Activity, Arousal, and the MSLT in Patients with Insomnia

Michael H. Bonnet PhD and Donna L. Arand PhD

Dayton Department of Veterans Affairs Medical Center, Wright State University, and Kettering Medical Center

INTRODUCTION

MANY METHODS HAVE BEEN DEVELOPED TO MEASURE SLEEPINESS. The most sensitive may be the Multiple Sleep Latency Test (MSLT), which measures the EEG latency to sleep onset several times during the day. The MSLT has been shown to differentiate several types of partial and complete sleep deprivation occurring on either an acute or chronic basis. The test has also have longer MSLT latencies. It was hypothesized that insomnia patients, who are at a higher state of physiological arousal, would be unable to relax while lying in bed and watching TV and therefore would have relatively longer sleep latencies in naps following TV watching (due to inability to relax) as compared to walking.

Design: Twelve patients with psychophysiological insomnia took Multiple Sleep Latency Tests after either watching television for 15 minutes or after a 5-minute walk following baseline, sleep deprivation, and recovery sleep conditions.

Results: Sleep latencies were significantly longer following the walk as compared to watching TV (11.9 vs. 6.9 min. respectively). Sleep latencies were 13.4 and 3.8 min. following baseline and sleep deprivation conditions. Heart period, used as a measure of physiological arousal, was significantly elevated throughout naps following the walk as compared to naps following TV viewing. Heart period was also significantly correlated with nap sleep latency.

Conclusions: The insomnia patients in this study had significantly increased arousal, as measured by heart rate, and significantly longer sleep latencies after walking as compared to resting. The magnitude of these changes was similar to that seen in normal subjects in a previous study. These data, in concert with previous work, support the contention that measured sleep tendency is a combination of sleep drive and level of central nervous system arousal, where arousal has both state and trait components.

Key words: Insomnia; sleep; sleep deprivation; arousal; mslt; heart rate; sleep disorders; hyperarousal; partial sleep deprivation; sleepiness

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Correspondence to: Michael H. Bonnet, PhD (151N), Dayton Department of Veterans Affairs Medical Center, 4100 W. Third Street, Dayton, OH 45428, Tel: (937) 267-3910, Fax: (937) 267-3910, Bonnet.Michael@DAYTON.VA.GOV
Just as the arousal caused by stress can make it more difficult for anyone to fall asleep, the physiological arousal associated with the walk produced longer MSLT latencies.

In patients with chronic insomnia, a constant higher level of arousal results in reports of chronic difficulty with sleep. If this arousal limits ability to fall asleep, then patients with insomnia who are in bed watching TV may not be able to relax and fall asleep as quickly as normals did in the recent study. Similarly, patients with insomnia put in a physiologically arousing situation, such as walking, may not display as large an increase in arousal as normals if their baseline level of arousal is already elevated. If either of these events occur in insomnia patients, nap latencies after watching TV or walking would be predicted to display smaller (or no) differences.

Under controlled conditions, heart rate has commonly been used as a physiological definition of arousal level. For example, studies have shown that heart rate is higher in people who are sitting up (as compared to lying down), standing, performing tasks such as mental arithmetic or involved in real world activities varying in difficulty. In the recent study of MSLT after relaxing or walking, heart rate was increased during naps following the walk; and in another recent study the degree of heart rate increase was significantly correlated with latency to fall asleep after sitting up or standing.

In the current study, the relative role of sleepiness and arousal in determining objective sleep latency in patients with insomnia was measured by independently varying arousal level (activity) and sleepiness (sleep deprivation). Heart rate was measured during sleep latency tests in this experiment as an independent indicator of residual arousal from preceding activity. It was hypothesized that because patients with insomnia have elevated levels of arousal, the expected differences in sleep latencies following walking would be reduced or absent compared with those following inactivity (watching television). It was also hypothesized that sleep deprivation would decrease sleep latency in both conditions.

