of time of day was not significant, despite of a trend showing longer RTs in the early morning. Omissions and errors were pooled across three successive test periods (i.e. 0800, 1000, and 1200 hours) and were submitted to the Friedman test. The number of omissions increased significantly with TSD ($Xr = 23.5$; $p < 0.0006$). The Wilcoxon test showed that they were greater between 0200 and 0600 hours compared to previous values, and that they continued to increase with every three successive test periods. The number of errors increased along with the time of TSD, but the difference was not significant.

In the case of AP, the main effect of deprivation showed a significant increase of the full band power from 4 to 20 Hz at C3 and C4 ($p < 0.001$ for both comparisons), and the difference came close to significance at T3 ($p < 0.02$). Similar ANOVAs were performed with AP for each traditional band. These analyses revealed significant main effects of deprivation for AP of all bands at C3 ($p < 0.003$ for all comparisons) (Fig. 1), of theta at T3 ($p < 0.009$), and of beta1 at C4 ($p < 0.001$). A similar, nonsignificant, increasing trend was observed for AP for the rest of the bands at C4, for alpha1 at T3, and for beta1 at T4 ($p < 0.02$ for all comparisons). The main effects of time of day were not significant for any EEG band, nor were the interactions between deprivation and time of day.

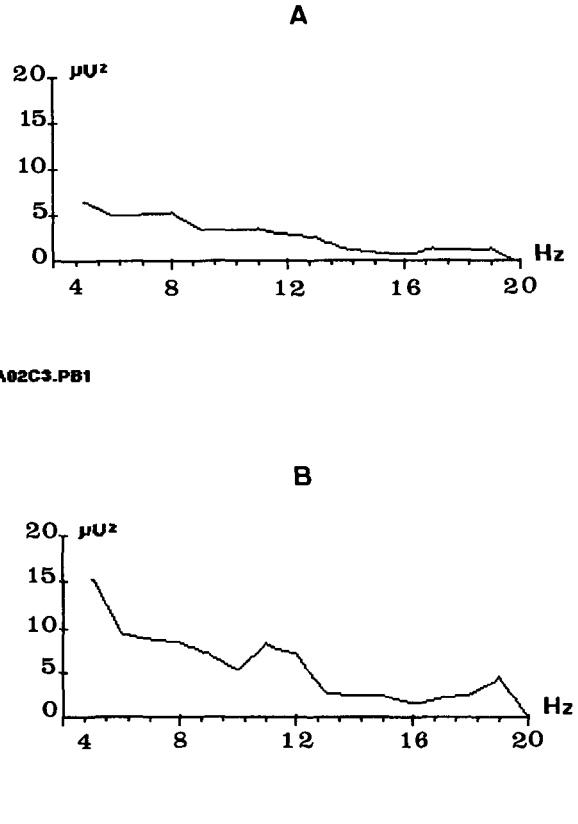

FIG. 2. Electroencephalographic spectra from 4 to 20 Hz in one subject. The changes in the EEG spectrum across 40 hours of TSD are illustrated. Each spectrum represents the mean absolute power over all target stimuli from the task at 1000 hours on the pre-deprivation day (A) and the task at 2200 hours on the post-deprivation day (B).

The effect of TSD on EEG power is exemplified in Fig. 2. The spectra for one subject at 1000 hours on the pre-deprivation day and at 2200 hours on the post-deprivation day are shown.

Oral temperature, discussed in detail previously (3), showed a significant main effect for time of day [$F(1,144) = 49.81$; $p < 0.001$], with significant minimum values at 0600, 0800, and 1000 hours and maximum values at 2000 hours. Neither the deprivation effect nor the interaction were significant.

Relationships between variables

To describe the variation over time, we computed Pearson product-moment correlations between the number of hours of previous wakefulness and RT, and between the number of hours of prior wakefulness and AP of the EEG bands. These correlations revealed significant differences in the ANOVAs with all of the data collected during the 40-hour period of sleep deprivation (21 measurements). The time course of RT, as well as that of AP changes, exhibited a linear increase with accumulating hours of wakefulness (Table 1). All bands at C3 (Fig. 3), beta1 at C4, and theta at T3 showed significant correlations at an alpha level of 0.001. Alpha2 at C4 and beta1 at T4 showed a weaker, but also significant, relation ($p < 0.01$). RT also correlated positively with the amount of prior wakefulness ($r = 0.85$; $p < 0.001$).

