

4

Psychomotor Vigilance Performance: Neurocognitive Assay Sensitive to Sleep Loss

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I. Overview

It is well established that sleep deprivation, both acute total and chronic partial sleep restriction, results in significant impairment of neurobehavioral functioning. To quantify the magnitude of such changes, as well as to track the temporal profile in neurobehavioral degradation as sleep loss accumulates, a wide array of neurocognitive assays have been used. However, many of these tests are not well suited for assessment of performance across multiple sleep-wake cycles owing to significant inter- and intrasubject variability. To provide an accurate and useful measure of performance during sleep loss, and the expression of waking neurobehavioral integrity as it changes dynamically over time, neurocognitive assessments must validly and reliably reflect fundamental aspects of waking functions that are altered by sleep deprivation. Since such neurobehavioral assays will need to be administered to persons of different aptitudes and repeatedly over time, they should be devoid of substantial inter-subject and intrasubject (e.g., learning) variability. Such assays should also provide meaningful outcome variables that can be easily interpreted relative to neurobiological and cognitive constructs. In this chapter we review one neurobehavioral assay that has met these criteria—the psychomotor vigilance task (PVT).

II. Sleep Deprivation and Neurocognitive Performance

It has long been established that sleep deprivation degrades aspects of waking neurobehavioral capability (reviewed in Refs. 1–4). In humans, numerous tests have been designed to capture specific elements of waking cognitive functions. The resulting neurocognitive performance measures provide an index of the degree of functional impairment present in an individual, and they have been used to answer both applied and theoretical questions about the nature of neurobehavioral capability in healthy people deprived of sleep.

Decades of research on human sleep deprivation have resulted in the view that the extent to which a given cognitive task reveals changes during sleep deprivation depends upon such task parameters as duration (5–7), complexity (8,9), response rate (10), and interest (5,8,11,12). As such, findings regarding the type and magnitude of performance impairment during sleep deprivation are, to a large extent, contingent as much on these task parameters as on any particular type of cognitive test. In recent years, a focus on the effects of sleep loss on specific brain regions—especially the prefrontal cortex (PFC)—has resulted in use of tasks that are thought to uniquely activate this substrate.

A wide variety of tests have been used in sleep deprivation experiments ranging from simple tests of reaction time (e.g., Refs. 1,2,13) to complex tasks of higher-order cognitive capacity and PFC function (e.g., Refs. 9,14,15). The diversity of performance tests available for use in performance testing leads to a fundamental question: *What are the criteria for an effective neurocognitive assay under conditions of sleep deprivation, where “effective” means theoretically meaningful, empirically sensitive, and practically useful?*

A. Stimulus-Response Approach

Since the first published experimental study of sleep deprivation and human cognitive performance in 1896 (13), investigators have employed a plethora of performance tests to measure the effects of sleep loss on neurobehavioral functioning. One common testing approach is the “stimulus-response” (S-R) method. This typically involves repeated presentation of visual or auditory stimuli, requiring a timely response from the experimental subject. Behavioral alertness is a core feature of the S-R approach as applied to the study of sleep deprivation. Examples of S-R tasks range from the attention-rich demands of simple reaction time tasks to sustained-attention vigilance tasks (as used in Ref. 2 and Ref. 16, respectively). S-R tasks predicated on sustaining attention over time have been used since the earliest studies of human sleep deprivation and performance (2,13). Several fundamental observations were made during these investigations, with experimenters describing phenomena that we now refer to as *microsleeps*, *hypnagogic reverie*, *lapsing*, *circadian* variation in performance, and increased depth of *recovery sleep* (2,3,13). Thus, the effects of sleep deprivation on the neurobiology of focused attention appear to be at the heart of the sensitivity of S-R

tasks to sleep deprivation (3). The mechanisms of attention have also recently been recognized to be a fundamental component of higher-order cognitive tasks subserved by the prefrontal cortex.

B. Executive Function Approach

In recent years, the effects of sleep loss have been increasingly evaluated using neurocognitive tests that focus on complex cognitive functions, particularly tests putatively subserved by the prefrontal cortex. Horne and colleagues, in particular, have championed the view that sleep loss uniquely affects PFC. Deficits on tasks subserved by PFC have been observed following sleep deprivation; examples include verbal fluency, creative thinking, nonverbal planning (15), confidence judgment (metamemory), temporal memory (14), response inhibition, verb-to-noun word generation (14,15) and word fluency (17,18). These findings have contributed to a frontal lobe hypothesis, which contends that sleep deprivation acts primarily in the frontal lobe, to produce frontal cortex dysfunction, reversible by recovery sleep (19).

A PFC-related function that has received particular study in the scientific literature is working memory. Imaging studies have indicated that performance on working memory tasks is reliant on dopamine receptors in the dorsolateral prefrontal cortex (20,21). It has been argued that working memory is dependent on a central executive attention system (22,23), and that constructs of executive attention and working memory are closely related (24), if not isomorphic (25). Importantly, performance on working memory tasks is predictive of performance on a range of other tasks of cognitive tests (25). Indeed working memory and its underlying executive attention are likely to be fundamental to performance on virtually any neurocognitive task. Put simply, without the basic ability to hold or sustain attention, it is impossible to perform any task in a goal-directed manner (3). Stable sustained attention is therefore a necessary, but not sufficient, criterion for normal cognitive functioning.

III. Criteria for a Neurocognitive Assay Sensitive to Sleep Deprivation

Proposed criteria for a sensitive behavioral assay for studying the temporally dynamic features of sleep deprivation are summarized in Table 1. Ideally, a neurocognitive assay for measuring the effects of sleep loss during waking performance should reflect an aspect of cognition that is: (1) basic to or essential for many expressions of performance, and (2) sensitive to the homeostatic drive for sleep in interaction with the endogenous circadian pacemaker. The ability to sustain attention on a task meets this first criterion because it is a basic feature of nearly all cognitive performance tests, including tests that have proven to be sensitive to sleep deprivation [i.e., from tests of vigilance (16) to creative thinking (15)]. A second criterion for an effective neurobehavioral assay of human sleep depriva-

Table 1 Criteria for a Neurocognitive Assay for Assessing the Effects of Sleep Deprivation

Criteria	Explanation/examples
Reflects a fundamental aspect of waking neurocognitive functions	Measures the ability to use attention or working memory over time Capitalizes on brain structures subserving basic cognitive functions
Suitable for repeated administration	Has a minimal learning curve Underlying psychometric properties do not change with repeated testing
Easily performed with no aptitude effects	Yields consistent results among a wide range of subject populations Can be taught quickly Can be used in laboratory experiments, simulator scenarios, and field situations
Task duration relatively brief	Prevents extraneous factors (e.g., lack of interest) from altering performance Easily integrated into experimental protocols involving repeated measurements
High signal load	Does not result in greatly augmented subject burden Provides a large number of behavioral samples in a brief period of time
Reliability	Challenges the subject to maintain cognitive output Provides test-retest stability Reflects trait-like inter-individual differences
Validity	Convergent validity—sensitive to many forms of sleep deprivation Ecological validity—sensitive to performance used in everyday functioning Theoretical validity—reflects changes consistent with theorized functions of sleep
Can be interpreted in a meaningful way	Yields metrics that can be translated to “real world” performance Yields metrics that can be related to sleep/wake physiology

tion is that the cognitive task should be easy to learn and perform (i.e., minimal intersubject variability in performance due to aptitude, and easily implemented in experimental protocols). This maximizes its utility across a larger segment of the population.

Since a large proportion of experiments investigating human performance during sleep deprivation involve repeated measures designs to properly evaluate temporally dynamic changes in neurocognitive functions over time, a performance task should ideally have a minimal learning curve to prevent masking effects from skill acquisition (26). It is important to note that exposing subjects to a

period of training prior to sleep deprivation may reduce learning, but it rarely completely eliminates learning during the experimental testing period, even if subjects reach asymptotic performance levels during training (26). Furthermore, repeated testing should not change the underlying psychometric properties of the test. For example, novelty is an important aspect of several tasks purported to measure PFC function, such as the Haylings Sentence Completion Task (27). Such tests are not suitable for multiple administrations during a study of sleep deprivation (i.e., within-subjects designs) as the task properties are significantly altered.

