Amplitude Reduction in Visual Event-related Potentials as a Function of Sleep Deprivation

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Summary: Eight adult males were subjected to 40 hours of total sleep deprivation (TSD). Reaction time in a visual task and electroncephalographam (C_3) were evaluated every 2 hours. One second of EEG before the stimuli was Fourier-transformed, and 750 ms after target and nontarget stimuli were averaged and visual event-related potentials (ERP) were obtained. Factorial analysis identified time windows that showed significant amplitude reduction and longer latencies with TSD: (1) 140 to 288 ms (P180-N242-P281); (2) 288 to 413 ms and 601 to 749 ms (N382; P718) and; (3) 531 to 601 ms (N500). Effect was strongest for N382 and P718, the amplitudes of which dropped to 20% of original size. The entire waveform recovered initial amplitudes and latencies after recovery sleep except for P718 latency. Waveforms within similar time intervals have been associated with attentional gating, sensory discrimination, target selection, uncertainty and decision processes. Amplitudes of the visual ERP were inversely correlated with hours of TSD, reaction time, and absolute power of the prestimulus EEG. Present results clearly show changes in fundamental neurophysiologic mechanisms as a result of TSD, indicating variability and reduction of the alertness mechanisms and changes in thalamocortical gating affecting attention, discrimination and decision-making.

Key words: Visual event related potentials; sleep deprivation; waking EEG; prestimulus EEG

Both physiologic and behavioral data have shown that total sleep deprivation (TSD) induces a decrease in speed and efficiency of cognitive processing (for review see refs. 1-3), particularly in the ability to sustain attention for prolonged periods of time.[4] This deterioration in cognitive functions must be due to changes of state in the brain mechanisms underlying such functions. Quantitative spectral analysis of EEG activity has demonstrated that alertness as assessed by EEG activity during relaxed wakefulness with eyes open and accumulating hours of wakefulness are linearly related; absolute power (AP) of the whole spectrum progressively increases with 40 hours of TSD, and the increase in AP of waking EEG is directly proportional to slowing of reaction time in a visual vigilance task.[5] Sleepiness in train drivers is associated with higher theta and delta AP, and failures to respond to external signals with theta and alpha AP.[6,7] These results suggest that deterioration of alertness and performance during sleep loss are due to changes in fundamental neurophysiologic mechanisms.

Event-related potentials (ERP) recorded from scalp provide higher temporal resolution and precision for temporal tracking of perceptual and cognitive processes than EEG. ERP techniques have been used successfully to investigate cognitive processing in humans. As a result, specific and nonspecific components of the ERPs have been linked to different mental processes such as stimulus analysis, attention, discrimination, retrieval of relevant memories or updating, evaluation of the significance of the

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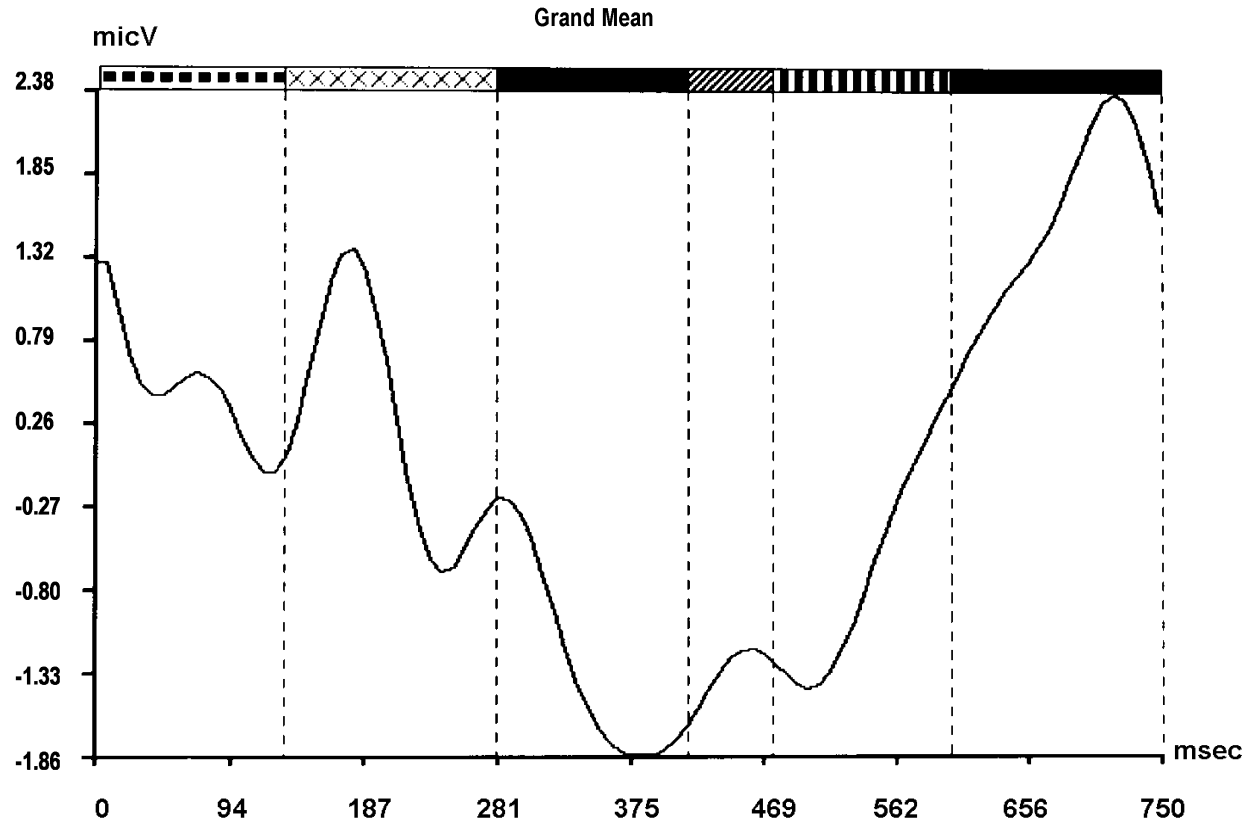

