

Decreased Attentional Responsivity During Sleep Deprivation: Orienting Response Latency, Amplitude, and Habituation

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Summary: Ever increasing societal demands for uninterrupted work are causing unparalleled amounts of sleep deprivation among workers. Sleep deprivation has been linked to safety problems ranging from medical misdiagnosis to industrial and vehicular accidents. Microsleeps (very brief intrusions of sleep into wakefulness) are usually cited as the cause of the performance decrements during sleep deprivation. Changes in a more basic physiological phenomenon, attentional shift, were hypothesized to be additional factors in performance declines. The current study examined the effects of 36 hours of sleep deprivation on the electrodermal-orienting response (OR), a measure of attentional shift or capture. Subjects were 71 male undergraduate students, who were divided into sleep deprivation and control (non-sleep deprivation) groups. The expected negative effects of sleep deprivation on performance were noted in increased reaction times and increased variability in the sleep-deprived group on attention-demanding cognitive tasks. OR latency was found to be significantly delayed after sleep deprivation, OR amplitude was significantly decreased, and habituation of the OR was significantly faster during sleep deprivation. These findings indicate impaired attention, the first revealing slowed shift of attention to novel stimuli, the second indicating decreased attentional allocation to stimuli, and the third revealing more rapid loss of attention to repeated stimuli. These phenomena may be factors in the impaired cognitive performance seen during sleep deprivation. **Key Words:** Sleep deprivation—Orienting response—Attention—Habituation.

Sleep deprivation has been shown to impact negatively on a wide range of cognitive, behavioral, physiological, and emotional measures (1-5). Given the increasing reliance of the armed forces, medical providers, business, and industry on sleep-deprived workers, these effects pose a serious safety threat, both to the workers and to the population as a whole.

The primary theory that attempts to explain the effects of sleep deprivation on performance is the "lapse hypothesis" proposed by the Walter Reed group (6). This theory regards decrements in performance due to sleep deprivation as being caused by brief periods of unresponsiveness that increase in frequency as a function of hours of deprivation. These authors also postulate that lapses are caused by lowered arousal levels. Kjellberg (3) elaborated this concept by defining lapses and decreased arousal more specifically, adding that, as arousal decreases gradually and falls below a

certain level, microsleeps (brief intrusions of sleep into wakefulness) occur. These microsleeps constitute the physiological basis for the performance lapses noted by others (1,6,7). The behavioral effects of sleep deprivation are congruent with the de-arousal theories in that: (1) if performance is affected, it is generally impaired; (2) the effect on performance is gradual and uneven; and (3) performance decrements can be counteracted by arousing/aversive stimulation, such as shocks or noise, and by motivation/incentive (8-12).

Most sleep-deprivation studies that have employed electroencephalogram (EEG) measures have found decreased alpha-rhythm abundance or decreased alpha-wave amplitude as a result of sleep deprivation (cf. 3,13,14). These EEG changes have generally been interpreted as indicating decreased arousal due to sleep loss, which supports the de-arousal theories (3). Most studies of the effects of sleep deprivation have focused on task performance, decrements in which have been theorized to be caused by underlying perceptual or cognitive changes due to sleep loss and, a few have postulated, changes in attentional response to novel stimuli or habituation of attentional response to repeated stimuli (5,15). However, although performance

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decrements and orienting-response (OR) habituation have been theoretically linked (cf. 16,17), empirical demonstrations of sleep-deprivation-induced changes in the physiological indices of attentional shift or capture, such as the OR, have not been made. In addition, it has been hypothesized that sleep deprivation might not affect the physiological indices themselves (e.g. OR habituation rate) but might instead magnify the degree of de-arousal caused by habituation (3).

The organismic OR is a complex of systemic physiological reactions occurring reflexively to new environmental stimuli and reflecting a shift in attention to, or capture of attention by, such stimuli (18–20). One of the most widely used measures of the OR is the electrodermal response (skin-conductance response). The electrodermal response is a stable and robust indicator of the OR that is easily measured, although the functional link of electrodermal activity to the organism's attentional orienting is not obvious (21).