**METHODS**

**Subjects**

Subjects (Ss) were required to be otherwise healthy, 18–50 year-old males and females. Potential Ss were solicited from sleep center referrals and from ads in the local papers for participants in sleep research. Individuals considered further had a screening questionnaire which indicated 1) that they had a sleep problem; 2) that it took them 45 minutes or more to fall asleep at least four nights each week or that they were awake for 60 minutes or more each night after falling asleep for at least four nights each week; and 3) that this condition had existed for at least one year.

Potential insomniac patients who indicated excessive caffeine consumption (more than 250 mg of caffeine per day), who were using psychoactive medication or drugs, or who had completed a drug or alcohol abuse program within the previous year, were excluded. Ss with a history of depression or psychiatric hospitalization within the previous year were excluded. Potential Ss who had histories strongly suggestive of circadian desynchrony (e.g., shift workers), sleep apnea, or periodic leg movements were excluded.

Subjects meeting the above criteria were invited to participate in the study after completing an informed consent and practicing on computer tests and questionnaires to be used in the study.

**Design**

After completing the consent, subjects were scheduled for an adaptation night. On this night, a standard clinical polysomnogram, including two eye channels, central and occipital EEG channels, chin and leg EMG channels, EKG, airflow, chest movements, and SaO2 was performed. To qualify for the study, insomnia patients were required to have an EEG sleep latency greater than 30 minutes or to have a sleep efficiency of less than 85%. Subjects with an apnea/hypopnea index greater than 10 or a periodic leg movement arousal index greater than 10 were disqualified.

Ss meeting the above criteria were invited to participate in the study proper.

All subjects were assigned their own room for the course of the study. Each room contained a standard hospital bed and furniture including a desk with an Apple IIGS computer. Subjects participated in the study in groups of 1–2 individuals. Subjects completed tests and questionnaires at their individual computer workstation in their room under technician observation via video monitors. Meals and breaks were scheduled in another area of the laboratory, which was also within technician observation. Ss performed computer tests, completed an MMPI and a sleep history, and were fed the same daily menu of food prepared at the lab during the day. Caffeinated beverages were not available. Subjects usually did not leave the lab during this day and did not engage in vigorous activity.

All protocol and nap times cited in this paper were specified for a subject who normally went to bed at 23:00 and awoke at 07:00. For subjects who normally went to bed somewhat later (or earlier), bed time and wake up time were adjusted to approximate normal weekday times as determined from questionnaires completed by the patients (bedtimes in this study ranged from 22:00 to 02:00). Testing and sleep latency tests were correspondingly moved to maintain similar circadian timing for all Ss on all nights.

Subjects spent three consecutive nights and the following days in the laboratory. The initial night was a baseline
night. On the second night, Ss remained awake in the laboratory (total sleep deprivation). On the third night, Ss were allowed to sleep normally. During each day, all Ss remained at the laboratory where they took a series of eight naps and performed brief computer tests. The daytime schedule is summarized in Table 1. During each day, Ss were fed standard meals. The daytime protocol was divided into four blocks (see table). EEG and EKG were recorded throughout all of the blocks except when Ss were walking. EEG was monitored to assure wakefulness throughout all periods except the MSLT (when sleep was allowed). Each block started with 10 minutes of standard computer tests and was followed by either 15 minutes of watching TV while lying in bed or five minutes of walking around the hospital building. Immediately after the walk or at the end of 15 minutes of television, MSLT calibrations were performed. Lights were turned out and MSLT tests began in three to four minutes. After the MSLT, Ss had a 15-minute break and then began the computer test cycle again.

After the computer tests, Ss received the pre-MSLT activity that they had not received during the prior MSLT.

Performance and mood were assessed with a battery of measures including the digit symbol substitution task from the WAIS (5 min.23), subjective sleepiness (10-point visual analog scale), Profile of Mood States (POMS), and oral temperature. In this study, the tests were used primarily to provide a period of consistent control activity. Except for subjective sleep report measures, the data were not analyzed.

Sleep recordings (LE - A2, RE - A2, C3 - A2, OZ - A1, V5—right clavicle, and time code) were made during nocturnal sleep periods, naps, and MSLT evaluations. All sleep and nap recordings were scored in 30 second epochs using Rechtschaffen and Kales24 criteria.