Figure 1.—Grand mean of the ERP for all subjects, conditions, target and nontarget stimuli. The horizontal bar at the top of the figure indicates the time windows corresponding to the five factors identified by principal component analysis: factor 1, black; factor 2, diagonal lines; factor 3, squares; factor 4, crosses; factor 5, vertical lines.

stimulus, and encoding of the event into semantic representations.[8]

Several studies have shown that ERPs are also influenced by arousal level and attention. Amplitude of ERPs is augmented with arousal under conditions of attention,[9-15] whereas progressive decrease in amplitude of the ERP by habituation and fatigue[16-23] and under sedative medication[24] has been reported.

The event-related desynchronization observed before and during cognitive processing is an electrophysiologic correlate of cortical activation and preparation to process sensory information.[25] There is evidence that the spontaneous activity preceding an external stimulation influences the behavioral response to that stimulus as well as the amplitude and latencies of peaks of the event-related potential.[26] We have previously reported the waking EEG changes and the time course of these changes during 40 hours of TSD, evaluated every 2 hours while engaged in a visual vigilance task; we observed a direct linear relationship between power of the EEG preceding the stimuli and hours of TSD and an inverse relationship between spectral power and reaction time.[27] This evidence taken together led us to hypothesize that the cognitive deterioration and reduced reactivity induced by TSD are consequences of neurophysiologic changes in brain processes that would lead to decreased amplitudes and longer latencies in the ERP. In the present work we analyzed the visual event-related potentials from the same subjects and from the same visual task as the 1996 study of Corsi-Cabrera et al.[27]

METHODS

Eight male volunteers between 22 and 30 years of age participated in the experiment. Volunteers were recruited from the university community through announcement. They were selected among potential subjects after an interview and a questionnaire on sleeping habits and health. All participants gave their consent to participate in a sleep-deprivation study, and were free from sleep disorders, neurologic problems, and medications, had regular sleep habits, and were right-handed as assessed by the Annet's test.[28]

After 1 night of habituation and 1 night of sleep ad libitum at the laboratory, subjects remained awake under continuous surveillance during 40 hours beginning after their

Table 1.—For each factor, the percentage of variance accounted for, the time interval and mean amplitudes and latencies of peaks included, and statistical results from ANOVAs

	Factor 1	Factor 2	Factor 3	Factor 4	Factor 5	
Pattern in Fig. 1						
Variance explained (%)	29.35	19.32	10.8	8.87	8.05	Total variance 76.38
Time window (ms) accounted in each eigenvector	288-413 601-750	421-523	0-132	140-288	531-601	750
Peak latency (ms) and voltage (micV)	N382 -1.86 P718 2.38	P460 -1.17 N500 -1.42	N46 0.45 P70 0.59 N125 -0.05	P180 1.8 N242 -0.68 P281 -0.21		
Significance between PRE-D day and POST-D day *	P<0.001 $F_{(1,104)}$ =54.27	--------	-------	P<0.001 $F_{(1,104)}$=16.12	P<0.001 $F_{(1,104)}$=12.53	

*Two-way ANOVAs, Variable A: PRE-D day vs POST-D day; Variable B: time of day.