Other criteria for a neurobehavioral assay of the cognitive effects of sleep deprivation include task duration and signal load (i.e., stimulus rate). Tasks that are very long with low signal rates can induce excessive levels of task-related fatigue, boredom, and reduced motivation, which can contaminate sleep deprivation effects. To avoid this problem, a cognitive task assay during sleep deprivation should require a relatively large number of responses in a short time period. The high signal load allows the experimenter to sample a greater amount of behavior involving sustained cognitive output, and avoid the criticism commonly leveled at low signal load tasks, that the subject falls asleep because he/she is in a passive state.

Finally, a cognitive performance assay used repeatedly during sleep deprivation should have high test-retest reliability; it should be demonstrated to be sensitive to a large proportion of the performance phenomena associated with sleep loss; and it should have the capacity to reflect aspects of “real world” performance (i.e., ecological validity).

IV. Psychomotor Vigilance Task (PVT) as a Neurocognitive Assay for Sleep Loss

With all of the above criteria in mind, the psychomotor vigilance task (PVT) was developed as a neurocognitive assay for tracking the temporally dynamic changes induced by interaction of the homeostatic drive for sleep and endogenous circadian pacemaker. Its focus is on measurement of the ability to sustain attention and respond in a timely manner to salient signals (28). With a combination of PFC executive attention and traditional stimulus-response testing, the PVT involves a simple (as opposed to choice) reaction time (RT) test—the avoidance of choice RT was deliberate to minimize continued learning and strategy shifts that can occur even in four-choice RT tasks. The PVT requires responses to a small, bright-red-light stimulus (LED-digital counter) by pressing a response button as soon as the stimulus appears, which stops the stimulus counter and displays the RT in milliseconds for a 1-sec period. The subject is instructed to press the button as soon as each stimulus appears, to keep the reaction time as low as possible, but not to press the button too soon [which yields a false start (FS) warning on the display]. Simple to perform, the PVT has only very minor learning effects

(29–31) on the order of a 1–3-trial learning curve (32) [which contrasts dramatically with the 30- to 60-trial learning curve of other supposedly simple learning tasks such as the digit symbol substitution task (33)]. The PVT interstimulus interval varies randomly from 2 sec to 10 sec, and the task duration is typically 10 min, which yields approximately 90 RTs per trial (i.e., a relatively high signal load). The sensitivity of the PVT can be increased by using longer task durations (e.g., 20 min), which can be useful when studying mild to moderate levels of sleepiness or in the assessment of interventions purporting to reduce sleepiness [e.g., various pharmacological agents, naps, work-rest schedules (34)].

A. PVT Reliability

Reliability statistics have been calculated for the PVT using data from $n = 9$ subjects who were allowed an 8-hr sleep opportunity per night (i.e., the control group) as part of a larger chronic partial sleep deprivation protocol (35). PVT performance was assessed throughout the waking portion of each day. Test-retest statistics were obtained using daily performance averages (from tests taken at 09:30, 11:25, 13:20, and 15:15) on a baseline day and 5 consecutive experimental days. Intraclass correlation coefficients (ICC) measure the proportion of the variance explained by between-subject differences, as opposed to within-subject error. The ICC indicated maximal reliability for the number of PVT lapses (ICC = 0.888, $p < .0001$) and median response times (ICC = 0.826, $p < .0001$), falling into the standardized “almost perfect” range for a measurement assay (as discussed in Ref. 36).

B. PVT Validity

The PVT was designed to be sensitive to sleep deprivation (experimental, occupational, and clinical) induced in many different ways (i.e., through sleep fragmentation, acute prolonged waking, chronic partial sleep restriction, etc.). Research has repeatedly shown this to be the case (Table 2) and demonstrated that the PVT captures the neurocognitive effects of sleep loss on wake state stability as reflected in sustained attention. Furthermore, the PVT is without the confounds induced by extraneous intersubject (e.g., aptitude) and intrasubject (e.g., learning) sources of variance that plague most other cognitive tasks. In fact, there is considerable evidence that the PVT meets all of the criteria in Table 1. Use of the PVT to reflect neurocognitive performance changes that are consistent with theorized functions of sleep (i.e., theoretical validity) and to demonstrate sensitivity to many forms of sleep deprivation (i.e., convergent validity) will be considered in the following sections of this chapter. In particular, evidence will be reviewed on the extent to which PVT performance reveals: (1) behavioral lapses, variability, and state instability; (2) circadian and homeostatic variation; (3) the effects of chronic partial sleep deprivation; (4) the benefits of interventions for the neurobehavioral effects of sleep loss; and (5) individual differences in vulnerability to sleep loss. Further, interpretation of PVT data will be considered, with partic-

Table 2 Summary of Published Literature on PVT Sensitivity to Sleep Deprivation

Context	References
Interaction of homeostatic sleep drive and endogenous circadian pacemaker	Dinges & Kribbs, 1991 (3); Wyatt et al., 1997 (59); Rogers et al., 2002 (106); Graw et al., 2004 (127)
Total sleep deprivation	Dinges et al., 1994 (105); Kribbs & Dinges, 1994 (32); Jewett et al., 1999 (30); Konowal et al., 1999 (94); Atzram et al., 2001 (95); Doran et al., 2001 (44); Van Dongen et al., 2003 (68)
Chronic sleep restriction (cumulative partial sleep deprivation)	Dinges et al., 1997 (29); Rowland, 1997 (107); Kuo et al., 1998 (108); Johnson et al., 1998 (109); Jewett et al., 1999 (30); Balkin et al., 2000 (110); Drake et al., 2001 (71); Van Dongen et al., 2003 (35); Van Dongen et al., 2003 (68); Belenky et al., 2003 (70)
Prophylactic naps in sleep-deprived subjects	Dinges et al., 1987 (77); Rosekind et al., 1994 (31)
Caffeine in sleep-deprived subjects	Wright et al., 1997 (84); Dinges et al., 2000 (76); Van Dongen et al., 2001 (78); Wyatt et al., 2004 (124)
Body posture changes in sleep-deprived subjects	Caldwell et al., 2003 (111)
Sleepiness in the elderly	Pack et al., 1997 (116); Maislin et al., 2001 (117)
Slow eyelid closures of the kind experienced by drowsy drivers	Dinges et al., 1998 (112); Mallis et al., 1999 (113); Price et al., 2003 (96)
Simulated night shift work in laboratory	Hughes et al., 2001 (114); Lamond et al., 2003 (115); Caldwell et al., 2003 (130); Lamond et al., 2004 (128)
Jet lag and simulated night flights in transoceanic pilots	Rosekind et al., 1994 (31); Neri et al., 2002 (118); Russo et al., 2004 (126)
Astronauts during space missions	Dijk et al., 2001 (119)
On-call demands of medical house staff	Geer et al., 1995 (120); Smith-Coggins et al., 1997 (121); Howard et al., 2003 (125)
Intra- and intersubject variability	Doran et al., 2001 (44); Van Dongen et al., 2003 (68)
Excessive sleepiness from untreated sleep apnea (OSA), and residual sleepiness in OSA patients treated with nCPAP	Kribbs et al., 1993 (89); Kribbs & Dinges, 1994 (32); Chugh et al., 1998 (87); Dinges et al., 1998 (88); Powell et al., 1999 (102); Dinges & Weaver, 2003 (34)
Effects of modafinil on residual sleepiness in OSA patients treated with nCPAP	Dinges & Weaver, 2003 (34)
Effects of bright light	Phipps-Nelson et al., 2003 (129)
Sleep history and apnea severity in commercial truck drivers	Pack et al., 2002 (122)
Effects of alcohol	Powell et al., 1999 (102); Powell et al., 2001 (101)
Sedating effects of melatonin	Graw et al., 2001 (123)

ular reference to drowsy driving, in order to establish that the PVT is sensitive to types of performance used in everyday functioning (i.e., ecological validity), and that PVT metrics can be meaningfully translated in “real world” terms. To set the validity of the PVT as a neurocognitive measure of sleep deprivation, we first review theoretical perspectives on the cognitive effects of sleep loss.