### Sleep Latency Tests

On each day in the laboratory, half of the subjects had 4 sleep latency tests following 15 minutes of resting wakefulness (watching TV ) at 08:25, 10:45, 13:05, and 15:25; and four sleep latency tests following 5-minute walks at 09:20, 11:40, 14:00, and 16:20. The other Ss had sleep latency tests following 5-min. walks at 08:15, 10:35, 12:55, and 15:15 and following 15 minutes of resting wakefulness (watching TV) at 09:20, 11:40, 14:00, and 16:20. Each test block began with Ss performing 10 minute of computer tests while sitting at a desk in their bedroom. At the completion of these tests, Ss either began a resting wake session or took a walk.

### Resting Wake

For resting wake observations, Ss laid down in bed and watched a TV that was placed in an elevated position at the foot of their bed. The room lights remained on. Ss were told to lie in bed and to stay awake. EEG was monitored continuously for the 15 minutes of the test, and the technician was instructed to interact with the subject if eye closure was noted on the video monitor or if signs of impending stage 1 sleep were noted on the polygraph. At the end of the 15 minutes, the technician entered the room, told the subject the nap would be next, and turned off the room lights. Sleep latency test calibrations and the sleep latency test followed immediately.

### Five-Minutes Walk

Subjects were instructed to take a five-minute walk. This walk usually included walking down two flights of stairs to the ground floor and walking around on the first floor or outside of the sleep laboratory building. As such, it was common for subjects to be exposed to other patients, bright light, and moderate temperature change. Ss were not given a specific course, distance, or pace. The walk was designed primarily as a 5-min. break from the laboratory to induce stimulation common to

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**Table 1.—Daytime schedule**

<table>
<thead>
<tr>
<th>Time</th>
<th>Activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>07:00 - 07:30</td>
<td>Breakfast</td>
</tr>
<tr>
<td>08:00</td>
<td>Computer Tests</td>
</tr>
<tr>
<td>08:10</td>
<td>Resting Wake: 15 min</td>
</tr>
<tr>
<td>08:25</td>
<td>Sleep Latency Test</td>
</tr>
<tr>
<td>09:00</td>
<td>Break</td>
</tr>
<tr>
<td>09:05</td>
<td>Computer Tests</td>
</tr>
<tr>
<td>09:15</td>
<td>Walk: 5 min</td>
</tr>
<tr>
<td>09:20</td>
<td>Sleep Latency Test</td>
</tr>
<tr>
<td>10:20</td>
<td>Computer Tests</td>
</tr>
<tr>
<td>10:30</td>
<td>Resting Wake: 15 min</td>
</tr>
<tr>
<td>10:45</td>
<td>Sleep Latency Test</td>
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<tr>
<td>11:20</td>
<td>Break</td>
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<tr>
<td>11:25</td>
<td>Computer Tests</td>
</tr>
<tr>
<td>11:35</td>
<td>Walk: 5 min</td>
</tr>
<tr>
<td>11:40</td>
<td>Sleep Latency Test</td>
</tr>
<tr>
<td>12:15 - 12:40</td>
<td>Lunch</td>
</tr>
<tr>
<td>12:40</td>
<td>Computer Tests</td>
</tr>
<tr>
<td>12:50</td>
<td>Resting Wake: 15 min</td>
</tr>
<tr>
<td>13:05</td>
<td>Sleep Latency Test</td>
</tr>
<tr>
<td>13:40</td>
<td>Break</td>
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<tr>
<td>13:45</td>
<td>Computer Tests</td>
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<tr>
<td>13:55</td>
<td>Walk: 5 min</td>
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<tr>
<td>14:00</td>
<td>Sleep Latency Test</td>
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<td>15:10</td>
<td>Resting Wake: 15 min</td>
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<td>15:25</td>
<td>Sleep Latency Test</td>
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<tr>
<td>16:00</td>
<td>Break</td>
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<tr>
<td>16:05</td>
<td>Computer Tests</td>
</tr>
<tr>
<td>16:15</td>
<td>Walk: 5 min</td>
</tr>
<tr>
<td>16:20</td>
<td>Sleep Latency Test</td>
</tr>
</tbody>
</table>

