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Cumulative Sleepiness, Mood Disturbance, and Psychomotor Vigilance Performance Decrements During a Week of Sleep Restricted to 4–5 Hours per Night

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Summary: To determine whether a cumulative sleep debt (in a range commonly experienced) would result in cumulative changes in measures of waking neurobehavioral alertness, 16 healthy young adults had their sleep restricted 33% below habitual sleep duration, to an average 4.98 hours per night [standard deviation (SD) = 0.57] for seven consecutive nights. Subjects slept in the laboratory, and sleep and waking were monitored by staff and actigraphy. Three times each day (1000, 1600, and 2200 hours) subjects were assessed for subjective sleepiness (SSS) and mood (POMS) and were evaluated on a brief performance battery that included psychomotor vigilance (PVT), probed memory (PRM), and serial-addition testing. Once each day they completed a series of visual analog scales (VAS) and reported sleepiness and somatic and cognitive/emotional problems. Sleep restriction resulted in statistically robust cumulative effects on waking functions. SSS ratings, subscale scores for fatigue, confusion, tension, and total mood disturbance from the POMS and VAS ratings of mental exhaustion and stress were elevated across days of restricted sleep ($p = 0.009$ to $p = 0.0001$). PVT performance parameters, including the frequency and duration of lapses, were also significantly increased by restriction ($p = 0.018$ to $p = 0.0001$). Significant time-of-day effects were evident in SSS and PVT data, but time-of-day did not interact with the effects of sleep restriction across days. The temporal profiles of cumulative changes in neurobehavioral measures of alertness as a function of sleep restriction were generally consistent. Subjective changes tended to precede performance changes by 1 day, but overall changes in both classes of measure were greatest during the first 2 days (P1, P2) and last 2 days (P6, P7) of sleep restriction. Data from subsets of subjects also showed: 1) that significant decreases in the MSLT occurred during sleep restriction, 2) that the elevated sleepiness and performance deficits continued beyond day 7 of restriction, and 3) that recovery from these deficits appeared to require two full nights of sleep. The cumulative increase in performance lapses across days of sleep restriction correlated closely with MSLT results ($r = -0.95$) from an earlier comparable experiment by Carskadon and Dement (1). These findings suggest that cumulative nocturnal sleep debt had a dynamic and escalating analog in cumulative daytime sleepiness and that asymptotic or steady-state sleepiness was not achieved in response to sleep restriction. **Key Words:** Sleep restriction—performance—cumulative sleepiness—partial sleep deprivation.

A fundamental theoretical and practical question in human sleep research concerns the quantity of nightly sleep that will lead to accumulation of daytime sleepiness and waking neurobehavioral deficits (i.e. the waking analog of cumulative sleep debt). Although it is generally the case that the less sleep obtained each night the greater the likelihood of cumulative waking deficits, it remains contentious as to what level of sustained sleep restriction leads to daytime sleepiness and measurable performance deficits (2–5).

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At one end of the continuum, namely restriction of sleep duration to between 0 and 4 hours, decrements in waking alertness are often observed. Cumulative increases in subjective sleepiness and performance deficits are consistently found during 2–5 days of total sleep deprivation (6–8). Experimental restriction of sleep to ≥ 1 but ≤ 4 hours for even a single night also has been reported to lead to reduced daytime sleep latencies (9,10) and to increased subjective sleepiness and performance deficits (5,11–17). More importantly, these effects for sleep limited to ≤ 4 hours/night have been reported to be cumulative—increasing across two to eight consecutive days of restriction (5,11,14,15,18).

In contrast, studies of sleep restricted to a range more commonly experienced by people (i.e. >4.5 but <6.5

hours/night) have produced inconsistent results. While some investigations have found that even a single night of sleep restricted to this range has been associated with daytime sleepiness and performance decrements (10,19,20), results from other studies in this range of restriction have been negative (5,14,21). Similarly, although cumulative increases in sleepiness have been reported (1,13,22), these cumulative effects have not been found in longer duration studies (5,23). The lack of evidence for cumulative waking deficits in alertness (especially performance deficits) following restriction of sleep to approximately 5 hours per night has led some to conclude that sleep limited to this range does not result in cumulative decreases in performance (3–6,24).

However, given the small number of studies of partial sleep deprivation (PSD) reported to date, the question of whether daytime sleepiness and waking neurobehavioral deficits "accumulate" when sleep is restricted to 60–80% (i.e. 5–6 hours) of typical durations remains unanswered. There are a number of factors that may have contributed to the disparate outcomes among studies of waking performance after sleep was confined to >4.5 hours but <6.5 hours/night. Particularly noteworthy is the fact that many of the studies that involved performance assessments across multiple days reported robust practice effects (11,14,22,23,25–27). These learning curves confound the measurement of "cumulative" performance deficits from sleep restriction and, therefore, compromise the validity of conclusions about the lack of such effects. The addition of a "practice control group" does not necessarily undo this confounding, especially if the interaction between practice and sleep loss effects is nonlinear over time (5). In other words, cumulative performance deficits may have been occurring, but they were obscured by the countervening learning curves; subtracting out the learning curve of a control group does not ensure that the residual is a product (or lack thereof) of sleep restriction. This conclusion is supported by the fact that many of the longer term studies (i.e. sleep restricted for weeks to months) that found no cumulative effects on daytime performance from sleep restricted to 4.5–6.5 hours/night did, however, observe increases in subjective complaints of headache, forgetfulness, reduced concentration, fatigue, irritability, and difficulty awakening (5,21,23,26). While such subjective data were often gathered anecdotally, the data suggest that the performance tests used in these studies were not detecting some or all of the neurobehavioral problems being experienced by subjects. This could have been the case if the sleepiness being experienced by subjects was less than that obtained with total sleep deprivation, which was the context in which the tests were validated.