V. Theories of How Sleep Loss Affects Cognitive Functions

There have been many descriptive hypotheses, but remarkably few theories to explain the cognitive effects of sleep deprivation. Early investigators assumed that because remaining awake for 3 or 4 days was so difficult, the ability to perform neurobehavioral tasks (ranging from finger tapping to IQ tests) should be lost when healthy, motivated persons were deprived of sleep (44). Although the seminal experiment by Patrick and Gilbert (13) reported that 90 hr of continuous wakefulness caused both motor and cognitive deficits in three adults, these findings were not replicated in early-twentieth-century experiments (reviewed in Ref. 2 and 44). Between 1923 and 1934, Nathaniel Kleitman published a series of reports on sleep deprivation (“experimental insomnia”) that were intended to clarify the literature (1,2). Kleitman, and a few associates, remained awake between 60 hr and 114 hr but were unable to provide conclusive evidence that sleep loss eliminated the ability to perform specific motor or cognitive functions, because subjects could often transiently perform at baseline levels even after days without sleep. This ultimately led some investigators to assert that the neurocognitive effects of sleep deprivation were primarily to reduce motivation to perform and not cognitive lesions (reviewed in Ref. 3). In subsequent years, refinements were made in experimental measures, cognitive tasks, and statistical analyses, which resulted in less extreme theoretical perspectives on the neurocognitive effects of sleep loss—fewer investigators felt the evidence supported either cognitive lesion hypotheses or motivation hypotheses. Rather, these perspectives were replaced by theories based on evidence that suggested sleep deprivation induced increasing cognitive variability (and hence unpredictability) in specific human neurobehavioral functions.

A. Response Blocks and the Lapse Hypothesis

The history of cognitive performance testing in human sleep deprivation is marked by several theoretical approaches. As described earlier, early investigators (e.g., Refs. 2,13) initially adopted a functional lesion framework. The possibility that sleep loss left no specific cognitive lesion, but markedly affected variability or stability of responding took decades to establish. The bias that sleep loss must produce cognitive lesions to have functional significance affected test design and, importantly, interpretation of experimental results. A particularly clear example of

the latter can be found in Kleitman's (2) exhaustive review of sleep deprivation experiments up to 1963. In describing a seminal experiment by Warren and Clarke (37), who studied four subjects during 48–65 hr of wakefulness, Kleitman stated, "The results were largely negative, as were our own by the same technique" (2, p. 225). However, a reading of the original published report by Warren and Clarke (37) reveals that their results were far from negative (3). Rather, their work, inspired by Bills' research on "mental fatigue" (38–40), laid the foundation for a new theoretical framework. Notably, Bills was not studying the effects of sleep deprivation, but rather, minute-by-minute changes in the performance of non-sleep-deprived subjects. Bills observed an increase in "blocks," defined as "a pause in responses equivalent to the time of two or more average responses" (39, p. 231), with time-on-task. Warren and Clarke (37) applied Bills' block-recording system, and found that while subjects were capable of baseline performance levels while fatigued, they demonstrated increasing numbers of these performance "blocks" as sleep loss progressed. They noted that sleep loss did not produce complete destruction of the aspects of cognitive function they measured, but instead resulted in moment-to-moment performance variability. More than a decade later, Bjerner (41) showed that "blocks" were accompanied by distinct changes in brain activity (EEG) and eye movements (EOG), although he argued these were not "microsleeps." A decade after that observation, in the 1950s, investigators at Walter Reed Army Research Institute (43) hypothesized that the unevenness of performance in sleep-deprived subjects was due to "lapses" [another word for "blocks" (3)], caused by "microsleeps" in EEG and EOG changes.

For approximately two decades, the "lapse hypothesis" was the dominant theoretical explanation for the effects of sleep loss on cognitive performance. In their seminal monograph, Williams, Lubin, and Goodnow (43) reported that performance lapses on experimenter-paced RT tasks increased with increasing hours of wakefulness, and that while poorest performance worsened, subjects were still able to perform at almost optimum levels between lapse periods. Thus, the longer subjects remained awake, the more variable their performance became. Importantly, this was observed regardless of the type (simple vs. choice) or duration (10 vs. 30 min) of RT task, and whether or not subjects were provided with performance feedback.

PVT Performance Reveals Lapses

Psychomotor vigilance task performance is exquisitely sensitive to lapses as classically defined by Bills (39), Warren and Clarke (37), Bjerner (41), and Williams et al. (43). Figure 1 displays consecutive individual reaction times during a 10-min PVT task from a single subject at 12, 36, 60, and 84 hr of wakefulness during an 88-hr total sleep deprivation protocol (44). After 12 hr of wakefulness, responses were maintained at a fast and consistent level. In contrast, much longer responses become evident in PVT trials undertaken as time awake increased. The lapses (conventionally defined at $RT \geq 500$ msec) demonstrated not only

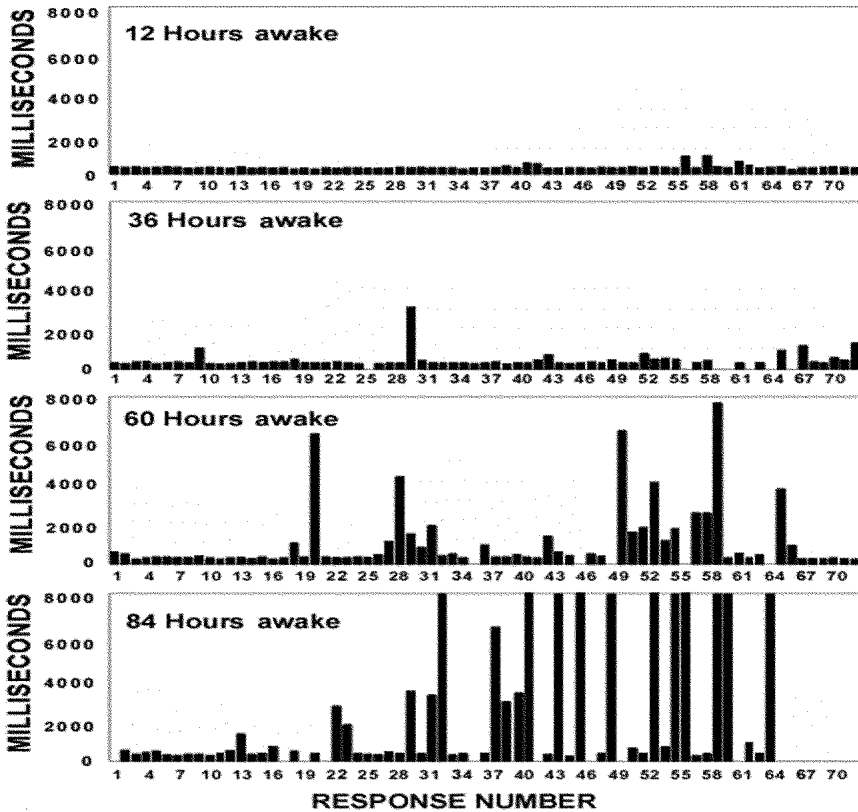


Figure 1 Individual PVT reaction times (msec) for a representative subject undergoing 88 hr (3.67 days) of total sleep deprivation. Reaction times are from the 10-min visual PVT test bouts at 20:00 on each day of deprivation at 12, 36, 60, and 84 hr of sustained wakefulness. Reaction times after presentation of each stimulus are represented by black bars. Blank spaces between reaction times represent false starts (errors of commission). Reaction times > 500 msec are termed performance lapses, or lapses in attention. After 12 hr of wakefulness, reaction times were comparable across the test bout, with no false starts. At 36 hr of wakefulness, there were occasional lapses in attention (RT > 500 msec), with some false starts near the end of the test bout. After 60 hr awake the frequency of performance lapses was increased a few minutes into the performance bout. At 84 hr of sustained wakefulness, there were significantly more lapses of attention, with RTs > 8000 msec, and a greater incidence of false starts. (From Ref. 44.)

increased frequency as hours awake increased, but they also increased in duration, and in variability (from RT to RT). These data clearly indicate that lapsing, which refers to a failure to respond in a timely manner to a stimulus one is expecting, is an easily recognized occurrence in the performance of sleep-deprived subjects. Lapsing has been consistently recorded in studies of sleep deprivation and

performance (e.g., Refs. 13,43–45), making it a primary outcome when using the PVT to assess sleepiness.