An important characteristic of the OR is its tendency to habituate (decline to zero in strength) over repeated unsequenced stimulus presentations and OR elicitation. OR-habituation rate is governed by the same variables that govern the elicitation of the OR (i.e. novelty, complexity, intensity), and it occurs most rapidly to neutral, non-reinforced, or non-salient stimuli (22,23). After habituation to a particular stimulus, the OR can again be elicited by a change in the frequency, intensity, or duration of that stimulus or by a change in stimulus modality (24). In addition, one interpolated presentation of a different stimulus can lead to the re-emergence of the OR to an originally habituated stimulus when it is presented again, dishabituation (20).

Although the question of OR changes during sleep deprivation has not previously been addressed directly, McDonald, Johnson, and Hord (25) did conduct two experiments examining the OR during drowsiness. These researchers found that normal subjects often showed signs of drowsiness during OR trials when EEG was measured (19 of 30 subjects in Experiment 1, 32 of 69 subjects in Experiment 2). "Drowsy" subjects exhibited increased 4–7-Hz (theta) wave activity and sometimes sleep spindles in the EEG. In both studies, electrodermal response to the habituation stimuli decreased similarly over trials for the drowsy and alert subjects, but alpha EEG desynchronized (an indication of the OR) when the habituation stimuli were presented to alert subjects and alpha EEG amplitude increased upon presentation of a stimulus to drowsy subjects. In fact, the alpha desynchronization response habituated over trials in alert subjects, while the alpha enhancement response increased over trials in drowsy subjects. Although it is not precisely clear what these differences mean, it is clear that the organismic OR is different during drowsiness. This supposition has re-

ceived somewhat indirect support from studies examining changes in event-related potentials (ERP, averaged evoked potentials correlating with attentional shift) resulting from sleep deprivation. However, due to the confusing nature of the sleep-deprivation ERP literature (differing stimuli, methods, and conditions across studies), few clear conclusions about attentional mechanism changes due to drowsiness/lack of sleep can be drawn (see e.g. 26,27).

Though the McDonald, Johnson, and Hord (25) study touches on some of the issues proposed for study in the current research, the lack of controlled sleep deprivation limits the generalizability of the findings to the effects of sleep deprivation on OR. Further, the possibility of pre-existing sleep disorders and of different amounts of sleep debt that might have affected orienting were not controlled in their subjects (28). Therefore, other factors may have differentiated the two groups in addition to tonic arousal state.

The current study examined the effect of 36 hours of sleep deprivation on orienting-response elicitation and habituation, with concurrent assessment of cognitive performance. Baseline sleep was monitored through self-report, and assignment to sleep deprivation and control groups was randomized. In addition, subjects were screened prior to participation to eliminate those with sleep disorders and irregular sleep/wake schedules.

METHOD

Subjects

Subjects were male undergraduates drawn from the subject pool of students enrolled in psychology courses at Louisiana State University. Only male subjects were eligible to participate because recent studies have shown that females are more likely to be electrodermally unresponsive and that female electrodermal responses differ during various phases of the menstrual cycle (29,30). One hundred ten subjects participated in initial screening. Of those, 22 were not invited to participate in the study due to medical or psychological conditions, sleep disorders, irregular sleep-wake schedules, or electrodermal non-response. Seventeen additional subjects did not appear at their assigned testing date, leaving a final subject number of 71.

Experimental design

A Solomon four-group design was employed in this research (31). Subjects were randomly assigned to one of two primary conditions: sleep deprivation (SD, experimental group) and non-sleep deprivation (NSD, control group). Control subjects were selected from

among individuals who had already agreed to participate in a sleep-deprivation experiment, but had not yet done so, to ensure that subjects in the two conditions did not differ in their willingness to undergo sleep deprivation.

The experimental and control groups were each further randomly subdivided into two subgroups. One subgroup of each primary group was OR tested only after the sleep deprivation/control period (time 2 only, T2). The other subgroup of each primary group was OR tested both before (time 1, T1) and after (T2) the experimental night. This within-subjects component was included to control for the possibility that high between-subjects orienting-response variability might obscure experimental effects.

Apparatus

EEG, electrooculograph (EOG), and electromyograph (EMG) measurements were recorded using a Puritan-Bennett/Biologic Sleepscan Premier computerized sleep-data collection and analysis system (polysomnograph). EEG, EMG, and EOG recordings were made using Grass Instruments silver/silver chloride cup electrodes (8-mm diam.).