spontaneous awakening between 0700 and 0800 hours and ending at 2400 hours of the next day. During all of this time they were allowed to engage in their own preferred activities but were prevented from sleep, lying down, vigorous physical activity, and intake of alcohol or stimulants such as caffeine. Every 2 hours, subject peformance was tested in a visual vigilance task and EEG was recorded (a total of 21 evaluations: 0800, 1000, 1200, 1400, 1600, 1800, 2000, 2200, 2400, 0200, 0400, 0600, 0800, 1000, 1200, 1400, 1600, 1800, 2000, 2200, and 2400 hours). After the 40 hours of sleep deprivation, recovery sleep was allowed beginning at 2400 hours, divided into three blocks of sleep interrupted by an enforced awakening during which subjects were again tested in the vigilance task and the EEG was recorded. Each block of sleep consisted of 3 hours of sleep or two full sleep cycles.

The visual discriminative task consisted of 150 visual stimuli displayed at the center of a computer monitor. Stimuli were white squares (3.7 × 3.3 cm) missing one of the four corners displayed one at a time for 50 msec. Interstimulus interval was randomly varied between 5 and 7 seconds. Before beginning the tests, one of the four squares was selected as target stimulus and was randomly alternated with the other three (in a ratio of 1:3). Subjects were instructed to remain as quiet as possible, to pay attention to the monitor and press the 'enter' key as soon as possible when the target stimulus appeared, and to do nothing when the other three squares appeared (nontarget stimuli). A computer program triggered the capture of EEG activity 1 second before (PRE-S EEG) and 750 msec after (POST-S EEG) every target and every one of the three nontarget stimuli and computed reaction time (RT), failures to respond to target stimuli (omissions), and responses to nontarget stimuli (errors). The total duration of the test was about 15 minutes. Subjects practiced the test before going to sleep the second night at the laboratory.

Electroencephalographic Activity

EEG activity was recorded with gold cup electrodes at C_3 referred to ipsilateral earlobe in a Grass model 8-16 E polygraph with filter settings at 1 and 35 Hz. Electrode impedance was kept below 10 Kohms. EEG activity was sampled at 128 Hz through an analog-to-digital converter with 12 bits resolution. The stored EEG epochs were carefully inspected, and segments showing eye movements, muscular artifacts, or stage 1 sleep were eliminated.

The corresponding voltage values of all artifact-free epochs of POST-S EEG of every subject, target and nontarget stimuli and conditions were submitted to principal component analysis in order to identify time intervals that covaried independently and those that covaried together, and to reduce and orthogonalize variables. This method provides information on the variance accounted for by successive factors sequentially across the ERP latency epochs that may correspond to successive stages of information processing, even in cases of temporal overlap of these processes. Varimax rotation of a reduced number of factors weighing more than 1.00 was obtained, and the rotated component scores were the raw data for further statistical analyses.[29]

All artifact-free trials of every subject and conditions from target and nontarget stimuli were averaged and the grand mean was obtained. The artifact-free epochs from all subjects and tasks were also separately averaged to obtain ERPs for each subject, target and nontarget stimuli, and conditions. Peaks were visually identified for each factor of the principal component analysis for the grand mean and for each subject's target, nontarget, and condition. Peak latency was determined from the recordings with the largest amplitude, and baseline-to-peak amplitudes were also calculated. Baseline was defined as the average voltage from 1000 msec preceding stimulus onset.

The corresponding artifact-free epochs of PRE-S EEG were submitted to fast Fourier transformation, and absolute power (AP) for the traditional broad bands was calculated. Only theta (4-7.5 Hz), alpha$_1$ (7.5-9.5 Hz), alpha$_2$ (10.0-12.5 Hz), and beta$_1$ (13-20 Hz) were considered for statistical analysis. Delta and beta$_2$ were excluded because the visuomotor nature of the task could induce eye movements that would be reflected on the delta band and muscular activity that would be confounded with high frequencies. AP was log transformed for statistical analysis.