VI. Beyond the Lapse Hypothesis

The lapse hypothesis marked a significant, conceptually consolidating advance in theoretical approaches to the effects of sleep deprivation on cognitive performance. However, as a largely descriptive explanation of performance during sleep loss, the lapse hypothesis never rose to the level of a theory (i.e., no conceptual basis for lapsing was proffered). It also quickly became clear that the lapse hypothesis failed to account for several aspects of cognitive impairment commonly seen in sleep-deprived individuals. In a series of articles, Kjellberg (46–48) concluded that lapsing was not an adequate explanation for performance impairment during sleep loss. He suggested that lapses were not discrete periods of lowered arousal, but rather that sleep loss lowered arousal, which, after reaching a certain threshold level, resulted in a performance lapse. He concluded therefore that sleep loss resulted in changes to other aspects of performance. Indeed, at least three phenomena are beyond the explanatory scope of the lapse hypothesis. These include optimum response shifts, time-on-task effects, and the increase in errors of commission (false responses) during extended wakefulness (for thorough reviews on this topic the reader is directed to Refs. 3,46–49).

A. Response Slowing

The lapse hypothesis explicitly predicted that responses between lapses would be normal, which has not proven to be the case (3,5). In addition to performance lapses, Kjellberg (47) noted a slowing of reaction times that was independent of lapsing. Response slowing, a phenomenon recognized by Williams and colleagues (43), has been experimentally demonstrated using 10-min simple reaction time tasks. For example, Lisper and Kjellberg (50) demonstrated that the fastest 25% of reaction times on an auditory RT task were impaired during 24 hr of sleep deprivation. Similarly, during 54 hr of sustained wakefulness, Dinges and Powell (6) observed a clear decline in what they termed the “optimum response” domain (fastest 10% RTs) on both 10-min visual and 10-min auditory sustained attention tasks. Psychomotor vigilance task performance has also been found to show similar adverse effects of sleep loss on the fastest RTs (33). [The sensitivity of such brief sustained vigilance tasks to sleep loss also showed that Wilkinson’s (8) claim in the 1960s that only long-duration (e.g., 40–60 min) vigilance tasks would be sensitive to sleep loss, was incorrect.]

Time-on-Task Decrements

A second observation that cannot be explained by the lapse hypothesis is the well-documented time-on-task decrement (5,6,32,44), which refers to systematic dete-

rioration in performance as a function of increasing duration of a cognitive task. Kleitman referred to this effect as a loss of endurance (2). In an experiment using a color-naming task, he observed that during sleep deprivation, significant slowing of response time and increases in errors could be seen as the task duration was extended from 1 to 12 min (103). He observed that during sleep deprivation most abilities could be maximally utilized by a “new effort” but that “the effect of increased effort disappeared when the test became one of endurance” (103, p. 150).

PVT performance in sleepy individuals frequently shows the effects of time-on-task on lapse rates at any severity of experimental sleep deprivation (e.g., see Fig. 1). Sleep deprivation can markedly worsen this “fatigue” effect in PVT performance, regardless of whether the elevated sleep pressure is experimentally induced (5), or through sleep disorders such as the obstructive sleep apnea syndrome (32). Thus, cognitive performance becomes more variable with both time awake *and* time-on-task (44).

Furthermore, it appears that time-on-task effects are not limited to immediate observable deterioration across single trials. A sleep deprivation experiment involving repeated PVT performance assessments revealed a “cost” associated with completing a longer-duration PVT trial. That is, extending the time-on-task (duration) of the PVT affected performance during subsequent trials. Figure 2 (data from Ref. 51) displays the performance of subjects who completed either a 10-min PVT (lower-workload group) or 20-min PVT (higher-workload group) every 2 hr during 40 hr of continuous wakefulness. Results from the *first* 10 min of the 20-min PVT group were compared to the results from the 10-min PVT group. Figure 2 illustrates a separation in PVT performance scores for the two groups after approximately 22 hr awake (i.e., from 08:00 to 12:00 and 16:00 to 20:00), indicating greater impairment in the high-workload group (i.e., 20-min PVT). Thus, higher cumulative workload further increased PVT deficits as sleep deprivation progressed. This suggests that there is a neurobiological cost to performing the PVT that becomes more evident as sleep loss progresses. It also suggests that in addition to factors such as time awake and time-on-task, prior workload should be accounted for when considering an individual’s impairment level during sleep loss.

Errors of Commission

A third limitation of the lapse hypothesis involved its inability to account for errors of commission, which involve responses when no stimulus is present (44). Studies have demonstrated a higher incidence of errors of commission with increasing hours of wakefulness (42,44). Importantly, errors of commission show the same profile of circadian-modulated increases across days of total sleep deprivation that is seen for errors of omission (i.e., lapses) (44). Such errors occur as premature responses during PVT performance, and are represented by blank spaces in between RT bars in Figure 1. After 12 hr of sustained

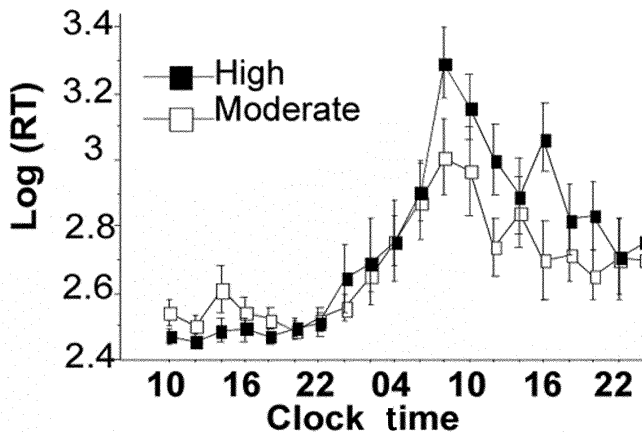


Figure 2 Mean PVT reaction times (log msec \pm sem) across 40 hr of total sleep deprivation. Data from the first 10 min of a 20-min visual PVT performance bout—high workload—are represented by the closed squares; data from the entire duration of a 10-min visual PVT performance bout—low workload—are represented by the open squares. The reaction time data (log transformed) were compared between the two groups using a mixed-model (workload by time awake) ANOVA. Reaction times during the first 10 min of the high-workload performance tests were significantly higher than those in the low-workload performance tests ($F_{9, 456} = 19.87, p < .001$). In addition, a decrease in reaction times across the 40 hr of wakefulness was evident in both workload groups ($F_{9, 456} = 2.17, p < 0.003$). (From Doran et al., 2000.)

wakefulness this subject experienced no false starts in a single 10-min PVT trial, compared to seven premature responses after 36 hr, five after 60 hr, and 15 after 60 hr. Of note, a time-on-task effect is also evident, with a greater number of false starts occurring later during each trial—a profile similar to that found for lapses.