Skin conductance was measured using Med Associates silver/silver chloride electrodes (20-mm diam.) on a Grass Model 7D polygraph (7P122 amplifier/pre-amplifier). Silver-silver chloride electrodes were used because they minimize the development of bias potentials and polarization. Skin-conductance response (SCR) electrodes were attached using double-sided adhesive collars to the palmar surface of the distal phalange of the middle finger and ventral forearm of subjects' non-dominant hand.

Procedure

Sleep deprivation group subjects ($n = 35$) underwent a total of 36 hours of sleep deprivation. During part of that time (the last 20 hours) they were housed at the testing site to ensure their compliance with the sleep deprivation procedures. During this 20 hours, subjects were in the company of experimenters and other subjects at all times. Control subjects ($n = 36$) were asked to approximate an "average" night's sleep on the experimental night. Average amount of sleep obtained by control group members during the experimental night was 7.59 hours, which did not differ significantly from the average amount of sleep obtained by all subjects across their 2-week pre-experimental monitoring (sleep diary) period. The control subjects were also asked to abstain from alcohol, caffeine, and tobacco during the 36 hours preceding testing so that

their physiological state would be more like that of the experimental group.

During OR testing, each subject received an abbreviated version ($C_2, C_3, A_1, A_2, F_3, F_z$, right outer canthus [ROC]) of the standard sleep-electrode placement (32) that was used to assess for micro-sleeps during OR testing in sleep-deprived subjects. After electrode attachment, subjects sat alone in a 15' \times 15' sound-attenuated, electronically shielded room. Psychophysiological recording equipment was located in an adjacent room. Subjects were given a 5-minute adaptation period while the equipment was being adjusted and calibrated. This was followed by recording of 2 minutes of baseline data. Subjects then heard a series of identical tape recorded tones (1 second duration, 600 Hz). Tones were presented at 55 dB to maximize the probability of eliciting orienting responses rather than defensive responses (33). Tones were presented with an average interstimulus interval (ISI) of 20 seconds (15, 20, and 25 second ISIs). The tone series was discontinued after two consecutive tones were presented without an orienting response to avoid over-habituation which might affect responses to later tone presentations (34). If responses did not habituate before trial 10, the tone series was discontinued at that time. Twenty seconds after the last tone, there was a dishabituating stimulus (DHS) (900 Hz, 55 dB), a tone different from the previous tones, intended to re-instate the OR. Twenty seconds after the dishabituating stimulus, the original tone was presented again (post-dishabituating stimulus [P-DHS]) to test for dishabituation (the reinstatement of the OR to the previously habituated stimulus). This tone was followed by a 2-minute silent interval and one additional presentation of the original tone to assess for spontaneous recovery of the OR [spontaneous recovery stimulus (SRS)]. This physiological testing procedure was performed at T1 and T2 for pre- and post-tested subjects and at T2 only for those subjects tested only after the sleep deprivation/control period.

Orienting responses were defined as an increase in skin conductance from a pre-response baseline of at least 0.05 μ Siemens (μ S) (measured baseline to peak) occurring 1–5 seconds after onset of the stimulus. Though some authors (35,36) have recently advocated the use of more restrictive latencies (i.e. 1–3 seconds following stimulus onset), it was not certain what effect sleep deprivation would have on OR latency, and as a longer latency was hypothesized, the traditional 1–5-second window was employed.

Habituation was defined as two consecutive tone presentations without OR, at which time the tone stimulus series was discontinued. Some authors (35,36) have suggested that reduction of the habituation criterion from three non-responses to two non-responses

achieves the goal of minimizing contamination by spontaneous activity in a similar fashion to the narrowing of the OR scoring window.

Approximately 21 days passed between T1 and T2. During this time, subjects were contacted once to remind them of their date of participation. Subjects were also called twice on the morning of their sleep-deprivation period to ensure that they were awake as scheduled. Sleep-deprivation subjects then reported to the testing site after the first 16 hours of their 36-hour deprivation period had passed. They spent the remainder of their deprivation period with the experimenters. Control subjects reported to the testing site at the end of the 36-hour control period. Control and experimental subjects were tested at the same time.