The following statistical analyses were performed: (1) to assess the difference between target and nontarget ERPs, one-way ANOVAs for repeated measures, one for each of the factors weighing more than 1.00, were computed with the rotated component scores of target and nontarget stimuli. There was no statistical significance between the ERP of the target and nontarget stimuli, and therefore the data of all stimuli, target and nontarget, were used for the rest of the comparisons; (2) to assess the effect of TSD on ERP, two-way ANOVAs for repeated measures, one for each of the factors weighing more than 1.00 using the rotated component scores, were computed with evaluations of the predeprivation day (PRE-D) and postdeprivation day (POST-D) as one variable and time-of-day as the other variable. Data from 1000, 1200, 1400, 1600, 1800, 2000, and 2200 hours from PRE-D and POST-D entered the analysis. Data from 2400, 0200, 0400 and 0600 hours were not considered in these analyses since there is no equivalent control; (3) latency of the peaks included in those factors showing significant TSD effects were submitted to two-way ANOVAs for repeated measures with evaluations of the PRE-D and POST-D as one variable and time-of-day as the other variable; (4) the effect of recovery sleep was assessed with one-way ANOVAs for repeated measures with evaluations from 1000 hours of PRE-D, 2200 hours of POST-D, and recovery as variables; (5) to assess the relationship between accumulating hours of wakefulness and ERP, linear regressions were calculated with the number of hours of sleep deprivation (from 1000 hours of PRE-D to 2400 hours of POST-D) as predictor variable and latency and voltage of ERP peaks as dependent variables; (6) the relationship between the changes in ERP and performance was calculated with Pearson product moment coefficients between RT and latency and amplitude of ERP peaks; and (7) the relationship between preparatory EEG activity and ERP was evaluated with Pearson product moment coefficients between ERP and PRE-S EEG.

RESULTS

The principal component analysis gave rise to five independent eigenvectors or factors which accounted for 76.34% of the variance. Table 1 shows for each factor the percentage of variance accounted for, the time interval and

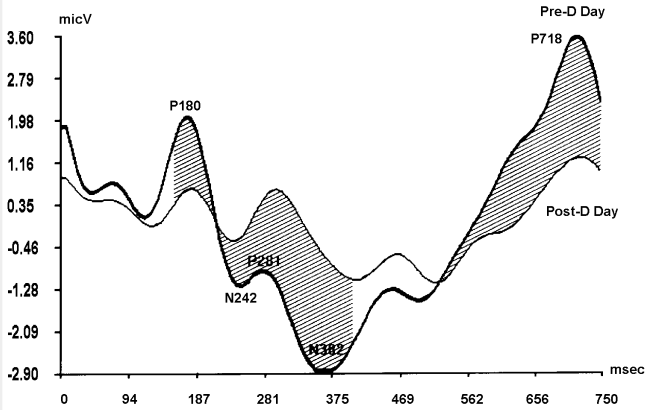

Figure 2.—This figure shows the effect of total sleep deprivation on the amplitude of the ERP. The broad line shows the mean of the ERP for all subjects, target and nontarget stimuli from 1000, 1200, 1400, 1600, 1800, 2000 and 2200 hours of the predeprivation day (PRE-D day) and the fine line the mean for the same hours of the postdeprivation day (POST-D day). Hatched areas show time intervals with significant differences between PRE-D day and POST-D day (Main effect of ANOVAs).

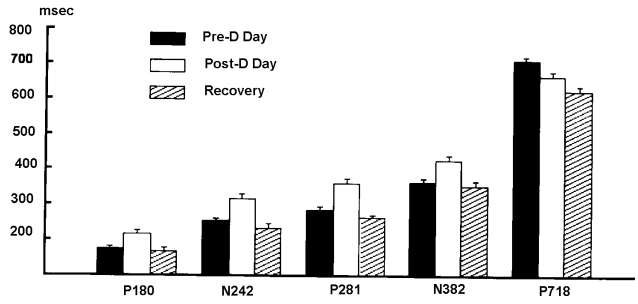

Figure 3.—Mean and standard error of latencies of the peaks of the ERP showing significant differences between predeprivation (PRE-D day), postdeprivation days (POST-D day) and recovery.

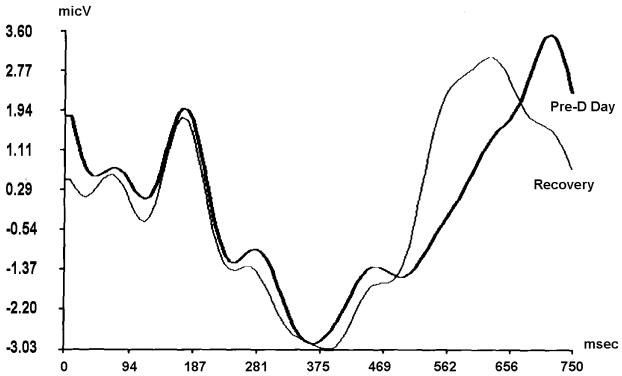

Figure 4.—This figure shows the effect of recovery sleep on the amplitude of the ERP. The broad line shows the mean of the ERP for all subjects, target and nontarget stimuli from 1000, 1200, 1400, 1600, 1800, 2000 and 2200 hours of the predeprivation day (PRE-D day) and the fine line the mean after recovery sleep.