State Instability

As described above, both errors of omission involving performance lapses (i.e., failing to respond in a timely manner to a stimulus that is present) and errors of commission involving false starts (i.e., responding when no stimulus is present) have been experimentally demonstrated to increase during sleep deprivation (42,44). Figure 3 shows the profiles of each of these two types of cognitive errors in PVT performance across an 88-hr period of continuous wakefulness, relative to a control condition involving a 2-hr sleep opportunity every 12 hr across the vigil (44). The ability to engage in behavior to compensate for the effects of sleep loss has been observed by numerous investigators (2,13,52). We have suggested that the concomitant increase in errors of commission reflects an increased compensatory effort (albeit inefficient) in reaction to the effects of sleep loss (44). If

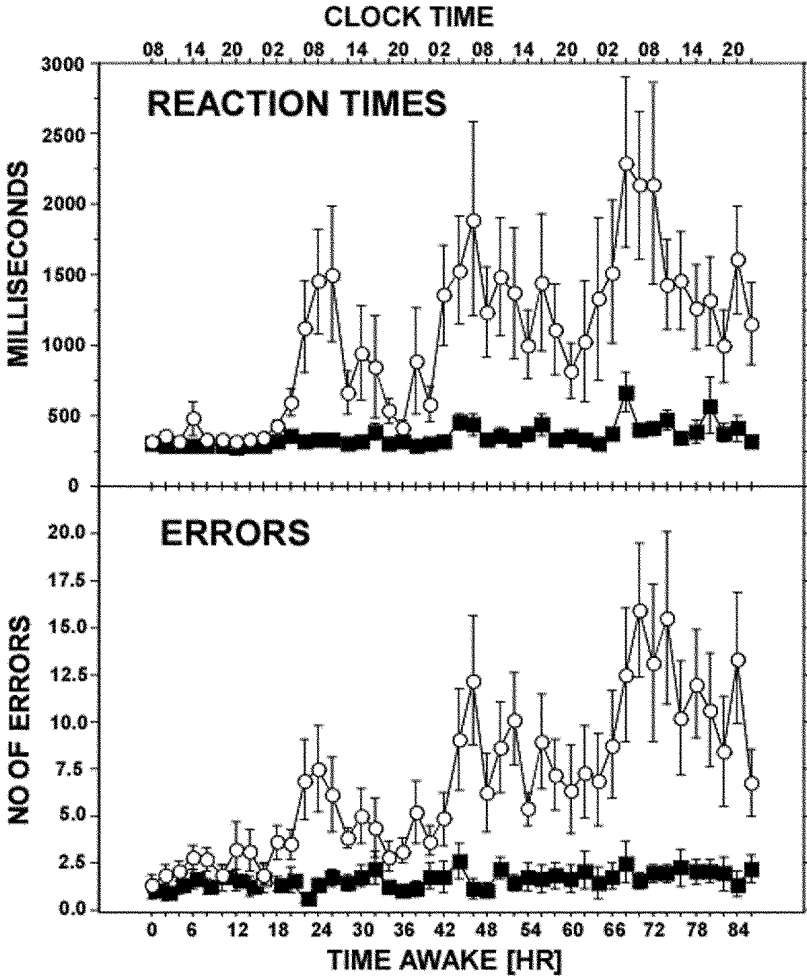


Figure 3 Mean PVT reaction times (msec) and false starts (errors of commission) during 88 hr of total sleep deprivation and 88 hours of sleep deprivation with two 2-hour nap opportunities each day. Subjects in the total sleep deprivation (TSD) group ($n = 13$) are represented by the open circles. Subjects in the 88-hr sleep deprivation plus two 2-hr nap opportunities (NAP) group ($n = 15$) are represented by the closed squares. Nap opportunity periods were at 02:45-04:45 and 14:45-16:45 each day. The top panel illustrates mean reaction times (\pm s.e.m.) for each test bout across the experimental protocol. Subjects in the NAP group demonstrated little variation in reaction times across the experimental period, while subjects in the TSD group experienced significant impairment in performance, reflected in the increasing reaction times as time awake increased, with circadian variation in performance capability evident. The bottom panel illustrates mean number of errors (\pm s.e.m.) per test bout across the experimental protocol. A similar pattern of performance degradation in this variable was evident for both the NAP and TSD groups. (From Ref. 44.)

this hypothesis is correct, increasing motivation and effort to perform well during sleep deprivation may actually have the unintended consequence of producing additional errors of another type.

To explain the neurocognitive effects of sleep loss, what we term the *state instability* hypothesis (44) was developed after observing: (1) the emergence of both errors of omission and errors of commission during sleep loss; (2) their covariation over time in a manner consistent with the interaction of the homeostatic drive for sleep and endogenous circadian pacemaker (33); and (3) their increase with time-on-task. According to this hypothesis, an individual performing under the stress of an elevated homeostatic sleep drive may be overcome by sleepiness to the point of falling asleep uncontrollably while performing, which in turn leads to compensatory effort to resist the rapid and brief intrusions of sleep. Consequently, the state instability means that at any given moment in time the cognitive performance of the individual is unpredictable, and a product of interactive, competing neurobiological systems (connections, receptors, molecules) mediating sleep initiation and wake maintenance. In this conceptualization, the neurocognitive effects of sleep loss are but one manifestation of the broader neurobehavioral consequences of both sleep initiation and wake maintenance neurobiology co-occurring, with inadequate reciprocal inhibition between them. Theoretically, the state instability concept suggests that there are multiple, parallel neurobiological mechanisms by which waking and sleep states can interact. This is consistent with the fact that there are a growing number of candidate molecules that could be involved in the co-occurrence of sleep and waking (53).

A focus of both the state instability and the lapse hypothesis is the use of sustained attention tasks as sensitive assays of cognitive performance variability during sleep deprivation. The difference between the two viewpoints is in the explanation for the variability. The lapse hypothesis contends that performance during sleep deprivation is essentially “normal” until it becomes disrupted by lapses (brief periods of low arousal) (43). In contrast, according to the state instability hypothesis (51), performance variability is produced by the influence of homeostatically controlled sleep initiating mechanisms on the endogenous capacity to maintain alertness, and therefore utilize executive attention (prefrontal cortex). With its relatively high signal rate (input) and utilization of millisecond changes in response time (output), the PVT is designed to be maximally sensitive to state stability-instability during cognitive performance.

VII. Sensitivity of PVT Performance to Neurobiological Causes of Elevated Sleep Drive

The theoretical and practical utility of any putative cognitive test of the effects of sleep deprivation must be grounded in its demonstrated sensitivity to known neurobiological sources of elevated sleep drive. The following sections review the sensitivity of the PVT to such factors.

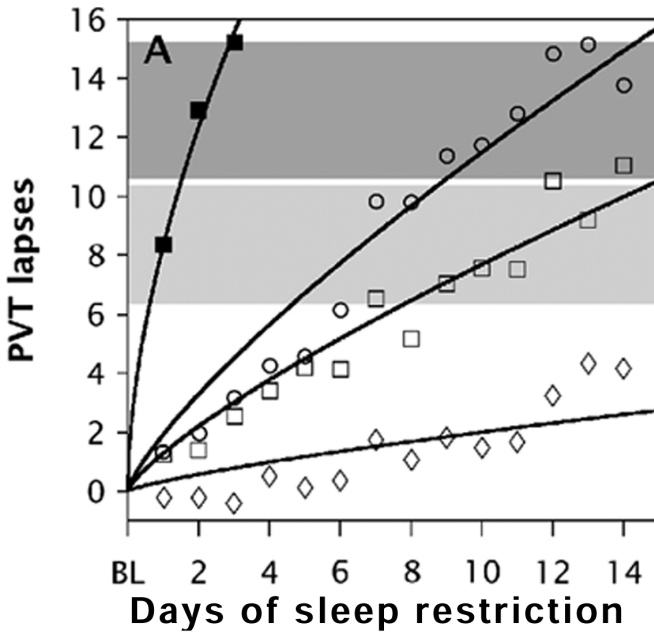


Figure 4 PVT performance responses to varying doses of daily sleep. Mean PVT lapses per day (07:30–23:30), measured at 2-hr intervals, expressed relative to baseline (BL), in subjects randomized to an 8-hr ($n = 9$; open diamond), 6-hr ($n = 13$; open square), or 4-hr ($n = 13$; open circle) sleep opportunity per day for 14 consecutive days or 0-hr ($n = 13$; closed square) sleep condition across 3 days. The curves represent statistical non-linear model-based best-fitting profiles of the PVT performance response to sleep loss. The mean (\pm s.e.m.) ranges of neurobehavioral functions for 1 and 2 days of total sleep deprivation (0 hr sleep) are illustrated by the light and dark bands, respectively, allowing comparison of the 3-day total sleep deprivation condition and the 14-day chronic sleep restriction conditions. (From Ref. 35.)