TABLE 1. *Correlation coefficients (r) between EEG absolute power and the amount of wakefulness (40 hours)*

	C3	C4	T3	T4
Theta	0.65***[a]	0.50*	0.57***	0.34
Alpha1	0.56**	0.46	0.26	0.27
Alpha2	0.60***	0.56**	0.48*	0.37
Beta1	0.64***	0.64***	0.41	0.56**
Total band	0.67***	0.64***	0.55**	0.27

[a] * indicates significance at $p < 0.05$; ** indicates significance at $p < 0.01$; and *** indicates significance at $p < 0.001$.

As a further check on the association between the AP and RT increment with TSD, correlations were computed between AP and RT (Table 2). Theta AP at C3 (Fig. 4) and T3, alpha1 at C3, and beta1 at C3, C4, and T4 showed significant correlations ($p < 0.01$). Full band power also correlated positively at C3, C4, and T3 with RT ($p < 0.05$). EEG power did not exhibit any significant correlation with OT.

To illustrate the changes in EEG power and RT with TSD, we selected the tasks with minimum and maximum hours of wakefulness at equivalent circadian points (1000 and 2200 hours). Power density for the conventional bands was expressed as a proportion of the value observed during the first task. The maximum increase reached 171% for alpha1, and the minimum reached 125% for the alpha2 band (Fig. 5).

To determine the effect of recovery, separate ANOVAs were performed on EEG power and RT with recovery (first task at 0800 hours, last task at 2400 hours, R1, R2, and R3 values) as one factor, and in the case of EEG power, with hemispheres as the second factor.

Results of ANOVAs showed significant main effects of recovery for all EEG bands and for RT ($p < 0.003$ for all comparisons). Pairwise comparisons confirmed the increment of power and of RT after deprivation. They also showed that in the case of EEG, AP of all bands recovered baseline values after the first block of sleep (there were no significant differences between the first task at 0800 hours and R1 values). RT, however, continued to be longer after R1 and R2, and it recovered the baseline value only after R3 (Fig. 6). In the case of AP, no significant main effects of hemisphere, nor hemisphere by recovery interactions, were found.

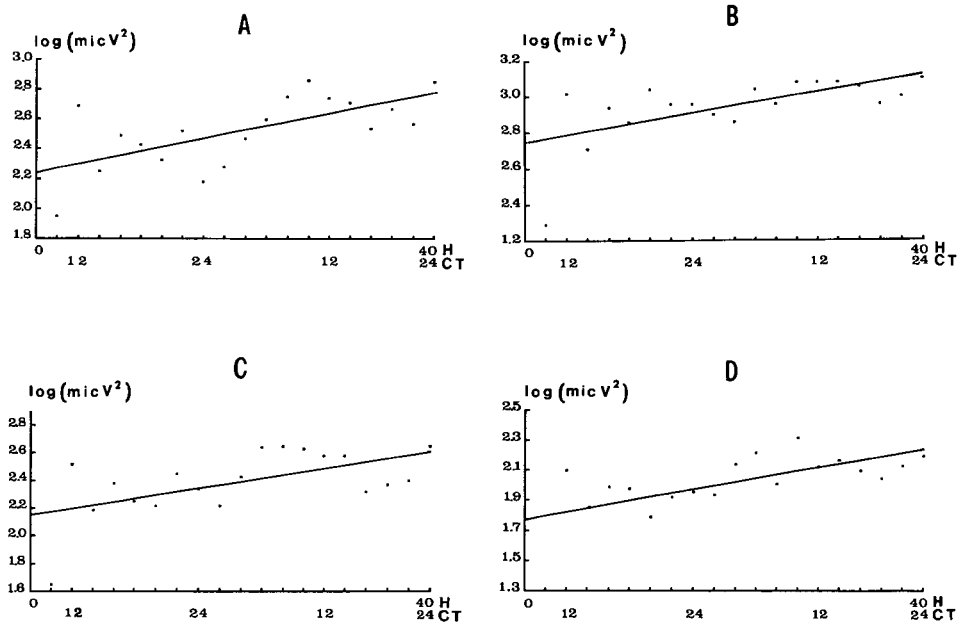

FIG. 3. Linear trend between 40 hours of sleep deprivation (TSD) and absolute power of theta (A), alpha1 (B), alpha2 (C), and beta1 (D) at C3, log transformed, every 2 hours during task performance. Clock time is also indicated (CT).

DISCUSSION

The EEG results clearly demonstrate that the effect of TSD is reflected on the EEG during the performance of a vigilance task. EEG power of the total band (4–20 Hz) was higher after than before TSD. The left central cortex was clearly most sensitive to TSD; the increase of power was significant for all bands at this derivation, followed by the right central and the left temporal derivations. As hypothesized, recovery sleep reverted the EEG changes induced by TSD; EEG recovered baseline values after the first 3-hour block of sleep.