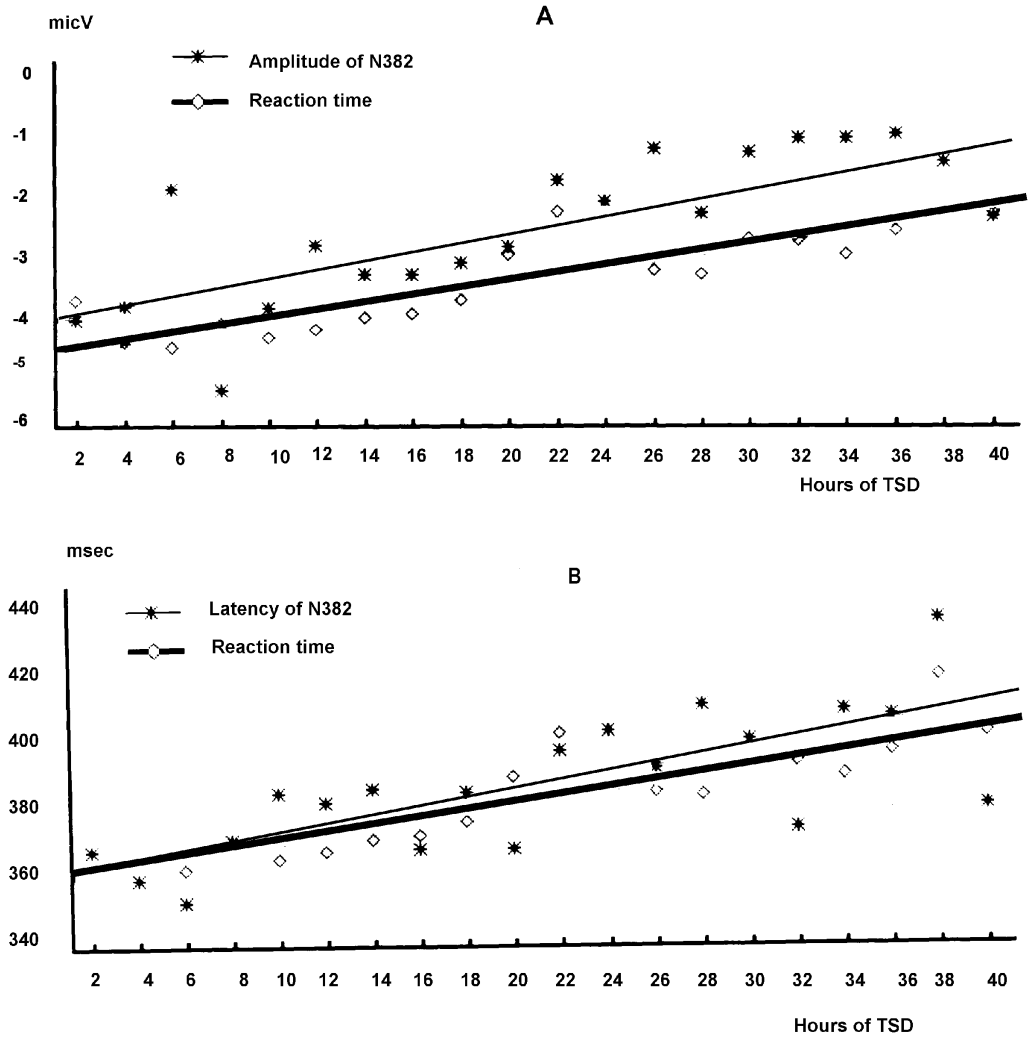

Figure 5—Slopes of reaction time (broad line) and amplitude (fine line in A) and latency (fine line in B) of peak N382 for the group as a function of hours of sleep deprivation. (TSD). Observe also the correlation between the increase in RT and the decrease in microvolts of N382 ($r = 0.54$; $p < 0.05$), and between the increase in RT and the increase in latency ($r = 0.58$; $p > 0.01$).

mean amplitudes and latencies of peaks included, and statistical results from ANOVAs. Figure 1 shows the grand mean, with time intervals included in each factor identified by principal component analysis. The identified five factors were formed by the following time windows and peaks: (1) the first factor (29.35% of variance) from 288 to 413 msec and from 601 to 750 msec, included N382 and P718 peaks; (2) the second (19.32% of variance) from 421 to 523 msec, included P460 and N500; (3) the third (10.80% of variance) from 0 to 132 msec, included N46, P70 and N125; (4) the fourth (8.87% of variance) from 140 to 288 msec, included P180, N242 and P281 and; the fifth (8.05% of variance) from 531 to 601 msec.