A. Cognitive Performance Relative to the Two-Process Model of Sleep-Wake Regulation

Changes in performance capability during continuous wakefulness can be conceptualized as a two-process interaction (33), derived from the two-process model of sleep regulation (54). Specifically, sleepiness and performance are influenced by the homeostatic sleep drive (producing monotonic increases in impairment) and by circadian rhythmicity (near 24-hr cycles) (33,58). Daily circadian modulation of neurocognitive performance has been consistently noted since the first studies of sleep deprivation and human performance (2,13).

The interaction between endogenous circadian rhythmicity and homeostatic sleep drive results in a pattern in neurobehavioral functioning such that cognitive performance capability increases across the diurnal portion of each day (despite

increasing time awake), and decreases across the night, reaching a nadir in the first 8 hr of the morning. A less marked and at times ephemeral midafternoon dip in performance has also been reported (55), referred to as the postprandial, or postlunch, dip (56,57). It is consistent with much scientific literature on the endogenous tendency for increased sleep propensity and napping at this time of day (104). Further, with increasing time awake, the escalating drive for sleep amplifies the circadian performance rhythm such that over successive days the level of impairment at the nadir of the rhythm becomes incrementally greater (42,57).

As noted earlier, an effective assay of the cognitive impact of sleep loss should be sensitive to the homeostatic drive for sleep in interaction with the endogenous circadian pacemaker (33,58). From Figure 3, it is evident that the PVT fulfills this requirement, with performance during 88 hr of sleep deprivation demonstrating both a monotonic component to impairment, which increases with increasing time awake, and a rhythmic oscillation in performance, which fluctuates in daily cycles.

Task sensitivity to homeostatic and circadian drives can be even more manifestly illustrated under constant routine conditions. Constant routine protocols allow investigators to study circadian rhythms without masking effects from factors such as sleep, physical activity, meals, light exposure, and social contact that will affect circadian phase markers. Subjects remain awake for longer than 24 hr (often several days), with fixed posture, ambient temperature (approximately 24°C), and lighting (typically <50 lux). Physical activity, meals and social contact are also kept constant (for a review of constant routine procedures, see Ref. 58). Van Dongen and Dinges (33) observed homeostatic and circadian modulations in subjective sleepiness and core body temperature during a 36-hr constant routine protocol ($n = 5$), which were closely reflected by the fastest 10% of PVT RTs. Similarly, a study by Wyatt and colleagues (59) demonstrated the interaction of homeostatic and circadian processes in the modulation of PVT performance. Thus, the psychomotor vigilance task appears to reflect the temporal dynamics of the two endogenous neurobiological systems (process S and process C) controlling daily wakefulness.

B. Chronic Partial Sleep Deprivation

Chronic partial sleep deprivation has been defined as “preventing subjects from obtaining their usual amount of sleep within a 24-hour period” (60, p. 221). A wide range of partial sleep deprivation paradigms have been conducted, including selective deprivation of particular sleep stages (61–63), gradual sleep reduction over time (64), fixed-duration, reduced sleep opportunities in continuous (65) and distributed schedules (65), and situations where the time in bed is specifically reduced relative to the individual subject’s habitual time in bed (29). The time span of these protocols has ranged from 24 hr (66) to 8 months (64).

Long-term investigations of partial sleep deprivation (from 21 days to 8 months) have not found consistent evidence of impairment (26,64,66,67). Such

conflicting results are likely due to inadequacy of measurement outcomes and the lack of appropriate laboratory controls for timing and duration of sleep periods (68). In contrast, more tightly controlled, laboratory-based studies of chronic partial sleep deprivation, using more sensitive cognitive performance outcomes, have found clear evidence of performance impairment (29,35,65,69–71). Importantly, findings indicate that the effects of partial sleep deprivation are cumulative, such that performance and alertness become progressively worse across days of sleep restriction (35,70).

The PVT has been a primary performance assay for demonstrating the cumulative neurocognitive effects from chronic partial sleep deprivation (35,70). For example, in the two largest laboratory-controlled dose-response experiments conducted to date on the neurobehavioral effects of chronic sleep restriction, cumulative increases were evident in the average number of PVT lapses per 24 hr across days of sleep restricted to 3, 4, 5, and 6 hr per night (35,70). Moreover, daily PVT lapse rates increased at a more rapid rate in the reduced sleep conditions. Figure 4 displays the results from the first of these studies, in which subjects were restricted to 4, 6, or 8-hr time in bed for sleep for 14 consecutive days (35). The results were compared to 88 hr of total sleep deprivation. Figure 4 illustrates the dose-response relationship between sleep opportunity and the degree of impairment in PVT performance. Interestingly, this cumulative impairment was found to be almost linear for lapse rates. Further, subjects randomized to the 4- and 6-hr sleep restriction conditions reached levels of impairment equivalent to those of subjects undergoing 1–2 nights of total sleep deprivation.

In an earlier experiment, cumulative increases in PVT lapses across 7 days of sleep restricted to approximately 5 hr per night (29) were shown to be strongly related ($r = -0.95$) to sleep onset latency as assessed by the Multiple Sleep Latency Test (MSLT) in a nearly identical protocol (72). It appears that PVT performance lapse frequency and the well-validated physiological measure of sleep propensity may reflect the same basic process of escalating sleep pressure with sleep loss.

C. Interventions to Reduce Sleepiness

Interventions such as naps and caffeine to counteract the neurobehavioral effects of sleep loss and sleepiness in healthy adults have been found to improve PVT performance (73–77). The PVT has been successfully used to track the effects of napping in laboratory (77) and operational (31) settings. The use of naps in the laboratory to augment the performance of sleep-deprived subjects is illustrated in the control condition of Figure 3, which shows that PVT errors of omission and commission during 88 hr of sleep deprivation are substantially reduced by 2-hour naps taken every 12 hr.

There is evidence of performance benefits from combining naps and caffeine consumption during sleep loss (79,80). Although these studies did not use

the PVT, performance on the PVT has been found to be sensitive to sleep inertia, which refers to feelings of grogginess and severe cognitive impairment after awakening from deep sleep. A potential reason for the advantage of combining naps and caffeine in sleep-restricted subjects has been demonstrated in a study by Van Dongen and colleagues (78), which revealed that sustained low-dose caffeine intake significantly reduced the effects of sleep inertia on PVT performance at awakening from naps during prolonged partial sleep deprivation.

Exposure to bright light during periods of sleep loss has also been used to attenuate performance degradation (e.g., Refs. 81–83). A modified version of the PVT has been used to assess the effects of countermeasure strategies involving combinations of bright light and caffeine (84) during 45.5 hr of total sleep deprivation. PVT performance was sensitive to differences between treatment groups, with best performance achieved by the bright-light/caffeine group, followed by the dim-light/caffeine group, with worse performance in the two groups who received placebo. Thus, it appears that in addition to showing the effects of sleep deprivation in healthy adults, PVT performance is sensitive to the alertness-promoting effects of bright light, naps, and caffeine in sleepy subjects.