* Half of the Ss had their Resting Wake first in each time block (8:15 in the first block) and half of the Ss had the Walk first in each block (8:15 in the first block).
patients in an outpatient hospital environment. When subjects
returned from the walk, they removed their shoes and
were reconnected to the polygraph machine. Room lights
were turned out, and the sleep latency test calibrations
began.

Following standard calibrations, research sleep latency
tests were performed. Because patients with insomnia fre-
quently do not fall asleep in the standard 20 minute MSLT
(giving an artificial upper limit), Ss were allowed a max-
imum of 30 minutes in bed during this study. However, Ss
were awakened earlier and the test terminated if sleep spin-
dles, k-complexes, or REMs occurred. Sleep latency was
scored in 30-second epochs to the onset of any stage of
sleep (usually stage 1). The sleep latency tests in this study
differed from the standard MSLT in that the standards
specify two-hour intervals between tests while tests in this
study occurred more frequently (60–90 minutes).

EKG data collection: Throughout the daytime test ses-
sions, EKG data were digitized by a National Instruments
NB-MIO-16 AD Board sampling at a rate of 500 samples
per second. A time code was digitized by a second channel
on the AD board and also printed out on the polygraph
paper to allow second by second matching of digitized
EKG with sleep stages and events. The EKG and time code
data were collected by LabView 3.0 software running on a
Macintosh II computer and stored on optical disk.

Analysis collection, the EKG and time data were visualized
and checked for artifacts with the LabView software and
output to a separate peak detection program used to con-
struct the tachogram and associated time code. As indicat-
ed earlier, heart rate data were recorded during all comput-
er tests, periods of watching TV, and MSLT. Mean heart
interbeat intervals for consecutive five-minute periods dur-
ing the MSLT will be reported.

Analyses

Data were analyzed by repeated measures ANOVA with
terms for activity level (walk or TV watching), prior sleep
(baseline, sleep deprivation, or recovery), time of test (four
times of day) and interaction. Pairwise comparisons were
performed with the Newman-Keuls test at the .05 signifi-
cance level using the Huynh-Feldt corrected degrees of
freedom. All reported results in the text will refer to statisti-
cally significant differences (p<.05) except where noted
otherwise.

RESULTS

Fourteen insomnia patients qualified for the study based
upon the screening criteria and began the study. Two of the
fourteen Ss had nocturnal sleep efficiency greater than 95%
and a sleep latency of less than 10 minutes on the baseline
night and were dropped from the study (for sleeping too
well to be considered an insomnia patient). Data from the
remaining 12 Ss—age 36 (sd 10.7), weight 162 pounds (sd
34), length of insomnia 5.4 years—will be reported. Six
subjects were female.

Sleep Data

EEG data from the screening, baseline, and recovery
nights are presented in Table 2. As can be seen from the
Table, some regression towards the mean was seen from the
Screen night to the baseline night and large changes in the
direction of improved sleep were found during recovery
from sleep deprivation.

MSLT Data

MSLT data across the day after baseline and sleep deprevi-
ation are plotted in Figure 1. Large, consistent main
effects were found for pre-nap activity (F1,11 = 22.5, p<
.001) and sleep deprivation (F2,22 = 21.9, p<.001). The
mean sleep latency on naps following the walk was 11.9
minutes (sd 5.3) and following TV watching was 6.9 min.
(Mean sleep latencies on naps following the walk was 11.9
minutes (sd 5.3) and following TV watching was 6.9 min.
and deprivation.