Effect of TSD on ERP

Factors 1, 4, and 5 showed significant main effects for TSD condition, whereas factors 2 and 3 did not show significant effects (Table 1). There were no significant time-of-day effects or interactions for any factor. Figure 2 shows the effect of TSD on the ERP. The amplitude of virtually all late components of the ERP was significantly decreased by TSD and the effect was stronger for peaks N382 and P718, both included in factor 1. The amplitudes of these peaks were 42% and 44% respectively, lower than during PRE-D. The latency of P180 [$F(1,104)=7.19$; $p<0.008$], N242 [$F(6,104)=6.82$; $p<0.00003$], P281 [$F(1,104)=17.42$; $p<0.0002$] and N382 [$F(1,104)=300.53$; $p<0.00001$] were significantly longer and the latency of P718 [$F(1,104)=5.70$; $p<0.01$] was shorter on POST-D (Fig. 3).

Effect of Recovery Sleep on ERP

There were no significant differences between initial values of the ERP and those after recovery sleep. In fact, the ERP recovered the same waveform as before PRE-D

Table 2.—Linear regressions of hours of wakefulness on amplitude and latency of ERP peaks and Pearson's correlations of amplitude and latency of ERP peaks with reaction time.

		Amplitude				
		P180	N242	P281	N382	P718
Hours of wakefulness	R^2	0.64***	0.22*	0.42***	0.58***	0.47***
	r	-0.79***	0.47*	0.64**	0.76**	-0.69***
Reaction time	r	-0.59**	------	-----	0.54*	-0.47**
		Latency				
		P180	N242	P281	N382	P718
Hours of wakefulness	R^2	0.23*	-----	0.36***	0.50***	0.28***
	r	0.48*	-----	0.61**	0.71***	-----
Reaction time	r	0.67***	0.47*	0.65***	0.58**	-----

N = 20; df = 18; * p < 0.05; ** p < 0.01: *** p < 0.001

after recovery sleep, except for peak P718 (p>0.05) latency, which was significantly shorter during recovery (Fig. 4).

Relationship between ERP and hours of wakefulness.—The amplitude of peaks P180, N242, P281, N382 and P718 and the latency of peaks P180, P281 and N382 showed linear increase with accumulating hours of sleep deprivation (Table 2). Figure 5 illustrates the slopes for amplitude (5A) and latency of N382 (5B) as a function of the number of hours of wakefulness. The greater the number of hours of wakefulness, the lower the amplitudes and the longer the latencies.

Relationship between ERP and performance.—RT was significantly slowed down with TSD as reported elsewhere.[27] The reduction in amplitudes of P180, N382, and P718 and the lengthening in latencies of all peaks was significantly correlated with the slowing of reaction time (Table 2 and Fig. 5).

Relationship between prestimulus EEG and ERP.—AP of theta, $alpha_1$, $alpha_2$ and $beta_1$ was significantly increased with TSD as reported elsewhere.[24] The amplitude decrease of the ERP time intervals included in factors 1 and 4 were significantly correlated with the increase in AP of theta (r=-0.64; p<0.01; r=-0.49; p<0.05), $alpha_1$ (r =-0.54; p<0.05; r=-0.54; p<0.05), $alpha_2$ (r =-0.51; p<0.05; r=-0.42; p<0.05), and $beta_1$ (r=-0.60; p<0.01; r=-0.59; p<0.01) and the decrease in amplitude of factor 5 with the AP of $beta_1$ (r=-0.48; p<0.05). The higher the AP the lower the amplitude of the ERP. Figure 6 illustrates this relationship for theta AP and factor 1.

DISCUSSION

The present results clearly indicate that TSD induces a progressive amplitude reduction and increase in latency in almost all late components of the event-related response during 40 hours of sustained wakefulness. The progressive amplitude reduction was linearly correlated with the slowing of the reaction time and with the prestimulus EEG. A significant proportion of variations was explained by accumulating hours of wakefulness.