Right after the T2 OR testing, subjects were asked to perform a portion of the Army Neurological Assessment Measures test battery (ANAM; Stroop task, memory search, mental arithmetic, and logical reasoning) (37). Data for a vigilance-tracking task was also collected; however, due to computer malfunction, it was not retrievable and, therefore, cannot be discussed in this report. The ANAM tasks were administered to assess performance during sleep deprivation, thus permitting comment on the relation of sleep-deprivation effects on performance to any OR elicitation and habituation changes found during sleep deprivation. Circadian fluctuations in OR measures and cognitive performance were anticipated by keeping assessment times constant throughout the study, across groups and across testing sessions. T1 and T2 testing sessions were always held between 6:00 p.m. and 8:00 p.m., and subjects in both sleep-deprivation and control conditions were tested together. Subjects were released from the study immediately following their completion of testing and debriefing.

RESULTS

Several preliminary analyses were performed to evaluate pre-experimental group equivalence. Experimental and control groups were not found to differ significantly on any examined measure—SCR amplitude to the first stimulus presented during physiological screening prior to T1 (tap on door), race, age, or self-report sleep-diary data.

No sleep-deprived subjects evidenced microsleeps during collection of T2 OR data. Subjects were seated with eyes open at the time of stimulus presentation, and all subjects displayed continuously fast, desynchronous, and low-amplitude EEG with infrequent alpha. Fifteen-second epochs were visually scored, and slow rolling eye movements, vertex spikes, EMG decrements, spindles, and K-complexes were absent during the presentation of OR stimuli in all subjects but

one, who exhibited a vertex spike 90 seconds prior to the presentation of the first stimulus. However, signs of sleep onset were absent thereafter, and only fast, desynchronous low-amplitude EEG was seen at the time of stimulus presentation. Therefore, data from all subjects was retained.

Within-subjects analyses

T1 to T2 changes in pre-tested SD and NSD subjects

Within-subjects analyses were planned prior to the study as a means of assessing intra-individual changes in OR and habituation during sleep deprivation versus normal sleep in the eventuality that variability was so great as to obscure between-subjects effects. Because this great variability did not occur, results of within-subjects analyses are discussed only briefly.

The following variables were examined by repeated measures ANOVAs with testing session (T1, T2) as a within-subjects variable and group assignment (sleep deprived, SD; non-sleep deprived, NSD) as a between-subjects variable: response latency and amplitude to the initial stimulus, mean response latency and amplitude to all habituation trials, response amplitude to the DHS, response amplitude to the P-DHS, and response latency and amplitude to the SRS. (Response latencies to DHS and P-DHS were not analyzed due to the large numbers of non-responders at T2, leaving too little power to conduct the ANOVAs.) Of the analyzed variables, seven of eight were found to violate the homogeneity of variance assumption of the ANOVA; as such, data analyses were performed on square-root-transformed data. SRS-latency data did not need to be transformed. Trials to habituation was also analyzed by within-subjects ANOVA. Because between-groups information is discussed in the between-subjects section, only within-subjects data will be described below. No significant main effects of pre-testing or interactions were evidenced in any test involving response amplitude. Main effects of pre-testing and interactions were also absent in analysis of latency to SRS and latency to the initial stimulus. An interaction of sleep status and testing session was significant in the analysis of mean latency to all habituation stimuli, although none of the paired comparisons emerged as significant in post hoc testing. A pronounced effect of pre-testing, however, was the decreased number of responses by subjects at T2, especially for the DHS and P-DHS. The within-subjects examination of trials to habituation revealed decreased trials to habituation as a result of pre-testing [$F(1,16) = 6.65$; $p < 0.02$; T1 mean = 5.44 (SD = 3.35); T2 mean = 3.55 (SD = 3.05)].