Finally, as Carskadon and Roth (2) observed, many of the sleep restriction studies conducted to date are methodologically limited by small sample sizes, a narrow focus on only a few classes of neurobehavioral outcomes, lack of experimental control over sleep obtained per 24 hours, allowing subjects to use stimulants (e.g. caffeine), infrequent assessment times within and between days, and a failure to quantitatively test for cumulative neurobehavioral changes relative to the cumulative sleep debts subjects experienced. To test the hypothesis that cumulative neurobehavioral deficits occur across days in response to cumulative sleep debt, we performed a study that restricted sleep to an average of approximately 5 hours/night for seven consecutive nights that included within- and between-day measurements on a range of neurobehavioral outcomes (i.e. subjective states, performance dimensions, and sleep latency). Specifically, we sought to establish the presence and temporal profile (i.e. onset, growth, magnitude, and asymptote) of changes in both daytime performance and subjective states across days of sleep restriction and their relationship to diurnal changes.

METHODS

Subjects

A total of 20 young adult subjects were recruited for the study. Two volunteers were excluded for high basal levels of sleepiness, and two failed to complete the protocol. The remaining 16 healthy young (mean age, 22.9 years) adult subjects (eight females and eight males) volunteered to have their nightly sleep limited for 1 week to "50% of their ideal sleep duration". The latter was obtained through interviews with subjects and in all cases exceeded their reported habitual sleep duration. Subjects served as their own controls.

Procedure

Sleep

Volunteers were interviewed to confirm normal sleep–wake cycles. Those eligible then kept daily sleep logs, and some wore wrist actigraphs for a period of 3–7 days prior to the in-lab protocol to confirm stable, habitual times for nocturnal sleep and morning awakening. Following this ambulatory phase, subjects had a polysomnogram (PSG) screen to ensure they had no sleep disorders and a multiple sleep latency test (MSLT) to ensure their daytime sleep propensities were between 8 and 20 minutes. Subjects then slept in the General Clinical Research Center of the hospital of the University of Pennsylvania. The first 2 days served as baseline nocturnal-sleep periods (B1, B2), which were consistent with the sleep durations subjects typically obtained at home. The subsequent 7 days involved sleep restriction

(P1–P7), and the final day (R1) permitted recovery sleep (ad libitum up to 10 hours). A subset of subjects (n = 8) had an 8th day of sleep restriction (i.e. P8), and another subset of subjects (n = 8) had a second recovery day (R2) of testing. Daytime naps were not permitted during any portion of the laboratory phase (except for MSLT). During the 7 days of sleep restriction, sleep was limited by delaying nocturnal-sleep onset and advancing wake-up equally. Sleep duration was monitored by a combination of behavioral observation, wrist actigraphy, and sleep logs, and daily sleep duration was calculated by averaging the minimum and maximum sleep duration each day as determined by these techniques. Each day subjects were tested at 1000, 1600, and 2200 hours on performance and mood battery. In between test bouts, subjects were free to engage in routine social activities and ambulation. Meals were provided to them. Caffeine, smoking, alcohol, and medications that can induce drowsiness were not permitted during the laboratory protocol. To simulate real world conditions, time information was available to them, light exposure was not controlled for, and females were studied without regard to menstrual-cycle phase.

Neurobehavioral tests

On laboratory protocol baseline days 1–2, sleep restriction days 1–7, and recovery sleep day 1, subjects were tested (1000, 1600, and 2200 hours) on a 20-minute performance and mood battery developed by us and validated in studies of total sleep deprivation (28,29). The battery included a 10-minute visual psychomotor vigilance task (PVT) for evaluation of sustained attention (30). This test has only a 1–3-trial learning curve (29). Analysis of PVT results is predicated on a multidimensional model of how sleepiness affects performance (7,29,31). For this report, three PVT performance metrics were evaluated: 1) the number of lapses (i.e. RTs $\geq$ 500 milliseconds) (32), 2) increases in the duration of responses in the lapse domain (i.e. mean 1/RT from slowest 10% RTs per trial), and 3) shifts in optimum reaction times (RTs) (i.e. fastest 10% RTs per trial). The number correct on a 4-item, probed recall memory (PRM) test (33) was included in the test battery for evaluation of working memory performance. A 1-minute serial-addition task (SAD) was included for evaluation of cognitive throughput (this latter test was added midway through the experiment, limiting results to eight subjects). The profile of mood states (POMS) (34) was interspersed with performance tasks during the test battery. The Stanford sleepiness scale (SSS) (35) was completed at the end of each performance bout (i.e. postbout). Multiple sleep latency tests (MSLTs) were performed at baseline and on day 5 of PSD in a subset of subjects (n = 8).

Thirteen of the subjects also completed (each morning after awakening and before bed at night) a daily sleep log (36) while in the laboratory, in which they made ratings on 100 mm visual analog scales (VAS) of sleep quality (very poor–excellent), morning tiredness (extremely tired–very refreshed), daytime alertness (alert–sleepy), stress (stressed–calm), happiness (happy–unhappy), health (sick–healthy), physical exhaustion (physically exhausted–energetic), and mental exhaustion (mentally exhausted–sharp). In addition, at the end of each day, subjects were asked to "list any illness, infection, pain, discomfort, worry, or problem" they had that day but to confine such listings to what they perceived to be significant problems (open-ended response). This entry was intended to more systematically capture three categories from the domain of subjective complaint (i.e. sleepiness, somatic, and cognitive/emotional problems) that have been reported anecdotally to increase with sleep restriction (23).