D. Obstructive Sleep Apnea Syndrome

Psychomotor vigilance task performance has also been shown to be sensitive to reduced behavioral alertness associated with obstructive sleep apnea syndrome (OSAS), and the efficacy of interventions for OSAS. Performance of patients with OSAS is impaired on tasks that rely on the ability to sustain attention (85,86). As a measure of behavioral alertness, PVT performance has been demonstrated to be a sensitive method for assessing the attentional capability of patients with OSAS (32,87,88). Kribbs and colleagues (89) found that PVT performance and sleepiness, measured by the MSLT, both reflected the benefits of CPAP use (reduction in respiratory events during sleep). Similarly, the PVT has been used to demonstrate the positive effects of modafinil (a wake-promoting compound) on the capacity to sustain attention in a group of OSAS patients (34).

VIII. PVT Sensitivity to Other Factors Relevant to Sleepiness and Sedation

The PVT has also proven to be sensitive to other factors associated with neurobehavioral vulnerability to sleep loss and its consequences. The following sections review a few of the more salient ones.

A. Interindividual Differences in Response to Sleep Loss

An important phenomenon that has been demonstrated using PVT performance results is that increasing time awake (sleep deprivation) in healthy adults is asso-

ciated with increasing between-subject differences in PVT performance (3,44,68). This can be seen in the 88-hour total sleep deprivation protocol (44) shown in Figure 3, as evidenced by increasing error bars across time. Figure 5 displays PVT reaction time means plotted against standard deviations for 13 subjects from that experiment (44). Linear regression lines were fit for each individual. The different lengths of the regression lines in Figure 5 indicate that the magnitude of impairment differs between individuals. Interindividual differences in susceptibility to the impairing effects of sleep deprivation are an important area of interest, receiving increasing attention. In one of the only systematic studies on the topic to date, Van Dongen and colleagues (68) investigated PVT lapses in the same individuals who underwent 36-hr periods of total sleep deprivation on two separate occasions. Results revealed that interindividual differences accounted for 78.9% of the variance in PVT lapses performance, demonstrating reliable trait-like differences in vulnerability to sleep deprivation as measured using PVT performance (i.e., stable differential vulnerability to the cognitive effects of sleep deprivation).

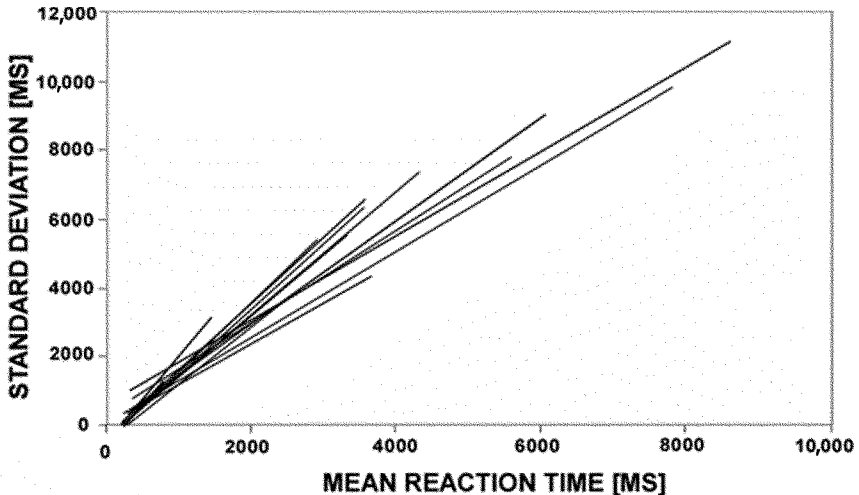


Figure 5 Least-square regression lines fit for the linear relationship between mean and standard deviation of PVT reaction times (msec). Data are from $n = 13$ subjects undergoing 88 hr (3.67 days) of total sleep deprivation. This figure illustrates that while all subjects experienced a decline in neurobehavioral performance on the PVT, as illustrated by increased reaction times when responding to the visual stimuli, there is a significant degree of interindividual variability in the magnitude of neurobehavioral impairment, evident by the differing lengths of the lines fit to the data. (From Ref. 44.)

B. PVT and “Real World” Performance: Drowsy Driving

When interpreting results of laboratory-based cognitive tests, it must be remembered that they are not absolute indicators of “real world” performance. Although a given cognitive task may track the direction of changes in functioning as sleep deprivation increases, such tasks often do not permit a direct extrapolation to estimates of ability to perform everyday tasks. The PVT has some advantages in this regard, because it taps the ability to sustain attention and respond quickly to salient signals—features of a great many real-world tasks. In this sense it has high ecological validity, especially for tasks that require paying attention and responding in a timely manner (e.g., operating any transportation vehicle; monitoring radar, x-ray, and surveillance equipment; etc.).

Motor vehicle operation is a task performed by the vast majority of adults. It is heavily dependent on the ability to sustain attention and respond quickly, and it can have serious medical and economic consequences if not performed reliably. Transport accident data suggest that after controlling for traffic density, there are two main determinants of motor accident frequency: time of day and time spent driving (90). Specifically, accident frequency increases with time spent driving (i.e., time-on-task), and it is temporally distributed in a bimodal fashion. When adjustments are made for exposure (i.e., the number of motor vehicles on the road), crashes cluster disproportionately between 00:00 and 07:00, with a secondary peak around 15:00 (91–93). If inability to sustain attention were a major contributor to such accidents, it would be expected that a similar distribution would be found for prolonged performance lapses on the PVT. Indeed, this is the case. A study investigating PVT lapses greater than 30 sec, which are evidence of severe drowsiness attacks while attempting to perform, found that these attacks had a distribution similar to that of roadway crashes (see Fig. 6), with peaks of occurrence at 07:00 and at 16:00 and increasing prevalence with time-on-task (94).

From data described earlier (92,94), it is apparent that PVT lapses can be considered a temporal indicator of vulnerability to hypovigilance and sleepiness attacks of the kind that can occur during drowsy driving. Furthermore, PVT sensitivity allows a more fine-grained analysis of the circumstances leading up to a 30-sec sleep attack (95). Figure 7 displays PVT reaction times in the 6 min leading up to the first uncontrolled sleep attack in two experiments. A clear increase in reaction times (i.e., lengthening of lapse durations) prior to a 30-sec sleep attack is evident (95). These results suggest that such severe sleep attacks are not simply periods of nonresponding that punctuate normal alert performance; rather, a period of escalating impairment is evident during the min leading up to a 30-sec lapse. Not only is there temporal evidence that PVT lapses may be indicative of sleep attacks, but there is extensive evidence of a close correlation between PVT lapses and percent of time in slow eyelid closures while driving (96–98). The risks posed by increasing periods of slow eyelid closures while driving are obvious.

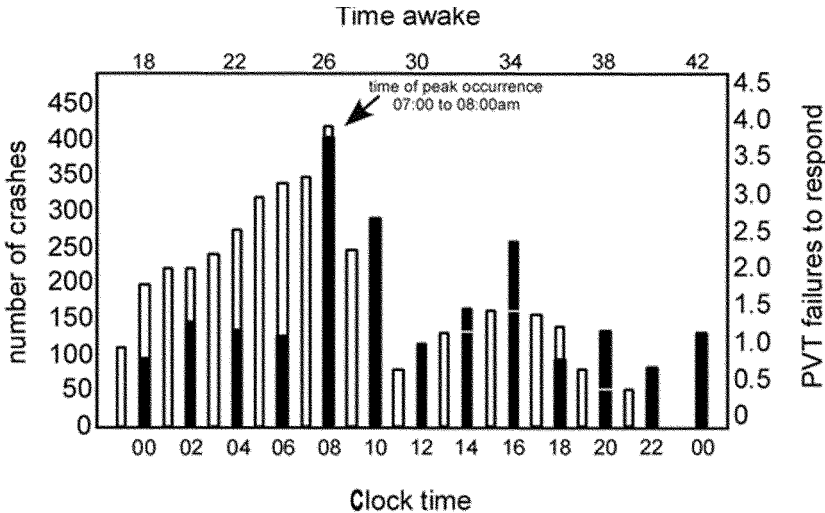


Figure 6 Frequency histogram for time of day of motor vehicle crashes and fall-asleep attacks on the PVT. The open bars represent the 60-min frequency of 4333 crashes in which the driver was judged to be asleep but not intoxicated across the 24-hr day; and the solid bars represent fall-asleep attacks (failure to respond for 30 sec on the PVT) in $n = 14$ subjects, measured at 120-min intervals across 42 hr of total sleep deprivation. Both the fatigue-related crashes and fall-asleep attacks follow an equivalent temporal profile across the day, with occurrences increasing across the nocturnal period, and peaking between 07:00 and 08:00. (Adapted from Refs. 92 and 94.)