| TABLE 2.—Sleep parameters: mean values with (standard deviation) |
|-----------------|-----------------|-----------------|-----------------|-----------------|
|                  | SCREEN          | BASELINE        | RECOVERY        | F               |
| TOTAL SLEEP (Min)| 341 (64)        | 389 (63)        | 448 (32)        | 32.9 S<B<R      |
| % STAGE 1        | 17.2 (8.8)      | 19.3 (8.4)      | 13.2 (6.1)      | 3.98 B>S>R      |
| % STAGE 2        | 44.3 (13)       | 48.2 (12)       | 56.2 (9.5)      | 9.04 B=S>R      |
| % STAGE 3        | 1.5 (2.2)       | 0.9 (1.6)       | 2.6 (3.3)       | 3.58            |
| % STAGE 4        | 0.9 (2.8)       | 0.9 (3.2)       | 1.4 (4.7)       | 0.88            |
| % SWS            | 2.5 (4.8)       | 1.9 (4.5)       | 4.0 (7.3)       | 3.23            |
| % REM            | 14.5 (5.3)      | 15.3 (6.2)      | 21.4 (6.0)      | 11.5 B=S>R      |
| %WAKE            | 21.4 (15)       | 15.2 (13)       | 5.1 (5.7)       | 16.6 S<B<R      |
| STAGE 1 LATENCY (Min)| 41.0 (29) | 20.5 (11)       | 7.7 (7.9)       | 8.95 S>B>R      |
| SLEEP EFFICIENCY | 70.5 (15)       | 84.7 (13)       | 94.9 (5.7)      | 16.6 S<B<R      |
| REM LATENCY (Min)| 99 (36)         | 133 (98)        | 71 (47)         | 3.59            |

S = SCREEN; B = BASELINE; R = RECOVERY
NOTE: Significant differences noted.
following baseline sleep compared with 3.8 minutes (sd 5.2) after sleep deprivation. Following baseline sleep, MSLT latencies in the walk and TV watching conditions were respectively 16.2 (sd 5.2) and 10.5 (sd 5.2) min. The univariate ANOVA showed a significant sleep deprivation by time interaction (F6,66 = 6.16, p=.002). However, this interaction, which came primarily from the final day of the study when MSLT values started at almost sleep deprivation levels and then increased to baseline levels during the course of the day, was not significant in the multivariate ANOVA (F = 1.932, p=.222). No other significant interactions were seen.

**EKG Data**

The average interbeat interval from the first and last five-minute segments of each MSLT from the baseline day were entered into a repeated measures ANOVA with terms for the first or last five-minute, period (two levels), walk or TV condition (two levels), and time of day (four levels). Sleep deprivation data were not entered into this ANOVA because several Ss fell asleep in less than five minutes after sleep deprivation (and therefore the last five-minutes periods would be the same as the first). These data are plotted in Figure 2. The standard errors of the means for all periods were about .05 seconds. There were no significant interactions. The main effect for first or last MSLT epoch was significant (F1,11 = 15.4, p=.002) and indicated that heart period became longer in all MSLT evaluations from beginning to end (respective heart period means were .943 and .966, which can be converted to heart rates of 63.6 vs. 62.1 beats per minute - bpm). The main effect for walking or TV prior to the MSLT was significant (F1,11 = 5.44, p=.04) and indicated that heart period was longer in MSLT evaluations following TV watching than walking (respective heart period means were .967 and .942, which can be converted to heart rates of 62.0 versus 63.7 bpm). The main effect for time of day was significant (F3,33 = 6.92, p=.001). Pairwise comparisons indicated that heart period was longer in the second MSLT evaluation as compared to the third (respective heart period means were 0.998 and 0.911, which can be converted to heart rates of 60.1 and 65.9 bpm). The effect of sleep deprivation upon heart period was tested in a second ANOVA using data from the first 5-minute segment from the MSLT on the baseline and deprivation day (effects for sleep deprivation, activity, and time). There were no significant interactions in this ANOVA. There were significant main effects for all three factors. The values for Activity and Time were similar to those in the first ANOVA. The main effect for sleep deprivation was F1,11=7.90; p=.017. Heart period was longer in MSLT evaluations following sleep deprivation as compared to baseline (respective heart period means were .992 and .943, which can be converted to heart rates of 60.5 versus 63.7 bpm). Because Ss sometimes fell asleep in less than five minutes after sleep deprivation, 30-second periods of heart rate from the beginning of the MSLT were substituted in those cases for both the sleep deprivation and baseline observations (in an attempt to control for heart rate changes secondary to sleep onset). In this ANOVA, the effects for walking or TV prior to the MSLT and circadian rhythm remained significant at similar levels to the initial analysis, but the F-value for sleep deprivation (F1,11=2.30; p=.157) was no longer significant (means were 60.6 and 63.0 bpm).