Statistical analyses

Evaluation of systematic changes in each neurobehavioral parameter across days (i.e. sleep restriction effect) and within days (i.e. diurnal effect) as well as their interaction, were assessed by within-subject analysis of variance (ANOVA), with significance levels corrected for sphericity by Greenhouse-Geisser epsilon. Significant F ratios were further analyzed by single degree of freedom polynomial contrasts for linear, quadratic, and cubic trends across days of sleep restriction (polynomial analyses were only performed on data for which there was significant variation across days). These analyses included baseline days in order to evaluate the change in neurobehavioral functions from baseline to increasing days of sleep restriction. In addition, ANOVAs were performed on each variable both including (i.e. B1, B2, P1, P2, P3, P4, P5, P6, P7, and R1) and excluding recovery day data (i.e. B2, P1, P2, P3, P4, P5, P6, and P7). The latter was done to confirm that systematic changes across days were not due simply to recovery sleep; polynomial trend analyses for data from B2 to P7 yielded information on the nature of the cumulative changes across days. However, it is important to note that every ANOVA that was significant without including recovery data was also significant with the inclusion of recovery data. When appropriate, paired t tests were used to compare outcomes at discrete time points.

RESULTS

Subjects averaged 7.41 hours [standard deviation (SD) = 0.90] sleep during baseline nights, 4.98 hours

TABLE 1. *Summary of ANOVA results and single degree-of-freedom polynomial contrasts for subjective sleepiness and mood*

	Day[a]		Polynomial trend			Time of day[b]		Polynomial trend		
Variable	$F_{7,105}$	p[c]	Type	$F_{1,15}$	p	$F_{2,30}$	p[c]	Type	$F_{1,15}$	p
SSS (postbout)	10.22	0.0001	linear	9.01	0.001	4.50	0.029	linear	1.71	—
			quadratic	2.92	—			quadratic	10.34	0.006
			cubic	27.99	0.0001					
POMS fatigue	14.45	0.0001	linear	26.52	0.0001	1.52	—			
			quadratic	3.89	—					
			cubic	14.40	0.002					
POMS vigor	5.45	0.002	linear	8.22	0.012	1.36	—			
			quadratic	7.75	0.014					
			cubic	11.87	0.004					
POMS confusion	6.20	0.001	linear	9.88	0.007	0.92	—			
			quadratic	3.17	—					
			cubic	12.57	0.003					
POMS tension	3.88	0.008	linear	10.95	0.005	2.77	—			
			quadratic	2.71	—					
			cubic	0.12	—					
POMS anger	1.56	—				1.40	—			
POMS depression	1.11	—				0.23	—			
POMS TMD	9.56	0.0001	linear	19.32	0.001	0.90	—			
			quadratic	7.28	0.017					
			cubic	10.89	0.005					

ANOVA, analysis of variance; SSS, Stanford sleepiness scale; POMS, profile of mood states.
[a] Main effect for day (B2–P7).
[b] Main effect for time of day (1000, 1600, and 2200 hours).
[c] Corrected by Greenhouse-Geisser epsilon.

(SD = 0.57) sleep during restriction nights, and 7.94 hours (SD = 0.58) sleep during recovery nights. Consequently, restricting sleep to 50% of ideal (preferred) sleep duration resulted in restricting it to an average 67% of habitual sleep duration, which was an average reduction of 2.43 hours per night for seven consecutive nights.

Subjective sleepiness and mood ratings during test bouts

Sleepiness and POMS were rated during each neurobehavioral assessment bout each day. Table 1 displays the results of the ANOVAs run on these subjective data as a function of time of day (1000, 1600, and 2200 hours) and day of study (B2, P1, P2, P3, P4, P5, P6, and P7). There were no statistically significant interactions (df = 14, 210) between day and time of day for any subjective ratings, and therefore these nonsignificant F ratios are not shown in Table 1. SSS ratings were the only subjective variables to show statistically reliable differences as a function of time of day; sleepiness ratings at 1000 hours were significantly above those at 1600 hours and 2200 hours ($F_{2,30}$ = 4.50, p = 0.029). Subsequent analyses focused on the main effects of days of sleep restriction.

The most consistent effect on subjective sleepiness and mood was a reliable change across days of sleep restriction (i.e. B2–P7). As is evident in Table 1, with the exception of two mood dimensions (anger-hostility and depression-dejection), sleep restriction resulted in elevations in subjective sleepiness ratings (p = 0.0001) and POMS subscales for fatigue-inertia (p = 0.0001), confusion-bewilderment (p = 0.001), tension-anxiety (p = 0.008), and total mood disturbance (p = 0.0001). Similarly, vigor subscale scores from the POMS declined across days (p = 0.002). Polynomial analyses of data from B2 through P7 revealed significant linear trends across days in sleepiness (p = 0.001), fatigue (p = 0.0001), vigor (p = 0.012), confusion (p = 0.007), tension (p = 0.005), and total mood disturbance (p = 0.001). However, except for tension scores, significant cubic trends across days were also evident for each of these subjective variables (Table 1).

