The Consequences of a Week of Insomnia II: Patients with Insomnia

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Summary: Insomnia patients present with a consistent set of complaints that they generally report as secondary to their poor sleep, including increased tension/confusion, decreased vigor, personality disturbance, subjective overestimation of poor sleep, increased body temperature, increased 24-hour whole-body metabolic rate, and longer MSLT latencies. If there is a relationship between the poor sleep and the secondary symptoms, then particularly poor sleep should exacerbate those symptoms. Ten patients with insomnia were identified on the basis of a 2-night screening protocol, then slept in the laboratory for 10 additional nights. On 7 of the nights, the insomnia patients had their wake-after-sleep-onset increased so that their total sleep time was 80% of that on their second screening night, resulting in an average of 254 minutes (of 480 minutes in bed) of sleep. The spectrum of changes seen in these patients with insomnia who had very poor sleep for a week was characteristic of mild partial sleep deprivation, and not consistent with exacerbation of symptoms found in patients with primary insomnia. Specifically, (1) these patients had a reduction as opposed to an increase in the MSLT values, but the MSLT values at the end of the week remained within normal limits; (2) these patients had decreased (as opposed to increased) whole metabolic rate following nights of particularly poor sleep; (3) these patients tended to underestimate (rather than overestimate) their subjective sleep latency while being given particularly poor sleep; and (4) these patients displayed no significant change in body temperature, subjective anxiety, or MMPI scores following particularly poor sleep. It was concluded that the secondary symptoms reported by patients with primary insomnia are probably not related to their poor sleep per se. Data from previous studies that varied physiological arousal were used to support the contention that the secondary symptoms of patients with insomnia, and perhaps the poor sleep itself, occur secondary to central nervous system hyperarousal.

Key words: Sleep; insomnia; sleep fragmentation; sleep deprivation; partial sleep deprivation; sleep disorders; metabolism

INSOMNIA is a common sleep disorder with both nocturnal symptoms and daytime consequences. Typical sleep laboratory findings for patients with idiopathic (ICD 780.52-7)1 or psychophysiological (ICD 307.42-0)1 insomnia [for purposes of this paper, reference to “insomnia” should be taken as idiopathic or psychophysiological insomnia only] include long sleep latencies or excessive wake time during the night. Also observed are subjective fatigue,2,3 frequently accompanied by long latencies or inability to fall asleep on the multiple sleep latency test (MSLT)2,4,6; increased stress, anxiety, or depression 2,2; psychopathology, as measured by the Minnesota Multiphasic Personality Inventory (MMPI)7; increased physiological activation, as indexed by measures such as body temperature,8-10 whole body metabolic rate,2 or heart rate8,11-14; and consistent overestimation of sleep latency and time spent awake during the night.15,16

Patients report that their poor sleep leaves them tired and unable to perform at normal levels during the day. Insomnia may also be reported to cause stress or anxiety, and patients feel that improved sleep would alleviate this anxiety. However, it is equally likely that both the poor sleep and the whole constellation of secondary complaints are all actually symptoms of another underlying disorder.
Recently, evidence has been presented that these forms of "primary" insomnia, hereafter simply referred to as "insomnia," may actually be related to hyperarousal. As such, it can be hypothesized that the increased physiologic activity found in patients with insomnia reflects increased central nervous system activation that also interferes with the expression of sleep. One study tested this hypothesis by producing chronic physiological activation through the administration of caffeine. The chronic use of caffeine increased arousal level as measured by whole-body oxygen use, and subjects (Ss) reported increasing levels of daytime fatigue while their MSLT latencies remained significantly elevated as compared to baseline values. Anxiety, as measured by the anxiety scale of the MMPI (Pt), moved significantly toward psychopathology.

In another study, a yoke-control design was used to produce the EEG sleep distribution of a group of patients with insomnia in a group of matched normal sleepers for a week. It was hypothesized that if poor sleep by itself was responsible for the secondary symptoms reported by patients with insomnia, then normal sleepers given a similar sleep pattern would also begin to display the secondary symptoms. The results of the study indicated that normal sleepers given such poor sleep suffered from mild sleep deprivation but did not develop changes in mood, personality, or physiological activation typically seen in insomnia patients. However, it is possible that insomnia patients have a different response to their poor sleep than normals have to a similar sleep pattern.