C. Quantifying Impairment: PVT Performance and Alcohol

Attempts have been made to assess the magnitude of performance decrement during sleep loss using positive controls with widely accepted and quantified levels of impairment. Since the impairing effects of alcohol intoxication are readily recognized via public policy and legal regulation, studies have been conducted comparing the effects of sleep deprivation and alcohol.

Experimental comparisons between the performance effects of alcohol intoxication and sleep deprivation have found quantitative and qualitative similarities for numerous performance parameters including unpredictable tracking (9,99), vigilance (100), and response latency in a logical reasoning task (9). These studies have suggested that after 17–18 hr of sleep deprivation, performance is equivalent to (or greater than) that of a person with a blood alcohol concentration (BAC) of 0.05% (the legal driving limit in Australia) (100), and that after 20–25 hr awake, performance impairment is equivalent to (or greater than) a BAC of 0.10% (the legal driving limit in many states in the United States) (9).

Similar results have been found for PVT performance. Powell and colleagues (101) measured PVT performance after acute (1 night without sleep) or

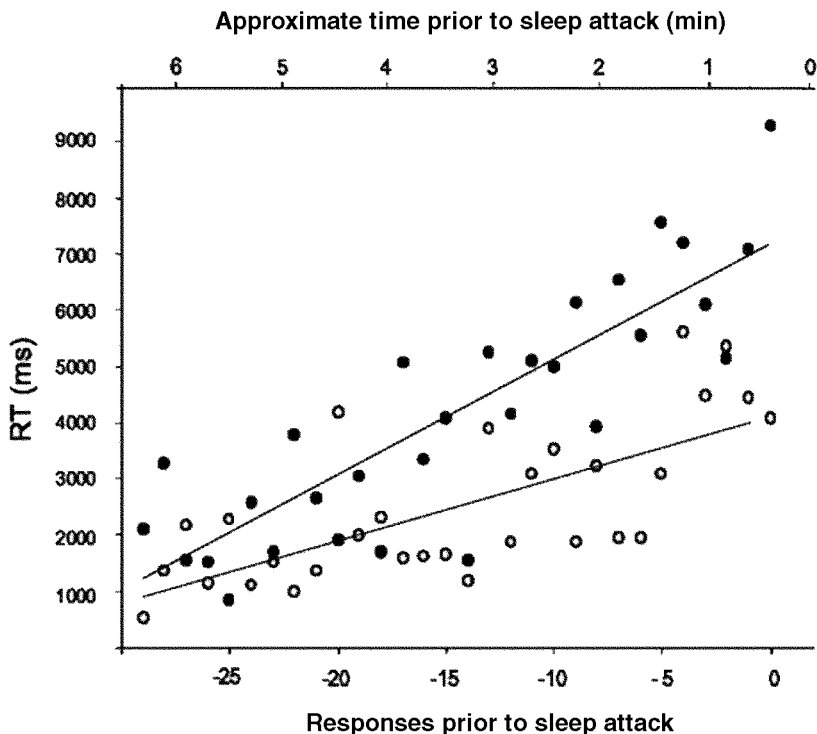


Figure 7 PVT reaction times prior to the first uncontrolled sleep attack during total sleep deprivation. Fourteen subjects completed 42 hr of total sleep deprivation and completed a 20-min PVT every 2 hr (represented by the closed circles); 19 subjects completed 88 hr of total sleep deprivation and completed a 10-min PVT every 2 hr (represented by the open circles). The number of test bouts (up to 30) prior to an uncontrolled sleep attack (failure to respond for 30 sec on the PVT) is represented on the bottom abscissa, with time prior to the sleep attack (up to 6 min) represented on the top abscissa. In both subject groups a progressive decline in performance on the visual PVT was evident within minutes of an uncontrolled sleep attack on console. This study also demonstrated an increase in subjective sleepiness (measured using the Stanford Sleepiness Scale) in the test bouts prior to the one in which the first sleep attack occurred. Taken together, these findings suggest that even a very sleepy subject cannot fall asleep while performing computerized tasks without some levels of awareness. (From Ref. 95.)

partial (2 hr less sleep per night for 1 week) sleep loss compared to alcohol intoxication (mean concentration = 0.089 g/dL). They found that performance impairment on the PVT was not significantly different in the alcohol and sleep deprivation intervention groups, and the magnitude of impairment was similar. They also compared healthy subjects given alcohol with patients with untreated obstructive sleep apnea on measures of psychomotor vigilance performance

(102). Those with sleep-disordered breathing had worse RT performance than subjects with blood alcohol concentrations of 0.057% or greater.

Such research suggests that while the PVT is extremely sensitive to sleep deprivation, this does not negate its relevance to “real world” performance risks. That is to say, PVT impairment from sleep loss may be an indicator of “real world” task decrement to a degree that may be considered of operational (and legal) concern.

IX. Summary and Conclusions

Accurate assessment of neurobehavioral performance capability during sleep deprivation protocols requires cognitive performance assays to be: (a) indicative of a fundamental aspect of waking cognitive function; (b) easily performed; (c) minimally affected by aptitude and learning; (d) as brief as possible; (e) valid and reliable; (f) sensitive; and (g) able to provide meaningful outcome variables that can be easily interpreted. In this chapter we have reviewed the evidence that the psychomotor vigilance task meets these requirements.

The PVT was developed as a neurocognitive test of behavioral alertness to track temporally dynamic changes induced by interaction of the homeostatic drive for sleep and endogenous circadian pacemaker, focusing on assessing ability to sustain attention and respond in a timely manner to salient signals. Repeated administration (every 2 hr during waking periods) of the PVT to subjects allowed 8 hr sleep per night for 5 nights demonstrated the reliability of performance on this task across experimental days. Intraclass correlation coefficients indicated maximal reliability for both number of PVT performance lapses and median response times.

The PVT has been tested under a number of conditions recognized to induce neurocognitive deficits due to sleep loss, including total sleep deprivation, chronic partial sleep deprivation, and sleep fragmentation. Irrespective of the mode of sleep loss, results of extensive experiments on PVT performance have demonstrated that the task is capable of capturing the effects of sleep loss on stability of sustained attention, and that it can reliably reveal the accumulation of cumulative state instability in chronic sleep loss. As an assay of the neurocognitive effects of sleep loss, the PVT has also been used to assess the effectiveness of countermeasures to sleep loss (e.g., naps, caffeine, modafinil). The PVT has also been used to quantify daytime functioning levels in patients with OSAS, in relation to drowsy driving and in alcohol intoxication protocols.

Taken together these studies illustrate the efficacy and sensitivity of the PVT in the assessment of neurocognitive performance in a number of experimental, clinical, and operational paradigms. Because of its high degree of reliability, validity, lack of dependence on aptitude, and ability to be repeatedly administered, the PVT can be used to quantify the effects of sleep loss, and other manipulations, on neurobehavioral capability across a number of days. Studies

have suggested that PVT performance has relevance to “real world” risks, such as drowsy driving and alcohol impairment.

Acknowledgments

The substantive evaluation on which this chapter was based was supported by NIH grant NR04281; Air Force Office of Scientific Research grant F49620-00-1-0266; NASA Cooperative Agreement NCC 9-58 with the National Space Biomedical Research Institute and the Institute for Experimental Psychiatry Research Foundation.

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