Figure 1 displays the results for SSS scores including the data for baseline days and the recovery day. Analysis of changes across adjacent days revealed that SSS scores increased significantly from baseline day (B2) to the first day after a night of restricted sleep (P1) (t = −4.40, df = 15, p = 0.001) and remained elevated for all subsequent sleep restriction days relative to baseline (p = 0.003–0.0001). There were no statistically significant differences between SSS scores on P1 and the subsequent 5 days (P2–P6). However, after the P7 night of restricted sleep, SSS ratings were significantly elevated above all previous days (P2–P6) (p = 0.039–

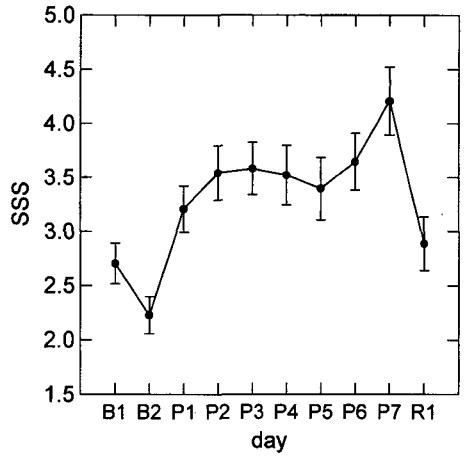

FIG. 1. Temporal profile of mean [standard error of the mean (SEM) bars] Stanford sleepiness scale (SSS) scores after each of two baseline nights (B1, B2), after each of seven consecutive sleep restriction nights (P1–P7), and after a recovery night of sleep (R1) for 16 subjects. Means are for SSS ratings made at the end of performance bouts at 1000, 1600, and 2200 hours each day. Sleep duration averaged 7.41 hours on B1 and B2 nights, 4.98 hours on P1–P7 restriction nights, and 7.94 hours on R1 recovery nights. There was significant variation in SSS scores across days ($F_{9,135}$ = 9.63, p = 0.00001). In general, SSS ratings on baseline and recovery days were significantly above those on sleep restriction days, and the ratings made on P7 were significantly above those on other sleep restriction days (see text).

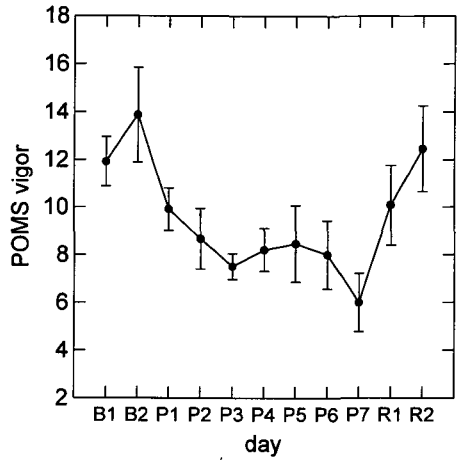

FIG. 2. Temporal profile of mean (SEM bars) vigor subscale scores from the profile of mood states (POMS) for eight subjects who were tested after a second recovery night (R2). Abscissa designation same as in Fig. 1. As with all 16 subjects there was significant variation in vigor subscale scores across days ($F_{10,70}$ = 5.29, p = 0.0052) for these eight subjects. Vigor scores on baseline and the second recovery day (R2) were significantly below those on sleep restriction days, and the scores on sleep restriction day P7 were significantly below those on other sleep restriction days (see text).

0.006). This further elevation of subjective sleepiness on P7 appears to be a genuine effect of sleep restriction rather than due to demand characteristics or end-of-experiment expectations. This conclusion is based on the data from a subset of eight subjects who were told that sleep restriction would continue for 10 days, but, in fact, it stopped after an additional night of sleep restriction (P8). Their SSS scores also rose significantly on P7 and remained elevated on P8.

Following a night of recovery sleep (R1), SSS sleepiness scores were significantly lower relative to ratings made on P7 (p = 0.0004), P6 (p = 0.004), P5 (p = 0.038), P4 (p = 0.012), and P3 (p = 0.007). There was a trend for a reliable difference relative to P2 (p = 0.0635) and no difference between SSS ratings on R1 relative to B1 and P1, suggesting that subjects had returned to near baseline levels of subjective sleepiness. However, SSS ratings on R1 were elevated relative to ratings on B2 (t = −2.16, df = 15, p = 0.047). To determine whether this reflected inadequate recovery after only one night of recovery sleep, analyses were performed on the subset of subjects (n = 8) who were also evaluated after a second night of recovery sleep (R2). Their SSS scores after R2 were not significantly different from their scores on B1, B2, and R1, but they were at the level of B2 and, like the latter, were significantly below ratings after one night of restricted sleep (P1 mean = 3.17, R2 mean = 2.41; t = 2.26, df = 7, p = 0.058). This suggests that, in terms of subjective sleepiness, recovery was complete after two full nights of sleep. Exactly the same sleep restriction and recovery profiles were observed for vigor, fatigue, and confusion subscales of the POMS. The results for POMS vigor are shown in Fig. 2 for the eight subjects who had two nights of recovery sleep.

Fatigue-inertia subscale scores from the POMS showed a pattern nearly identical to SSS results. There were significant increases in fatigue scores from B2 to P1 (t = −5.775, df = 15, p = 0.0001) and from days P2–P6 compared to P7 (p = 0.042–0.006). Unlike SSS ratings, however, fatigue scores also increased significantly from P1 to P2 (t = −2.90, df = 15, p = 0.01). The confusion-bewilderment and tension-anxiety subscales from the POMS, as well as total mood disturbance scores (Table 1), yielded a pattern of results similar to that found for the fatigue subscale (P1–P7 significantly above B2, P2 significantly above P1, and P7 significantly above all or most of the means for P2, P3, P4, and P5).