The waveforms included in the time interval from stimulus onset to 132 ms (N46-P70-N125) did not change significantly with TSD. Feature analysis and attentional gating have been associated with middle latency components (10-100 ms) particularly to N100.[8,30] The lack of significant changes in amplitudes and latencies of peaks N46, P70, and N125 suggest that early stages of cognitive pro-

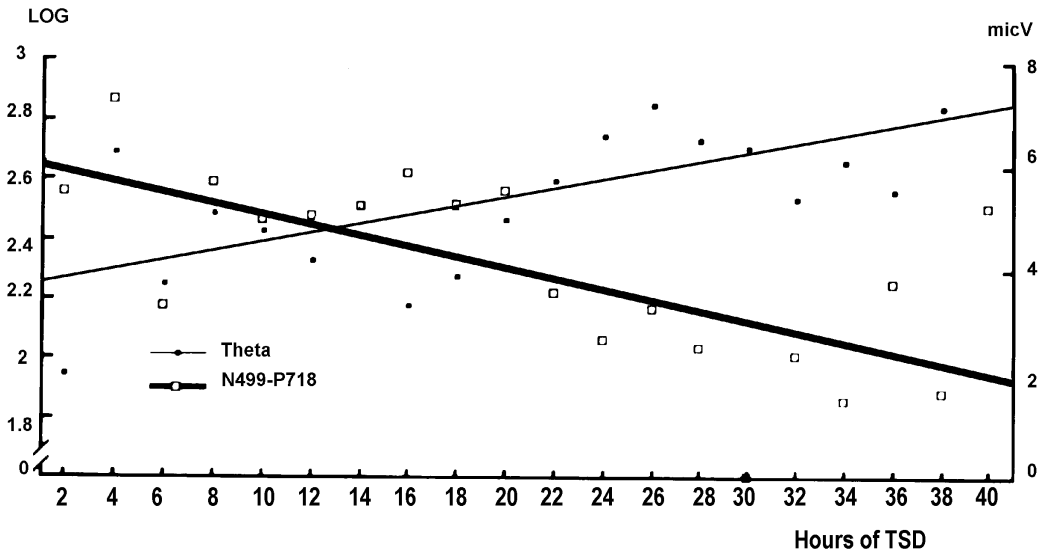

Figure 6.—Correlation between theta absolute power before the stimuli and amplitude of N382 for the group. Left axis shows absolute power of theta, log transformed, and right axis amplitude of N382 in microvolts.

cesses are not altered by 40 hours of TSD. However, although we did not find a significant reduction in N125 peak amplitude, the time interval identified as the fourth factor by factorial analysis began at 140 ms, including the slope from N125 to peak P180, and its amplitude was significantly reduced after TSD, indicating alterations in attentional gating.

On the other hand, the amplitude of P180-N242-P281 complex and of N382, N500, and P718 showed a progressive and linear reduction with TSD, the strongest effect shown by N382 and P718 with amplitudes reaching only 42% and 44% respectively of their original size. Peak latencies were also longer after TSD, except P718 latency, which was shorter. The entire waveform of the ERP recovered its amplitude baseline values as well as its latency values after recovery sleep, with the exception of the P718 latency, which remained shorter. Several late-task waveforms have been described within similar time intervals, and have been associated with processes related toattention, discrimination, and uncertainty of the decision made. The decrement in ERP after TSD could be due to changes in fundamental physiologic mechanisms involved in these processes.

A generic component represented by a negativity of around 250 ms for attended visual stimuli[13,14] has been associated with processes such as sensory discrimination and target selection. Lovrich et al[15] have reported an additional negative peak at N310 associated with N250, forming a complex N250/N310 for a visual form-discrimination task, which they interpreted as reflecting neural processes involved in selecting the appropriate target. A negativity lasting at least 500 ms, which has been called "processing negativity," has been associated with late processing of the attended stimuli.[31] The reduction of amplitude and the lengthening of latencies within the same time intervals observed here after TSD indicate that neural processes involved in late attending processes and in discriminating and selecting the appropriate target become inconsistent and seem to require a longer time after TSD.

The amplitude reduction observed with TSD is consistent with that previously described with low levels of alertness and fatigue. The common result from most of these studies is a progressive amplitude reduction of N1-P2 (around 100 and 175 ms) with decreasing vigilance before the onset of true sleep,[16-19] a finding well documented for the auditory[9,32] and visual systems[10-15] with ethanol[21] and in narcoleptics.[33,34] The observed prolongation of latency is in accord with findings in sleep-deprived subjects for P2 latency.[35] The latency of N310 also varies as a function of the difficulty of the task,[21,36] suggesting that the task becomes more difficult with TSD.

Sleepiness and sleep have also been associated with a negative waveform with a latency of about 350 ms. Its amplitude is reported to be negatively correlated with the level of arousal, and to increase with 24 hours of TSD.[37] This study has not confirmed the amplitude increase with TSD. Some differences between the two experiments might have influenced the results and render the comparison of results difficult. In the study by Pressman et al,[37] subjects were sitting up in bed with lights on immediately prior to a nap and were given an auditory task, whereas in this experiment, subjects were sitting in front of a computer and were given a visual task.