Although subjects were right-handed and had to press the ENTER key every time they detected the target stimuli, the greater response of C3 to TSD cannot be attributed to the motor response alone. This is because during relaxed wakefulness it is also at the left central cortex where the stronger effects are observed (3). In addition, the effect is observed for all EEG bands and for non-target stimuli as well. Of the four derivations explored, C3 represents the best choice for studying the effect of TSD on the EEG.

The increment of EEG power during the performance of the task with TSD followed the same pattern observed for the EEG during relaxed wakefulness, that is, a linear increase with accumulating hours of wakefulness. However, EEG during task performance showed a greater response to TSD than EEG during resting conditions. The maximum increase, reaching 171%, was observed for the alpha1 band, followed by theta (146%) and beta1 (143%), during task performance. These values compare to 37% and 26%, for theta and beta1, respectively, for the EEG during relaxed wakefulness in the same subjects under similar TSD conditions (3).

The present results agree with those reported by Torsvall and Akerstedt (4), who found higher theta and

TABLE 2. *Correlation coefficients (r) between EEG absolute power and reaction time*

	C3	C4	T3	T4
Theta	0.54**[a]	0.42	0.50*	0.31
Alpha1	0.46*	0.43	0.11	0.29
Alpha2	0.37	0.34	0.27	0.25
Beta1	0.50*	0.45*	0.34	0.56**
Total band	0.49*	0.47*	0.45*	0.28

[a] * indicates significance at $p < 0.05$; ** indicates significance at $p < 0.01$.

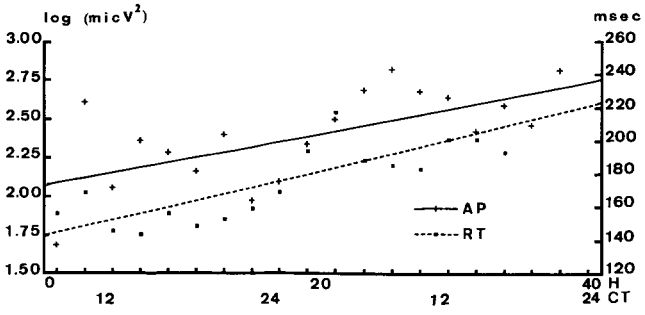

FIG. 4. Linear trend between 40 hours (H) of sleep deprivation and theta absolute power (AP) at C3, log transformed, and reaction time (RT), in milliseconds, every 2 hours. Clock time is also indicated (CT).

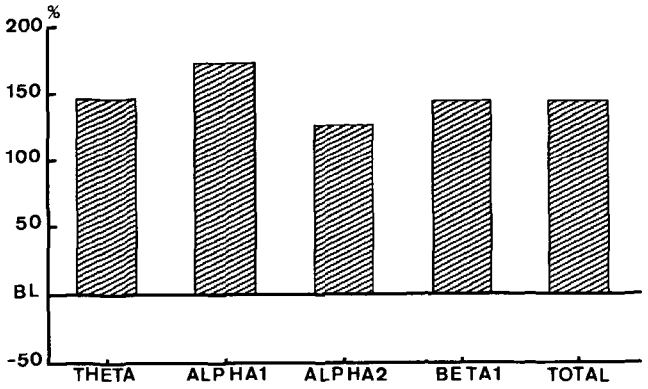

FIG. 5. Power density for the conventional bands during the last task, expressed as a proportion of the value observed during the first task (BL).