Performance

Table 2 displays the results of the ANOVAs run on each performance variable from the PVT as well as the PRM and the SAD tasks. Results are shown for the time-of-day factor (1000, 1600, and 2200 hours) and the sleep restriction factor (B2, P1, P2, P3, P4, P5, P6, and P7). Similar to SSS results, but unlike mood data (Table 1), four of the five performance parameters showed statistically significant (p = 0.004–0.0001) variation by time of day (main effects). However, sim-

TABLE 2. *Summary of ANOVA results and single degree-of-freedom polynomial contrasts for performance variables*

	Day[a]		Polynomial trend			Time of day[b]		Polynomial trend		
Variable	$F_{7,105}$	p[c]	Type	$F_{1,15}$	p	$F_{2,30}$	p[c]	Type	$F_{1,15}$	p
PVT fastest 10%	3.69	0.018	linear	5.92	0.028	27.29	0.0001	linear	38.37	0.0001
			quadratic	2.66	—			quadratic	7.49	0.015
			cubic	2.72	—					
PVT lapse total[d]	7.11	0.0001	linear	24.59	0.0001	8.53	0.004	linear	9.37	0.008
			quadratic	3.23	—			quadratic	4.49	0.051
			cubic	2.66	—					
PVT slowest 10%[e]	9.69	0.0001	linear	21.44	0.0001	11.70	0.0001	linear	20.56	0.0001
			quadratic	9.82	0.007			quadratic	1.11	—
			cubic	6.35	0.024					
PRM correct	1.51	—				3.56	0.05	linear	1.81	—
								quadratic	5.65	0.031
SAD correct	2.85[f]	—				0.77	—			

PVT, psychomotor vigilance task; PRM, probed recall memory; SAD, serial-addition task; RT, reaction time.
[a] Main effect for day (B2–P7).
[b] Main effect for time of day (1000, 1600, and 2200 hours).
[c] Corrected by Greenhouse-Geisser epsilon.
[d] ANOVA conducted on transformed lapse frequency ($\sqrt{x} + \sqrt{[x + 1]}$).
[e] ANOVA conducted on transformed data (1/RT).
[f] Based on data from eight subjects.

ilar to sleepiness and mood data, these time-of-day main effects did not interact (df = 14, 210) with effects across days for any performance parameter—therefore, these nonsignificant F ratios are not shown in Table 2. In general, the time-of-day effect on the three PVT parameters was associated with poorest performance at 1000 hours and best performance at 2200 hours (data for 1600 hours was close to that of 1000 hours for lapse total and duration but similar to that of 2200 hours for fastest RTs). The significant time-of-day main effect on PRM performance was associated with best performance at 1600 hours.

As indicated in Table 2, PVT performance parameters displayed significant variation across days of sleep restriction (B2–P7). Fastest RTs slowed across days (p = 0.018), while lapse frequency increased (p = 0.0001). In both cases, the pattern of change across days contained only significant linear polynomial trends (fastest RTs, p = 0.028; lapse total, p = 0.0001). Like subjective data, however, PVT slowing (i.e. lapse domain duration or slowest 10%) not only varied systematically across days (p = 0.0001) but also contained significant linear (p = 0.0001), quadratic (p = 0.007), and cubic (p = 0.024) trends. In all cases, PVT performance parameters were worse on P7. As with subjective parameters this effect did not appear to be due to demand characteristics because performance deficits continued at this elevated level in the subgroup of eight subjects who had an 8th day of sleep restriction (P8).

Memory performance on the PRM task showed a trend toward poorer performance across days of sleep restriction with subsequent recovery, but the pattern was not statistically reliable (B2–R1, $F_{8,120} = 1.66$, p = 0.15). Addition task performance on the SAD yielded a nearly significant main effect across days (p = 0.063), but this effect was in the direction of a linear improvement in performance due to learning (i.e. a practice effect).

Analysis of PVT performance changes across days revealed that unlike subjective SSS ratings and POMS fatigue scores that increased significantly after the first night of sleep restriction (P1) relative to baseline (B2), declines in PVT fastest RTs and increases in lapse totals did not undergo such rapid changes. Lapse frequency data are shown in Fig. 3. Lapsing did not increase significantly after the first night of sleep restriction (B2 vs. P1, $t = -1.20$, df = 15), but did increase significantly between the first and second nights of sleep restriction (B2 vs. P2, $t = -2.86$, df = 15, p = 0.012; P1 vs. P2, t = −2.81, df = 15, p = 0.013). Increases in lapses on subsequent days of sleep restriction were modest (like subjective sleepiness and fatigue), but by P7 there were more lapses than on P1 (p = 0.001), P2 (p = 0.046), P3 (p = 0.069), and P4 (p = 0.035). Similar results were obtained for decreases in the fastest RTs across adjacent days. In contrast, results for changes in the PVT slowest 10% (lapses domain duration) were comparable to those obtained for subjective sleepiness and fatigue; significant slowing occurred after the first night of sleep restriction (B2 vs. P1, $t = 2.14$, df = 15, p = 0.049) and continued after the second night (P1 vs. P2, $t = 3.02$, df = 15, p = 0.009). Like SSS and POMS vigor scores (Figs. 1 and 2), PVT slowing on P7 was significantly worse than all other sleep restriction days including P6 ($t = 2.30$, df = 15, p = 0.036), and like subjective data, PVT performance parameters did not

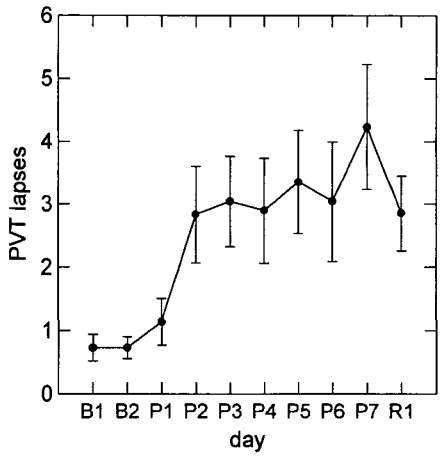

FIG. 3. Temporal profile of mean (SEM bars) psychomotor vigilance task (PVT) lapses after each of two baseline nights (B1, B2), after each of seven consecutive sleep restriction nights (P1–P7), and after a recovery night of sleep (R1) for 16 subjects. Means are for three 10-minute PVT trials performed at 1000, 1600, and 2200 hours each day. Sleep duration averaged 7.41 hours on B1 and B2 nights, 4.98 hours on P1–P7 restriction nights, and 7.94 hours on R1 recovery nights. There was significant variation in PVT lapse frequency across days ($F_{9,135}$ = 5.85, p = 0.0006). In general, PVT lapsing began to occur significantly after night P2 and again after night P7.