**MSLT and EKG Data**

To determine whether consistent relationships existed between heart rate and nap latency, heart rate and sleep latency were averaged across the four baseline naps after walking or watching TV, and correlations were performed on the nap and heart rate means. These correlations were respectively r =-.554 (p=.06 with 10 df) and r =-.688 (p<.02) in naps after walking and after watching TV, and indicated that as heart period became shorter (i.e., higher heart rate), sleep latencies were longer. When all baseline walking and TV watching MSLT heart rate and sleep latency values were averaged, the correlation was r = -.644 (p<.05 with 10 df).

**DISCUSSION**

The sleep screening data show that patients had a mixture of sleep onset and sleep maintenance insomnia. The patients slept somewhat better on the baseline night, probably due to adaptation to the laboratory and regression toward the mean after the single screening night. As expected, recovery sleep after sleep deprivation resulted in a large increase in sleep efficiency. Little increase was seen in slow-wave sleep following sleep deprivation probably...
because these Ss had little slow-wave sleep under baseline conditions. As a result, there was a large rebound in REM on the recovery night, as seen in elderly subjects with little slow wave sleep. Total sleep deprivation also significantly decreased sleep latency by an average of 9.6 minutes, as measured by the MSLT.

The brief walk prior to the MSLT had a significant impact on heart rate during the MSLT. The average increase was statistically significant but numerically small (1.7 bpm). However, heart rate remained elevated throughout MSLT evaluations following the walk. These data indicate that the walk did produce physiological activation in these patients. The increase in heart rate was of the same relative magnitude as seen in normal sleepers participating in a similar study (12 - 2.2 bpm increase).

The data indicate that sleepiness as measured by sleep latency varied as a function of the activity which preceded the test in the insomnia patients. MSLT latencies were reduced overall by 5.0 min. when subjects relaxed in bed and watched TV prior to the sleep latency test as compared to walking. The magnitude of this change is similar to that seen in normal sleepers participating in a similar study (12 - 5.8 min. reduction).

Data from the current study have also shown a significant correlation between heart rate and nap latency across the baseline day. The level of this correlation (r= -.644) can be compared with a correlation of r= -.52 found between heart rate maximum and sleep latency in a maintenance of wakefulness design and a correlation of r= .633 reported by Johns et al in normal young adults during sleep onset at night. These data should not be viewed as cause and effect. Heart rate is probably only a convenient correlate of underlying changes in autonomic arousal. However, if a correlate of underlying arousal can control as much as 40% of the variance in sleep latency, the implication is that the underlying level of arousal plays a central role in allowing sleep onset.

The current study tested the hypothesis that insomnia patients would be unable to relax while lying in bed and watching TV and therefore would be at a similar state of arousal whether they watched TV or whether they walked. The data do not support this hypothesis. As measured by heart rate, the insomnia patients were at a higher level of arousal than the normals in the previous study after watching TV (nap heart rates were respectively 62 and 58 bpm), but both groups also had a similar increase in heart rate after walking (64 and 60 bpm respectively). Changes in baseline MSLT (the groups had different amounts of sleep deprivation) after TV and walking were similar in insomnia patients and normals (MSLT values after watching TV were respectively 10.5 and 6.7 minutes; MSLT values after walking were respectively 16.2 and 13.0 minutes.). These data support several studies that indicate that insomnia patients are generally at a higher level of physiological arousal than normals, but the insomniacs in this study were not at maximal arousal. As such, events such as the walk here, or additional stress in real life, produce a similar increase in arousal that results in a similar degree of masking of sleep tendency. As shown by the deprivation condition, however, increased drive to sleep still decreased MSLT latencies.