A slow positive wave extending at least 400 to 800 ms has been related to uncertainty and difficulty of decision processes, and its latency has been related to decision time and stimulus uncertainty—as uncertainty increases, decision takes longer time and greater effort.[38,39] The amplitude reduction observed in P718 with TSD may reflect a deterioration in the final evaluation of the decision made, and the shortening of its latency a precipitation in decision-making. These processes are associated with frontal lobe functioning. Present results agree with the increasing evidence that sleep is necessary for the restoration of the frontal cortex,[40] and indicate that 1 recovery night is not enough to restore the damage to these processes caused by 40 hours of TSD.

According to the well-established circadian oscillation in vigilance level and task performance[3] significant time-of-day effects could be expected on the ERP; however, there were no time-of-day effects or interactions between deprivation and time of day on any of the ERP variables analyzed. The highly significant correlation between ERP variables and the amount of accumulated wakefulness, and the lack of time-of-day effects and interactions between time of day and deprivation, indicate a monotonic linear deterioration. The lack of time-of-day effects was not surprising; circadian effects could be more probably expected around 0400 and 0600 hours, when body temperature reaches its minimum, and 2400, 0200, 0400, and 0600 hours were not considered in the two-way ANOVAs, since there is no equivalent control. To observe true circadian variations, a free-running paradigm is needed. On the other hand, equivalent time-of-day points from PRE-D and POST-D entered the analysis. The lack of significant diurnal variations between 1000, 1200, 1400, 1600, 1800, 2000, and 2200 hours is congruent with the lack of significant results reported previously for reaction time and EEG during performance of the task,[27] in contrast to significant diurnal variations with a peak of EEG power at 1800 hours observed in the resting EEG when the subject is doing nothing.[5] These facts reinforce our proposition that engagement in a task is enough, even with 40 hours of sleep deprivation, to mask or counteract diurnal variations observed during relaxed wakefulness.

The amplitude reduction of the visual ERP and the prolongation of latencies were linearly correlated with deteri-

oration of reaction time in a dose-response way. These results are consistent with previous findings showing that reaction time is negatively correlated with the amplitude of N1-P2[20] and positively correlated with the latencies of P2 (175 ms) and N2 (325 ms),[8,13,41,42] and suggest that alertness and performance during sleep loss are inversely proportional, probably reflecting a single underlying process or two or more processes with some determinants in common.

Waveform differences between target and nontarget stimuli are observed when one stimulus is task-relevant and of low probability. In that case there is an inverse relation between stimulus probability and waveform amplitude, particularly of P300. In the present study, no significant waveform differences between target and nontarget stimuli were found, which may be due to the type of operations demanded by the task, more related to sustained attention, discrimination, and unpredictability than to detection of infrequent stimulus as in oddball tasks. Both target and nontarget stimuli were relevant to the discriminative task, and the probability of the target stimulus was higher (25%) than the probability in typical oddball tasks. In fact, the amplitude of N1-P2 for target as well as for nontarget stimuli has been correlated with accuracy for discrimination,[10] and similar amplitudes have been observed for attended and unattended stimuli when stimuli are unpredictable.[8]

A drop in size of ERP as stimuli are repeated, even when subjects are paying attention to them, has been described in prolonged and multiple recording sessions.[39,43,44] Although results obtained in this study could have been partially influenced by habituation, this was probably not the case. The changes in the ERP were significant between predeprivation and postdeprivation day, and not between successive evaluations (lack of significant differences in time-of-day effects), and, finally, the changes observed in the ERP with TSD were similar to a dose-response curve, and the waveform recovered its baseline values after recovery sleep.

A further possible explanation for our results without invoking a concept of habituation is that there is a result of greater variability due the instability of alertness mechanisms induced by TSD as suggested by Aguirre and Broughton.[34] However, the high correlations between prestimulus EEG and the amplitude of the ERP also suggest a true reduction of the alertness mechanisms. Amplitude of the ERP was inversely correlated with AP of prestimulus EEG, indicating a preponderance of increased inhibitory processes and/or decreased excitatory processes (theta and alpha power)[45] together with an effort to maintain alertness (beta power), as we have previously reported.[27]

The efficiency of sensory, motor, and cognitive processes is determined by the degree of preparatory unspecific arousal, accurately timed to the relevant stimuli.[22] The significant relationship between prestimulus EEG and amplitude reduction of the ERP suggests that the decrement in ERP could be due to changes in fundamental neurophysiologic mechanisms. Surface-recorded potentials may be considered, as the result of the spatial average of potentials generated by the underlying neural mass and its amplitude depends on the number of cortical neurons that are relatively synchronized, excited, or inhibited at a particular moment. The reduction in amplitude with TSD may be related to changes in the thalamocortical gating mechanisms.[45]

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