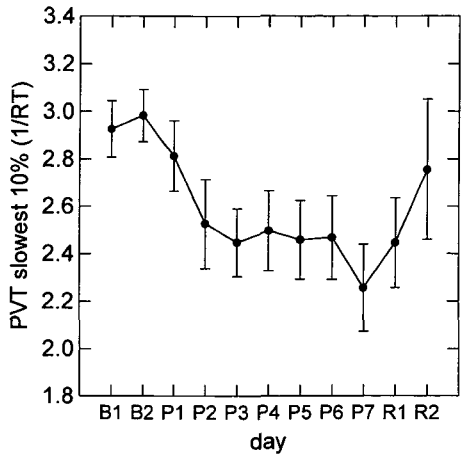

FIG. 4. Temporal profile of mean (SEM bars) PVT duration of slowest 10% RT responses in the lapse domain (i.e. mean 1/RT from slowest 10% RTs per trial) for eight subjects who were tested after a second recovery nights (R2). Abscissa designation same as in Fig. 1. As with all 16 subjects there was significant variation in slowing across days for these eight subjects ($F_{10,70}$ = 4.44, p = 0.01). PVT slowest 10% on baseline and the second recovery day (R2) were not significantly different from each other but were significantly different from P3, P4, P5, P6, and P7.

fully normalize until after a second recovery night of sleep. For example, as shown in Fig. 3, PVT lapses at R1 were still elevated relative to B1, B2, and P1 ($p < 0.006$). However, like subjective sleepiness, fatigue, and vigor, a second night of recovery sleep (R2) resulted in normalized performance (i.e. little or no lapsing). This is illustrated in Fig. 4 for PVT slowest 10% (lapse duration).

MSLT

MSLT results, which were obtained only on days B2 and P5, were consistent with subjective sleepiness, fatigue, and mood disturbance outcomes as well as objective PVT performance findings. Mean MSLT decreased significantly from B2 (mean = 11.06 minutes, SD = 2.37) to P5 (mean = 3.03 minutes, SD = 2.04) (t = 9.03, df = 7, p = 0.0001) indicating increased sleep propensity and reduced alertness after sleep restriction.

Daily log: VAS ratings

Results from analyses of the six bipolar VAS ratings regarding how subjects "felt overall" each day (alert–sleepy, stressed–calm, happy–unhappy, sick–healthy, physically exhausted–energetic, and mentally exhausted–sharp) are presented in Table 3. While there were no statistically reliable changes in global ratings of physical exhaustion, happiness, and health across study days, there were significant increases during sleep restriction in subjects' ratings of overall sleepiness (p = 0.032), mental exhaustion (p = 0.009), and stress (p = 0.004). The profile of increasing VAS sleepiness was similar to that found for SSS (Fig. 1) with the most marked increases in sleepiness occurring after the first night (P1) and during the final days (P6 and P7) of sleep restriction (cubic trend p = 0.028). Mental exhaustion ratings increased primarily across the first 3 days of sleep restriction (quadratic trend p = 0.006). The profiles of VAS ratings of stress were similar to those of

TABLE 3. *Summary of ANOVA results and single degree-of-freedom polynomial contrasts for visual analog scales from daily log*

Variable	Day[a]		Polynomial trend		
	$F_{7,84}$	p[b]	Type	$F_{1,12}$	p
Alert–sleepy	2.86	0.032	linear	3.25	—
			quadratic	1.32	—
			cubic	6.22	0.028
Mentally exhausted–sharp	3.73	0.009	linear	6.54	0.025
			quadratic	10.84	0.006
			cubic	0.53	—
Physically exhausted–energetic	1.56	—			
Stressed–calm	4.04	0.004	lincar	11.83	0.005
			quadratic	2.89	—
			cubic	3.11	—
Happy–unhappy	1.39	—			
Sick–healthy	2.51	—			

[a] Main effect for day (B2–P7).
[b] Corrected by Greenhouse-Geisser epsilon.
[c] Based on data from 13 subjects.

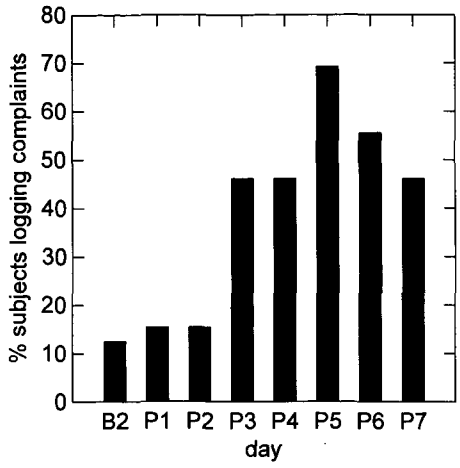

FIG. 5. The proportion of subjects (total n = 13) who on baseline day (B2) and each of seven sleep restriction days (P1–P7) wrote in a complaint in their daily log in response to the instruction to list any significant "illness, infection, pain, discomfort, worry, or problem" experienced that day. These entries generally consisted of three types: complaints of sleepiness, somatic complaints, and complaints of cognitive or emotional problems (see text for examples).

the POMS tension-anxiety subscale (Table 1) showing a linear increase across days ($p = 0.005$).