TABLE 1. Raw score means and standard deviations, homogeneity, and *t* test results for cognitive tasks

Task	Measure	SD ^a mean (SD)	NSD ^b mean (SD)	Homogeneity <i>F</i> (p) <i>t</i> (p) one-tailed
Stroop task 1	% Corr. ^c	97.59 (11.17)	98.39 (3.84)	<i>F</i> (1,63) = 8.45, <i>p</i> < 0.001 ns
	RTC ^d	1,119.49 (1,500.12)	1,382.78 (2,211.99)	<i>F</i> (1,63) = 2.17, <i>p</i> < 0.02 ns
Stroop task 2	% Corr.	97.96 (4.16)	98.39 (2.91)	<i>F</i> (1,63) = 2.05, <i>p</i> < 0.05 ns
	RTC	720.56 (139.36)	685.84 (157.32)	ns ns
Stroop task 3	% Corr.	90.93 (17.90)	93.56 (9.83)	<i>F</i> (1,63) = 3.31, <i>p</i> < 0.001 ns
	RTC	1,141.92 (369.01)	1,142.59 (455.82)	ns ns
Memory	% Corr.	85.21 (14.96)	89.91 (12.05)	ns ns
	RTC	998.80 (229.70)	905.81 (292.14)	ns 1.61 (<i>p</i> < 0.05)
Arithmetic	% Corr.	91.64 (16.02)	93.61 (4.87)	ns ns
	RTC	2,609.22 (869.47)	2,553.74 (193.03)	ns ns
Grammatical	% Corr.	52.47 (12.94)	55.09 (8.54)	<i>F</i> (1,61) = 2.30, <i>p</i> < 0.01 ns
	RTC	6,873.70 (1,634.85)	5,992.90 (1,336.39)	ns 2.29 (<i>p</i> < 0.01)

^a SD = sleep-deprived subjects.

^b NDS = non-sleep-deprived subjects.

^c % Corr. = percent correct.

^d RTC = reaction time of correct responses in milliseconds.

Between-subjects analyses

Sleep deprivation and pre-testing effects on T2

Cognitive measures. All cognitive-task data were analyzed using Student's *t* tests. Homogeneity-of-variance tests indicated that for many of the cognitive tasks, the variances of the samples were significantly heterogeneous; therefore, *t* tests were performed on data subjected to square-root transformation. *F* ratios for homogeneity tests, raw score means and standard deviations, and results of *t* tests for cognitive tasks are provided in Table 1. The sleep-deprived and control subjects differed significantly in the following ways: (1) groups displayed significantly different variability on five measures: reaction time to initial Stroop task, accuracy on all three Stroop tasks, and accuracy on the grammatical reasoning task. In all but one of these analyses, the sleep-deprived group was more variable. (2) Sleep-deprived subjects responded more slowly on the grammatical reasoning and memory search tasks, and a non-significant trend ($t = 1.35$, $p < .09$) toward less accurate responding by sleep deprived subjects was noted on the memory search task.

Physiological measures (electrodermal orienting response). Due to the violation of the homogeneity-of-

TABLE 2. Means and standard deviations for OR latency to all stimuli (in seconds)

Stimulus	Group	Raw score mean (SD)	<i>F</i> (p)
Initial	SD	1.35 (0.54)	<i>F</i> (1,55) = 3.33, <i>p</i> < 0.04
	NDS	1.07 (0.36)	
	Pre/post ^a	1.13 (0.36)	
	Post	1.28 (0.55)	
All habituation trials ^b	SD	2.65 (3.07)	<i>F</i> (1,64) = 3.24, <i>p</i> < 0.04
	NSD	1.71 (2.16)	
	Pre/post	2.28 (2.92)	
	Post	2.12 (2.49)	
DHS	SD	1.59 (0.72)	<i>F</i> (1,35) = 8.00, <i>p</i> < 0.004
	NSD	1.00 (0.31)	
	Pre/post	1.28 (0.62)	
P-DHS	SD	1.69 (1.46)	ns
	NSD	1.15 (0.53)	
	Pre/post	1.38 (0.66)	
SRS	SD	0.42 (1.33)	ns
	NSD	1.19 (3.97)	
	Pre/post	1.20 (4.13)	
	SD	0.41 (0.71)	ns

^a Pre/post refers to subjects tested at T1 and at T2; Post refers to subjects tested only at T2.

^b Signifies significant interaction, $F(1,64) = 5.63$, $p < 0.02$.

variance assumption of the ANOVA by large differences in variances between groups, all amplitude and latency analyses were conducted using square-root-transformed data. However, raw score means are reported to aid in interpretation of the data.