Patients with insomnia typically overestimate their sleep latency and underestimate their total sleep time. However, patients with insomnia can typically differentiate nights with greater total sleep time or shorter sleep latencies as "better." As such, it might be hypothesized that "poor" nights of sleep—sleep with increased wake time and decreased total sleep—would, in general, exacerbate the secondary symptoms if those symptoms were related to sleep per se. Alternatively, it could be hypothesized that the secondary symptoms might change little or even improve after nights of reduced sleep if those symptoms were unrelated to the poor sleep.

One means of investigating the relationship between sleep patterns and secondary symptoms in patients with insomnia is to change the sleep of identified patients with insomnia systematically, and to measure the impact of these changes on hyperarousal and the other symptoms of primary insomnia. In the current study, patients with sleep-maintenance insomnia were identified by standard sleep criteria, and wake-after-sleep-onset was systematically increased for a week. If the symptoms of these patients worsened during the period of exceptionally poor sleep, then those symptoms could be seen as secondary to the poor sleep. On the other hand, if the secondary symptoms remained constant or improved, then some factor other than poor sleep itself would be seen as responsible for the secondary symptoms.

**METHODS**

**Subjects**

Potential patients with insomnia were identified by responses on a screening questionnaire. To be considered further, subjects were required to indicate (1) that they had a sleep problem; (2) that they had periods of 60 minutes or longer after falling asleep each night for at least 4 nights each week; (3) that this condition had existed for at least 1 year; and (4) that they usually spent about 8 hours in bed each night.

**Exclusions.**—Potential subjects 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. Also, Ss with a t-score above 75 on the depression scale of the MMPI were excluded. Ss with a history of depression or psychiatric care in the past 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 providing an informed consent and completing 2 hours of acclimatization to the laboratory with practice on computer tests and questionnaires to be used in the study.

**Screening.**—After practice, subjects were scheduled to spend 2 nights and the intervening day in the laboratory. Bed times and wake times on all study nights were set with respect to each subject’s habitual time of going to bed and arising. All times listed herein are based on a bed time of 23:00 and a wake time of 07:00. When participants had different habitual sleep times, all events throughout the days were shifted equally with the sleep times. On both nights, a standard clinical polysomnogram, including two eye channels, central and occipital EEG channels, chin and leg EMG channels, EKG, airflow, and chest movements, was performed. On the first night, blood oxygen saturation (SaO₂) was also recorded. On the second night, whole-body metabolic measures were recorded throughout the night.

On the intervening day, Ss had the MSLT, underwent metabolic observations, performed computer tests, completed an MMPI and a sleep history, and were fed standard meals. Caffeinated beverages were not available. Subjects usually did not leave the lab during this day. Following the second night in the lab, subjects completed some brief computer tests and were allowed to leave.

Subjects were assigned individual rooms 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 one or two individuals. Subjects completed all tests and questionnaires at their individual computer workstations in their rooms under technician observation via video monitors, and were not allowed to sleep while performing computer tests (video monitoring) or during metabolic observations (EEG monitoring). Meals and breaks were scheduled in another area of the laboratory also within technician observation.

Tests.—Performance and mood were assessed with a battery of measures, including the Memory and Search Test (MAST) (1, 3, and 5 letters),19 proofreading (10 minutes), hand tremor (2-minute insertion of a stylus into a 4 mm opening with percentage of side-touching time measured), the digit-symbol substitution task from the WAIS (5 minutes),20 computer-modified Williams Word Memory Test of immediate free recall,21 visual vigilance (30 minutes),22 subjective sleepiness (10-point analogue scale), Profile of Mood States (POMS), and oral temperature. The tests were administered in repeated batteries during the breaks between MSLT observations. For all subjects on all measures except MSLT, performance during continuous operations was automatically scored by the computer and output in a format suitable for statistical analysis.

MSLT and metabolic observations.—Four-channel sleep recordings (LE-A2, RE-A2, C3-A2, OZ-A1) were made during MSLT evaluations. The MSLT was performed at 10:00, 12:00, 14:00, 16:00, 18:00 and 22:00. The timing of the MSLT differed from the standard, in that an additional nap was placed at 22:00. The 22:00 time was chosen empirically so that the decrease in total sleep time from the screening night would approximate the reduction in total sleep time that occurred when normal young adults were given the sleep of patients with insomnia (about 70 minutes).18 The experimental total sleep time was divided by 4, and Ss were allowed to accumulate up to that amount of total sleep during each quarter of the night. When the sleep quota was achieved, Ss were awakened until the beginning of the next quarter of the night. For awakenings of 5 minutes or longer, the S was given the option of watching TV during the awakening as a means of helping to stay awake. Records were typically scored the following day to allow feedback to the technicians on how closely they were meeting the specified criteria for a given subject.