Daily log: complaints

A tally was made of subjects' responses on the daily log to the open-ended instruction to list any significant "illness, infection, pain, discomfort, worry, or problem" they experienced. This entry was intended to more systematically capture three categories from the domain of subjective complaint that have been reported anecdotally to increase with sleep restriction (23). Figure 5 displays the proportion of subjects who listed a complaint of any kind on each day of the protocol. Complaints generally were of three types: 1) sleepiness complaints included reports of daytime sleepiness, excessive tiredness, difficulty staying awake during the daytime, etc.; 2) somatic complaint examples included reports of headaches, gastrointestinal problems, sore joints, etc.; 3) cognitive/emotional complaints consisted of reports of problems of concentration, lassitude, emotional lability, etc. As shown in Fig. 5, fewer than 15% of subjects reported a complaint on any day up to and including P2, but this percentage increased three-fold on P3 and remained elevated thereafter. No single category of complaint dominated these elevated percentages.

DISCUSSION

Restricting sleep to an average of approximately 5 hours (±35 minutes) a night for seven consecutive nights had a clearly measurable effect on neurobehavioral markers of alertness, especially measures of sleepiness, fatigue, mood disturbance, stress, and PVT performance lapsing. Cumulative growth functions in each of these domains were not only statistically reliable across days but they were also surprisingly similar in temporal profile. Subjective sleepiness and fatigue increased immediately and significantly in response to sleep restriction while some aspects of psychomotor vigilance performance, notably the frequency of lapses, showed a significant elevation after the 2nd day of sleep restriction. Moreover, virtually every performance and mood variable that was sensitive to sleep restriction displayed continued growth of deficit in the final day or two of PSD, as evident in significant linear and cubic trends in the profiles of the sleepiness, mood disturbance, and PVT performance variables (Tables 1–3). MSLT results confirmed that daytime sleep propensity had increased significantly by the 5th day of sleep restriction. While time-of-day effects were statistically significant in many measures that responded to sleep restriction, there was no evidence of an interaction between PSD days and time of day, at least not for the three times we sampled in the period from 10:00 a.m. to 10:00 p.m. This does not rule out, however, possible differential effects of sleep restriction on other circadian phases not sampled in this study.

The effects of sleep restriction appeared to level off (i.e. cease to accumulate) between the 2nd and 5th day of PSD for subjective sleepiness (Fig. 1), and between the 2nd and 6th day of sleep restriction for PVT performance variables (Figs. 3 and 4). This suggests that following an initial shift in daytime sleepiness levels some "adaptation" to sleep restriction occurred during the subsequent 4–5 days, which is consistent with the perspective that sleepiness resulting from PSD in the range of 5–6 hours can be adapted to (3,6,24). Although we did not polysomnographically monitor sleep during PSD, this apparent adaptation (i.e. steady state sleepiness level) may have been the result of changes in sleep itself during restricted nights. Brunner and colleagues (37) reported that sleep restricted to 4 hours/night across four nights showed a rapid increase in electroencephalograph (EEG) power density in the delta and theta frequencies with a peak in slow-wave activity, which then appeared to achieve a steady state across subsequent days of PSD. They attributed these EEG spectral changes to non-rapid eye movement (NREM) sleep homeostasis. However, cumulative increases across PSD days have also been observed in sleeping EEG power density in the high delta band and concomitant decreases in the low alpha band (37,38) that have been hypothesized to reflect pressure for REM sleep (37), as have changes in nocturnal-sleep latency (37). These latter changes during sleep restriction may have an analog

in continuing increases in daytime sleepiness of the kind we observed.

Regardless of what produced the leveling off of sleepiness that we recorded in the middle of the week of sleep restriction, this apparent steady state was ephemeral. By the 7th day of PSD, and in some cases the 6th day, subjective sleepiness, fatigue, and PVT performance deficits were again rising in a statistically reliable manner. This finding raises the possibility of a step-like function in sleepiness within subjects across days of sleep restriction, at least for this dosage of PSD. The further elevation of subjective sleepiness and performance lapsing on P7 was confirmed to continue on P8 in the subgroup of eight subjects who were tested for an additional day of sleep restriction. Thus, the further elevation of sleepiness on P6–P7 appears to be a genuine effect of sleep restriction, not attributable to demand characteristics or end-of-experiment expectations.

The profile of cumulative performance changes that we observed across 7–8 days of acute sleep restriction to approximately 5 hours/night is novel among studies of the effects of sleep restriction on performance. Our findings are similar, however, to MSLT data acquired by Carskadon and Dement (1) in a study of sleep restricted to 5 hours per night for seven consecutive nights. To fully evaluate just how similar the results were from the two experiments, we obtained and re-analyzed the raw MSLT data from Carskadon and Dement (1). We were particularly interested in contrasting the temporal dynamics of changes in our subjects' PVT performance (lapsing) during sleep restriction to the comparable dynamics of their MSLT results. Figure 6 displays the profile of results from the two experiments. The similarity between our PVT lapse function across days of PSD and the MSLT function of Carskadon and Dement (1) is remarkable—the correlation between the two data sets across the 8 days (i.e. last baseline day through 7th day of sleep restriction) depicted in Fig. 6 was $r = -0.95$ ($p = 0.0001$). The distance weighted least-squares functions fit to the two data sets show the characteristic increase in responses across the first 2 days of sleep restriction, followed by less change during the next 4–5 days, followed by a further decrease in alertness in the last day or two. The dominant polynomial trend in both of these functions was linear ($p = 0.0001$), which means that the most appropriate conclusion regarding the increases in PVT lapses and the decreases in MSLT values found in a comparable experiment of 7 days of sleep restricted to 5 hours (1) is that these changes are cumulative and show little evidence of having reached an asymptote. At least within the conditions of these two experiments, therefore, cumulative nocturnal-sleep debt had an analog in cumulative diurnal sleepiness. This suggests that persons who experience limited sleep durations may be at risk for developing cumulative waking neurobehavioral deficits. This risk may apply to a wide range of persons including nightshift workers, on-call medical personnel, and patients with sleep apnea who have reduced nightly durations of continuous positive airway pressure (CPAP) use (39).