OR latency. Orienting-response (OR) latency to initial stimulus at post-test (T2) was subjected to a 2 (number of testing sessions) \times 2 (sleep status) analysis of variance (ANOVA). A main effect of sleep-deprivation status was evidenced, $F(1,55) = 3.33$, $p < 0.04$. Sleep deprived (SD) subjects took significantly longer to orient to the initial stimulus than did NSD subjects (SD mean = 1.35 seconds, NSD mean = 1.07 seconds). No other statistically significant main effect or interaction was found. All means and standard deviations for latency to T2 stimuli are presented in Table 2.

Mean OR latency to all T2 habituation stimuli (initial stimulus plus all stimuli presented before the dishabituating stimulus, i.e. all habituation trials) was tested in a 2 (number of testing sessions) \times 2 (sleep status) ANOVA. A main effect of sleep deprivation emerged, $F(1,64) = 3.24$, $p < 0.04$. Sleep-deprived subjects were significantly slower in orienting to stimuli than were subjects who had not undergone sleep deprivation (SD mean = 2.65 seconds; NSD mean = 1.71 seconds). Though there was no main effect of pretesting, a significant interaction also was found, $F(1,64) = 5.63$, $p < 0.02$: Post hoc testing (Tukey hsd) revealed that pre-tested sleep-deprived subjects had significantly longer mean T2 latencies than did pre-

tested non-sleep-deprived subjects. Mean OR latency to T2 stimuli of sleep-deprived and non-sleep-deprived subjects who were tested only at post-test did not differ significantly from each other.

A 2 (number of testing sessions) \times 2 (sleep status) ANOVA also was used to examine latency of orienting responses to the DHS. A significant main effect of sleep deprivation on DHS-response latency was found, $F(1,35) = 8.00$, $p < 0.004$. OR was significantly delayed in sleep-deprived subjects as compared to non-deprived subjects (SD mean = 1.59 seconds, NSD mean = 1.00 seconds). There was no main effect of pretesting nor was there a significant interaction of sleep status and pre-testing.

Examination by 2 \times 2 ANOVAs revealed that neither OR latency to the P-DHS nor OR latency to the SRS differed by sleep status or number of testing sessions. Statistically significant interactions were also absent.

OR amplitude. Orienting-response amplitude to the initial stimulus at post-test was subjected to a 2 (number of testing sessions) \times 2 (sleep status) analysis of variance (ANOVA). A main effect of sleep-deprivation status was evidenced, $F(1,62) = 2.94$, $p < 0.05$, with sleep-deprived subjects having lower-amplitude responses than the non-deprived subjects (SD mean = $0.51 \mu\text{S}$; NSD mean = $2.17 \mu\text{S}$). A main effect for pre-testing was not found nor was the interaction significant. Means and standard deviations for OR amplitude to all post-test stimuli are presented in Table 3.

A 2 (number of testing sessions) \times 2 (sleep status) ANOVA also was used to examine amplitude of orienting responses to the P-DHS. Both sleep status, $F(1,64) = 3.21$, $p < 0.04$, and number of testing sessions, $F(1,64) = 7.43$, $p < 0.01$, emerged as significant main effects. Sleep-deprived subjects exhibited lower-amplitude responses to the P-DHS than non-deprived subjects (SD mean = $0.10 \mu\text{S}$; NSD mean = $0.21 \mu\text{S}$). The T2 responses of pre-tested subjects were also of lower amplitude than those of subjects tested only at T2 (pre-tested mean = $0.05 \mu\text{S}$; post-test only mean = $0.24 \mu\text{S}$). The interaction term was not significant.

The following additional variables were examined using 2 (number of testing sessions) \times 2 (sleep status) ANOVAs: OR amplitude to the dishabituating stimulus, OR amplitude to the spontaneous recovery stimulus, and mean OR amplitude to all habituation (pre-DHS) stimuli. Significant main effects or interactions were absent in all of these analyses.