Data analyses.—The primary comparisons for this study were from nights 1 (baseline), 2 (poor sleep 1), 8 (poor sleep 7), and 9 (recovery night 1), and the following days. Repeated-measures analyses of variance (ANOVA) were performed for these 4 nights (conditions). For measures that were repeated across each day—MSLT, performance, and mood observations—a term for time of test (3-5 df) was added to the ANOVA. Pairwise comparisons were performed with the Newman-Keuls test at the .05 level using the Huynh-Feldt degrees of freedom. All reported results in the text will refer to statistically significant differences unless noted otherwise. Results on the many performance tests were similar; therefore, only data from MSLT, vigilance, short-term memory, MAST, proofreading, and the POMS subscales will be presented.

RESULTS

Subjects

The 10 participating subjects (6 male, 4 female) ranged in age from 21 to 48 years (mean=34.6, sd=7.8) and from 135 to 220 lbs (mean=164, sd=27). Their screening values on the MMPI depression (D) and anxiety (PT) scales were respectively 62 (sd=11) and 68 (sd=11). The depression score was somewhat lower than that reported for a large group of patients with insomnia screened to eliminate psy-
psychiatric disorders, probably as a result of the MMPI screening cut-off. The anxiety scale score matched the score reported for patients with insomnia screened to eliminate psychiatric disorders.

Sleep data.—Nocturnal sleep-stage data are presented in Table 1. Sleep data from the second screening night can be found in the far right two columns of the table. As expected from selection criteria, sleep was poor, with an average of almost 2 hours of wakefulness after sleep onset. In comparing the screening data to the initial baseline night in the study proper (first column), it can be seen that sleep was somewhat better on the baseline night, but that sleep efficiency still remained in the insomnia range. REM latency decreased from 100 to 67 minutes from the screening nights as a result of fewer skipped first REM periods, but both values were within one standard deviation of normative values. Sleep parameters moved in the direction of poor sleep on the experimental poor-sleep nights, and several of the changes, including decreased total sleep, decreased stage 2, decreased REM, increased wake time, and increased arousal index, were also statistically significant. On the right side of the table, average values for all 7 of the experimental poor-sleep nights are presented next to the data from the screening night. It can be seen that there was a reduction of total sleep time (TST) from 70-80 minutes in comparing the screening night value with the experimental poor sleep nights.

The actual distribution of sleep latency, REM, and total wake (sleep latency plus wake during sleep) across all study nights can be seen in Figs. 1, 2, and 3. Figure 1 shows objective and subjective sleep latency across study nights. No clear decrease in sleep latency was noted across sleep-loss nights. Interestingly, the insomnia patients overestimated their sleep latencies on the baseline and recovery nights, but not on any sleep-loss night following the first (which was preceded by no sleep loss). Figure 2, which presents REM across the nights, does not show any sign of increasing REM pressure across the nights until the first recovery night. Figure 3, which plots total sleep time across the nights of the study, demonstrates the experimental plan of consistently decreasing total sleep on the experimental nights. On the first recovery night, there was only one significant sleep stage difference compared to the baseline night, and that was a decrease in wake time from 57 to 34 minutes.

MMPI and POMS data.—The entire MMPI was administered prior to night 1 and again on the evening of the final poor-sleep night, because an earlier study had shown significant increases in anxiety (PT scale) when arousal level was increased for a week with caffeine. No meaningful or significant change was seen on any scale after 6 nights of poor sleep, and the values were similar to those seen in previous groups of patients with insomnia. Data from the POMS and Visual Activation Scale (VAS) were inconsistent (see Table 2). Significant main effects for condition were found for depression, vigor, and fatigue. Pairwise comparisons showed no significant condition differences for depression. Vigor was decreased only following the final poor-sleep night compared to recovery, and fatigue was increased across poor-sleep nights. These data were not paralleled by the VAS (sleepiness scale), which showed a significant increase in alertness only following the first recovery night as compared all other nights.