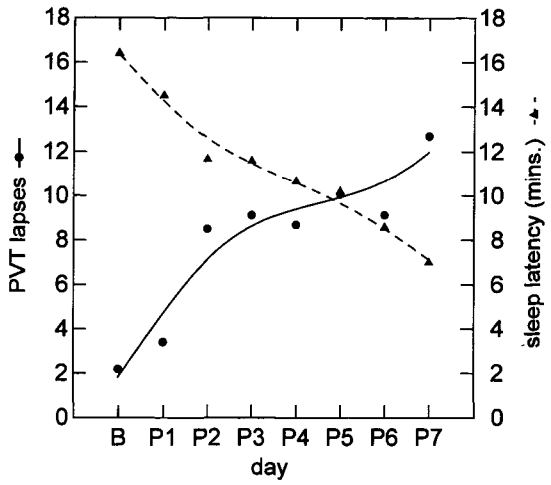

FIG. 6. Mean daily PVT lapse totals ($\sqrt{x} + \sqrt{[x + 1]}$) from the current study, and mean daily MSLT values from a study of sleep restricted to 5 hours/night for seven nights [data from Carskadon and Dement (1)]. Baseline data (B) is from day B2 in the current study and B3 from Carskadon and Dement. Days P1through P7 are consecutive days of sleep restriction in each study. Lines fitted to each dataset are distance-weighted, least-squares functions (tension = 0.50) that illustrate the cumulative change across all 8 days. The correlation between the means of the two datasets across the 8 days depicted is $r = -0.95$ ($p = 0.0001$).

Since the experiment relied on cumulative changes from baseline levels of alertness rather than a control condition of no sleep restriction for seven consecutive days, it is possible that the observed cumulative effects were not due to sleep restriction per se but rather to other procedural requirements of the study. We believe this is unlikely because the cumulative increases occurred in the same neurobehavioral measures that have proven sensitive to TSD (7,29,31)—for example, SSS, POMS fatigue and confusion subscales, VAS alertness and mental exhaustion ratings, and PVT lapses, fastest 10% and slowest 10%. Moreover, the lack of effects on other subjective dimensions (e.g. unhappiness and physical exhaustion) and the maintenance of the effects on day P8 make it unlikely that the cumulative effects of sleep restriction on alertness-related neurobehavioral measures were due to study procedures, subjects' expectations, or experimental demand characteristics. Thus, PSD appeared to be affecting sleepiness–alertness through an elevated homeostatic drive for sleep that was clearly confirmed by the limited MSLT data we obtained.

We observed an increased incidence of subjects re-

cording significant problems with sleepiness and increasing cognitive, emotional, and somatic complaints during the study (Fig. 5). This result is consistent with other studies of sleep restricted between 4.5 and 5.5 hours per night (5,23,26). We believe these anecdotal complaints from subjects may actually reflect the stressor-induced demands from PSD, including the challenge of coping with escalating daytime sleepiness, its cognitive effects, and the compensatory effort required to remain awake and motivated each day. These stress-related neurobehavioral effects from PSD may increase sensitivity to dysphoria and mood lability that are often attributed to chronically inadequate sleep but rarely seen in acute studies of total sleep deprivation. Whether some of these effects are mediated by alterations in stress-related neuroendocrine axes induced by sustained sleep restriction remains to be determined.

It is also noteworthy that we failed to observe effects from sleep restriction on the four-item PRM and the 1-minute SAD. The PRM was developed and validated by us in experiments on TSD (33). Relative to TSD, the less severe sleepiness of PSD resulted only in a trend in the direction of poorer PRM performance by the end of sleep restriction (B2–P7, $t = 1.74$, df = 15, $p = 0.10$). In contrast to all other tests, performance on the SAD task tended to improve across study days, indicating a continuing practice effect. Unfortunately, such learning curves, which confound the effects of sleep restriction, have been observed in many of the studies of PSD (5,11,14,22,23,25–27). Without the PVT data we might have concluded that sleep restriction to approximately 5 hours a night increased subjective sleepiness but did not affect performance. This is the problem of attempting to prove the null hypothesis, and it seems to have been the conclusion of some previous studies of sleep restriction in which task-learning dynamics confounded the need to sensitively detect changes in performance across days of sleep restriction. Clearly, future experiments on the effects of sleep restriction must give more thought to the nature of the specific performance tests being used repeatedly and their effects on the very phenomenon under study.

Recovery from the effects of sleep restriction for 7 days appeared to depend on obtaining two nights of recovery sleep (Figs. 2 and 4). The reasons for this remain unclear but suggest that a single night of recovery sleep may be inadequate following sustained partial sleep deprivation. Finally, we note that the temporal profile of the (cumulative) effects of sleep restriction will almost certainly also depend upon a number of factors not tested in this protocol such as sleep dosage and the abruptness and chronicity of the sleep restriction. Until these variables are thoroughly tested our conclusions are limited to acute restriction of sleep to approximately 5 hours per night for seven consecutive days, but within this range our subjects clearly showed neurobehavioral deficits even on a relatively brief test battery.

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