Trials to habituation. The effects of pre-testing and sleep deprivation on the number of trials to habituation was tested by 2 \times 2 ANOVA. Significant main effects of sleep deprivation [$F(1,65) = 4.00$, $p < 0.05$] and number of testing sessions [$F(1,65) = 4.74$, $p < 0.03$] were evidenced. Sleep-deprived subjects took signifi-

cantly fewer trials to habituate than did non-sleep-deprived subjects [SD mean = 3.65 (3.03); NSD mean = 5.09 (3.75)], and pre-tested subjects habituated significantly more rapidly than subjects tested only at post-test [T1/T2 mean = 3.55 (3.05); T2 only mean = 5.15 (3.69)]. No significant interaction was found.

DISCUSSION

Results of the current investigation revealed significant effects of sleep deprivation on both cognitive and physiological measures. In general, sleep deprivation was found to: (1) decrease electrodermal orienting-response amplitude to an initial and a post-dishabituating stimulus, (2) delay the electrodermal orienting response to an initial and a dishabituating stimulus, and (3) decrease the number of trials necessary for the OR to habituate to a novel stimulus. These findings were demonstrated within the context of increased performance variability on cognitive tasks after sleep deprivation (reaction times to the initial Stroop task, accuracy on all three Stroop tasks, and the grammatical reasoning task) and increased reaction time on two cognitive tasks (grammatical reasoning and memory search) following sleep loss. Taken together, these findings indicate that sleep deprivation decreased subjects' attentional responsivity to new information and simultaneously reduced the efficiency of their cognitive processing. Evidence of microsleeps was absent at the end of the sleep-deprivation period when OR performance variables were assessed. Therefore, it is believed that subjects remained in a state of wakefulness throughout orienting-response testing and that the performance decrements were due to the changes in primary attentional variables, rather than being secondary to microsleeps. One plausible explanation for the absence of microsleeps might be that the impending end of sleep deprivation, signified by the post-testing situation, may have motivated subjects to fight drowsiness and de-arousal and, thus, may have prevented microsleeps.

Cognitive tasks were employed in this study to document declines in ability that typically result from sleep deprivation. The finding of increased variability among sleep-deprived subjects (documented by significantly heterogeneous variances in the two groups) across several tasks, and the reaction time differences on memory search and grammatical-reasoning tasks indicates that the sleep-deprivation period significantly affected cognitive performance in the usual manner (38). This provided the framework within which the orienting response results were interpreted.

The extended OR latency found in sleep-deprived subjects may be considered similar to the increased latencies of several event-related potential (ERP) com-

TABLE 3. Means and standard deviations of OR amplitude to all stimuli (in μS)

Stimulus	Variable	Group	Raw score mean (SD)	Significance
Initial	Sleep status	SD	0.51 (0.78)	$F(1,62) = 2.94, p < 0.05$
		NSD	2.17 (6.70)	
	Sessions	Pre/post ^a	1.94 (6.72)	
		Post	0.72 (1.00)	
All habituation trials	Sleep status	SD	0.86 (2.97)	ns
		NSD	1.25 (3.34)	
	Sessions	Pre/post	1.65 (4.40)	
		Post	0.48 (0.73)	
DHS	Sleep status	SD	0.61 (2.51)	ns
		NSD	1.36 (5.29)	
	Sessions	Pre/post	2.28 (2.92)	
		Post	2.12 (2.49)	
P-DHS	Sleep status	SD	0.04 (0.09)	$F(1,64) = 3.21, p < 0.04$
		NSD	0.22 (0.84)	
	Sessions	Pre/post	0.05 (0.06)	
		Post	0.24 (0.82)	
SRS	Sleep status	SD	1.69 (1.55)	ns
		NSD	1.40 (1.03)	
	Sessions	Pre/post	1.26 (0.37)	
		Post	1.83 (1.79)	

^a Pre/post refers to subjects tested at T1 and at T2; Post refers to subjects tested only at T2.

ponents noted in sleep-deprived subjects in other studies (26,27), and the increased ERP component latencies noted in pathologically sleepy subjects (e.g. 39). It is of note that the ERP studies found significant differences only late in a 48-hour period of sleep deprivation, whereas the present study was able to document increases in latency following only 36 hours of sleep deprivation.