The literature indicates that sleep deprivation has inconsistent effects on heart rate. Some studies have shown increased heart rate after sleep deprivation, while other studies, as initially shown here, have documented decreased heart rate after sleep loss. This study implies that previous findings of decreased heart rate following sleep loss may have included some data from Ss falling asleep while their heart rate was being monitored resulting in decreased heart rate associated with the initiation of stage 1 sleep.

The current study can be criticized for not containing a control group of normal sleepers and for dropping two subjects who slept too well on their baseline night. A normal sleep group was not included because increased latencies on the MSLT have been found in several previous studies that did not directly compare insomnia patients and normal sleepers, and normal data from Ss undergoing an activity manipulation prior to sleep latency tests already existed. Ss who slept too well during the first night of the study were dropped because their inclusion could be viewed as biasing the results of the study towards those of normals (as possibly normal sleepers would have been included).

The MSLT was developed as a measure of sleep tendency. In ideal use as a measure of sleep tendency, the MSLT...
would only be administered in the absence of any arousing stimuli. The MSLT guidelines specifically instruct individuals using the test to minimize extraneous influences such as noise and light. Unfortunately, many arousing influences, such as pain, anxiety, motivation, or exposure to a hospital environment, cannot be easily measured or controlled. There is no empirical means of determining the effect of such variables upon the MSLT. Of equal importance, there is little empirical data to describe the magnitude or time course of impact of a broad range of possibly relevant variables upon this critically important test. Kronholm et al. using regression techniques, found five independent predictors of MSLT latency in addition to subjective sleepiness. Three of those five factors were associated with psychophysiological arousal. Stepanski et al. found a significant negative correlation between nocturnal total sleep time and MSLT latency—a result which cannot be understood if the MSLT only measures sleep tendency. These several studies demonstrate that 1) that the MSLT is sensitive to both sleep tendency and arousal; 2) that the MSLT results in a standard clinical environment cannot be interpreted correctly without reference to the state of physiological arousal; and 3) that relatively minor changes in physiological activation, even in aroused individuals, can have a large impact on the MSLT.

These results do not imply that the MSLT is a flawed test. It is an extremely sensitive test. The results do imply that the construct of sleep as a unitary system not influenced by other systems is incorrect. Much additional work needs to be done to understand how a broad range of external stimuli can affect measured sleep tendency. The simple physiological manipulations utilized in the current study and other recent studies were chosen because they are well-defined, easily replicated, and have a limited time of impact. If the impact from such trivial manipulations is large, the influence of significant sources of arousal such as grief, pain, or fear must be much larger. Such significant sources of arousal may not be easily identified, controlled, or time limited. Individuals also have a trait level of arousal ranging from calm to agitated. A constant test environment cannot control for all of these state and trait influences.

If each individual’s sleep latency is a combination of sleep tendency plus state arousal level plus trait arousal level, then insomnia patients may exemplify a case of high trait arousal masking sleepiness. Several previous studies have tried to explain apparent trait differences in sleep tendency as a function of chronic partial sleep deprivation or “sleep ability”. These previous results may alternatively be explained by trait differences in arousal level, which masks similar underlying sleepiness in differing amounts. When the MSLT is performed after varying degrees of sleep deprivation or sleep fragmentation, it is maximally sensitive to changes in sleepiness. When the MSLT is used after a normal night of sleep, all participants should have about equal amounts of sleepiness coming into the test. In such circumstances, the differences on the MSLT are probably due to differences in arousal.

Overall, the results of this study indicate the need for greater sensitivity to the role of state and trait physiological arousal in masking the expression of sleepiness in patients with arousal or sleep disorders. For researchers, it may be necessary to reexamine our knowledge of sleep within the framework of arousal. Disorders such as hypersomnia or narcolepsy, which have always been considered primary sleep disorders, could also be disorders of arousal.

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