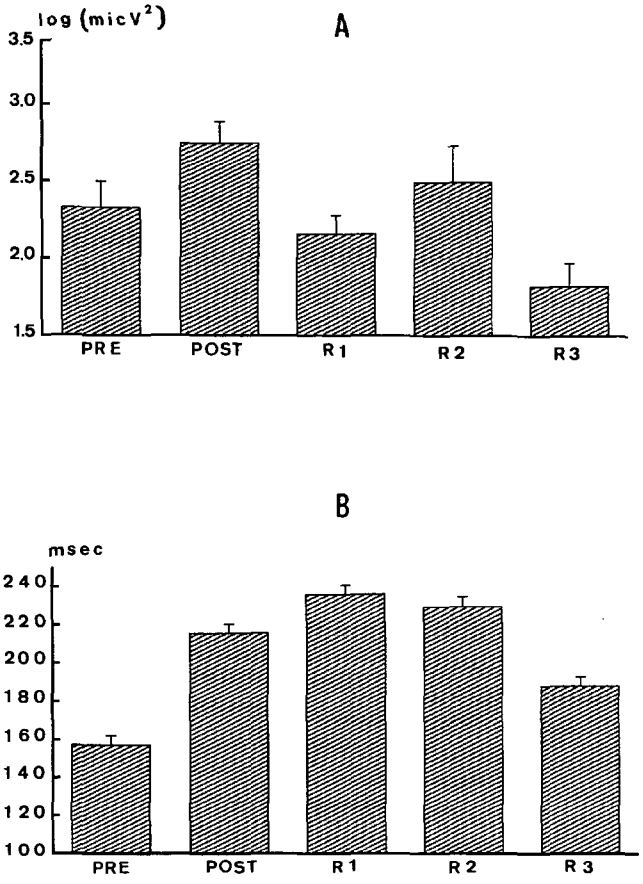

FIG. 6. Recovery. Shown are the mean and standard error of absolute power of theta at C3 (A), log transformed, and reaction time (RT), in milliseconds (B), during performance of the vigilance task at 0800 hours on the pre-deprivation day (PRE), at 2400 hours on the post-deprivation (POST) day, and after one (R1), two (R2), and three (R3) blocks of recovery sleep.

alpha activity associated with sleepiness and with failures to respond to external signals in train drivers during the night. The results in this study also agree with the increase of alpha and theta power preceding incidents of dozing off that were reported by the same authors (5). We also observed higher beta activity, however, along with the increase in theta and alpha power. A similar pattern, that is, a power increase of the whole spectrum, was observed during relaxed wakefulness with eyes open (3). Alpha activity, extending to lower frequencies, is considered to reflect thalamo-cortical inhibitory processes, which are triggered by sleep. Beta activity, however, is considered to reflect cortical activation (16). We interpret this combined pattern of higher power of slow as well as high frequencies as the reflection, on the one hand, of an increasing preponderance of inhibitory processes or decreasing excitatory processes as hours of wakefulness accumulate. On the other hand, it can be interpreted as a result of an effort to maintain alertness that is not enough to counterbalance the decrement caused by TSD on performance. If this is true, a beta reduction should be expected if hours of wakefulness are extended further. This hypothesis must wait to be tested.

An alternative explanation for the increase in beta power is the interference of electromyographic activity (14). The increased beta power was more accentuated at central than at temporal derivations, however, and the reverse relation should be expected if the increase in beta power were due to electromyographic activity. Furthermore, frequencies above 20 Hz were not considered.

As expected, TSD was associated with an increase in RT, particularly toward the end of the second day. A monotonic decrement of performance with TSD is a well-documented phenomenon (6). However, to our knowledge it has not been assessed simultaneously with EEG. The present results show a parallel increase of RT and of EEG power, which is stronger for theta, beta1, and for the full band.

Surprisingly, the main effect of time of day on RT was not significant. It had been expected to be significant according to the well-established circadian oscillation in the vigilance level and task performance (6), but a nonsignificant trend to longer RT around daybreak was observed. This lack of significance may be attributed to intersubject variability, perhaps due to uncoupled circadian cycles between subjects.

The lack of significant overall effects of time of day and the lack of interactions between time of day and deprivation, together with the positive, highly significant correlation between RT and the amount of accumulated wakefulness, indicate a monotonic linear decrement of performance. This was also reflected in the higher number of omissions, whereas the number of errors was kept constant along with the deprivation. This latter observation rules out the possibility of a decline in the willingness to respond to the task (17).

In addition, we found that the diurnal oscillation of

the EEG power, with a peak at 1800 hours that was during relaxed wakefulness, was masked in the EEG during performance of the task. Also, the correlation between OT and EEG power during relaxed wakefulness was not observed in the EEG while a task was being performed. These facts suggest that the engagement in a task is enough to mask or counterbalance diurnal oscillations, and they indicate that models of sleep regulation should take into account environmental demands on the organism in addition to homeostatic or circadian and ultradian processes.

In conclusion, the present results demonstrate that EEG activity during the performance of a vigilance task reflects the amount of accumulated wakefulness, and that the increment in RT is associated with the increase in AP, particularly in the left central cortex. We agree with Akerstedt and Gillberg (18) in that the EEG during relaxed wakefulness, as well as during cognitive engagement, may be successfully used to identify sleepiness, but the EEG during task performance seems to be more sensitive than that during relaxed wakefulness.

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