In the present study, both OR latency to the initial stimulus and OR latency to the DHS were found to be increased by sleep deprivation. Both of these stimuli represent the initial presentation of a novel stimulus. Latency differences between groups did not emerge to stimuli that were re-introduced into the environment (P-DHS, SRS). Although this may be interpreted as limiting the sleep-deprivation effect to novel stimuli, the very small number of subjects responding at all to re-introduced stimuli suggest that dishabituation and short-term spontaneous recovery may be phenomena that are themselves too fragile to reveal the effects of other conditions or processes such as sleep deprivation.

The other significant OR latency finding, an interaction of sleep status and testing session on mean OR latency across all habituation trials, indicates further attentional impairment in sleep-deprived subjects. In this interaction, only the paired comparison between sleep-deprived and non-sleep-deprived subjects tested at T1 showed significant differences in the OR latency across habituation trials at T2. In effect, sleep deprivation increased OR latency across all habituation trials only if there was a prior testing (T1). This may

have been due to a summation of the pre-testing and sleep-deprivation effects; pre-test habituation decreases latency for all subjects and post-test habituation further decreases latency, except in sleep deprived subjects, where the further decrease in latency is countered by the latency increasing effect of sleep deprivation. In subjects who were tested only at T2, the initial habituation effect, a decrease in latency was so strong that sleep deprivation could not exert a statistically significant inhibitory (latency increasing) effect. Together, these findings suggest that sleep-deprived individuals indeed may shift attention more slowly to stimuli that they have previously encountered, although the effect is more complex than for initial stimuli.

Orienting-response amplitude also was found to be decreased as a function of sleep deprivation, further indicating decreased responsiveness to the stimuli. This is contrary to the prediction of increased OR amplitudes after sleep deprivation by theories that hypothesize: (1) that the aversive/arousing nature of sleep deprivation would lead to increased OR amplitudes (e.g. 40,41) or (2) that sleep deprivation leads to drowsiness/dearousal, less OR habituation and, therefore, greater OR amplitudes (42).

The number of trials needed for habituation of the OR was found in the current study to be affected by both sleep status and testing session. The increase in habituation rate due to pre-testing is a standard finding in habituation research. However, the further increase in habituation rate for sleep-deprived subjects is novel, and is not consistent with previous research on drowsy

subjects, which showed longer habituation times, presumably due to drowsiness-induced de-arousal (42). Nonetheless, the more rapid habituation rate noted in the sleep deprived subjects actually is more consistent with the decreased attention and cognitive performance empirically observed in this and other studies and, with the lack of interest in tasks often noted anecdotally in those who have experienced sleep loss, because rapid OR habituation signifies reduced attention. These findings, like the decreased latency and decreased amplitude findings, help to explain further the decreased accuracy evidenced by sleep-deprived subjects on attention-demanding cognitive tasks. Reaction time and accuracy could very easily be compromised if subjects take too long to attend to and process a critical stimulus, and could be further impaired if attention to a critical stimulus decreases too soon after too few presentations.

SUMMARY AND CONCLUSIONS

The current research revealed that 36 hours of sleep deprivation increased OR latency to initially presented stimuli, decreased OR amplitude to stimuli, and decreased the number of trials necessary for the OR to habituate to a stimulus, all in the context of increased variability and reaction times on attention-demanding cognitive tasks. When the implications of these findings are examined as a whole, they indicate that sleep-deprived individuals are slower to attend to relevant environmental stimuli, exhibit less response to the stimuli, lose interest in stimuli more rapidly, and are slower and more variable in their processing of stimuli. The impact of these findings is enhanced notably by EEG data that indicated that microsleeps did not occur during OR stimulus presentation after sleep deprivation and, thus, were not an apparent cause of the observed OR phenomenon. These results show that a more basic psychophysiological phenomenon must be taken into account when the role of sleep deprivation on performance is discussed. Whereas countering the effects of sleep loss is often viewed as simply a matter of delaying sleep onset and intrusive microsleeps, the present data suggest that basic attentional mechanisms also are independently compromised by sleep deprivation. The alteration of this underlying attentional mechanism must be addressed in studies aimed at understanding the deleterious effects of sleep loss and in the studies attempting to correct them, lest the maintenance of wakefulness has suboptimal effect.

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