Subjective sleep evaluation data.—Subjective rating information for the EEG-recorded sleep nights reported earlier can be found in Table 3. The subjective data generally approximate the objective EEG sleep data. Subjects correctly indicated increased time awake on the poor sleep nights. Figure 1 allows direct comparison of subjective estimates of sleep latency with EEG values across the
entire study. Significant decreases in depth of sleep were noted by the final poor sleep night and recovery night, but patients reported no significant changes in their sleep quality throughout the study.

**Metabolic and temperature data.**—The individual VO$_2$ data from each of the four nocturnal recordings for each subject were time-matched and entered into a subject-by-condition repeated-measures analysis of variance. A similar repeated analysis of variance was computed for the daytime metabolic observations, which were time-linked to the start times of each metabolic period throughout the day. In the analysis of both the nighttime and daytime data, significant main effects for condition were found (respectively, F3,849=284, p<.001 and F3,261=33.38, p<.001). The data are plotted in Fig. 4. Pairwise comparisons indicated that metabolic rate was significantly different on each night as compared to all other nights. As shown in the figure, metabolic rate was increased on poor-sleep nights and decreased on the first recovery night as compared to baseline. VO$_2$ was also decreased following the first poor sleep night, and recovery as compared to the last poor sleep night.

No significant differences were found for oral temperature across the study (F3,117=0.998, NS).

**Psychomotor performance and MSLT data.**—Psychomotor tests analyzed included vigilance sensitivity...
(P(A)), MAST, short-term memory, and proofreading. On all performance tests, changes across the study were relatively minor. Significant differences were not found for vigilance, proofreading, or short-term memory. Significant condition main effects were found for the MAST single-letter search subtest, but examination of the condition means revealed consistently improving performance throughout the study, as would be expected from learning effects (respective means for baseline, P1, P7, and recovery were 61, 66, 68, and 84 lines searched).

Analysis of the MSLT data (F3,177=10.20, p<.0001) revealed that objective sleepiness was significantly increased following the final poor-sleep night as compared to the recovery and initial poor-sleep night, which did not differ. Objective sleepiness was increased in all conditions compared to baseline. Results for latency to stage 2 sleep were similar, except there were many observations with 20-minute latencies. Mean nap latencies from the individual MSLT observations can be found in Table 4.

**DISCUSSION**

In this study, sleep-maintenance insomnia was exacerbated in a group of patients carefully selected to display consistent idiopathic or psychophysiological sleep-maintenance insomnia, by experimentally increasing wake time during sleep for 1 week. It was hypothesized that if the complaints of these patients about their poor sleep, daytime dysphoria, fatigue, and other symptoms were related to their poor sleep, then these complaints and other symptoms would be exacerbated by the increased wake time after sleep onset. If these measures did not indicate increased pathology, then the increased wake time during the night was not responsible for the complaint.

The overall EEG data indicated good success in reducing the total sleep time of the patients with insomnia from screening and baseline levels over the week. Specifically, the goal of reducing TST to 80% of the screening night value was met, and the reduction in TST of about 80 minutes approximated the 60-minute reduction in TST in the study of normal young adults given the sleep of patients with insomnia.\(^{18}\) The tables and figures from this study can generally be compared to corresponding tables and figures from that earlier study\(^{18}\) to gain insight into the similarity of response of normals and patients with insomnia to these sleep reduction paradigms. Sleep-stage changes during the poor sleep condition were in the same direction and of similar magnitudes in patients with insomnia and in normals.

**Table 2.**—Profile of Mood States and VAS data

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Poor Sleep 1</th>
<th>Poor Sleep 7</th>
<th>Recovery 1</th>
<th>F3,117</th>
<th>P</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tension/Anxiety</td>
<td>5.2</td>
<td>5.6</td>
<td>6.4</td>
<td>5.9</td>
<td>1.026</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>6.2</td>
<td>6.8</td>
<td>9.4</td>
<td>7.8</td>
<td>3.610</td>
<td>.05</td>
<td>NONE</td>
</tr>
<tr>
<td>Anger</td>
<td>3.3</td>
<td>3.7</td>
<td>5.2</td>
<td>4.2</td>
<td>0.951</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Vigor</td>
<td>11.9</td>
<td>10.6</td>
<td>10.6</td>
<td>12.4</td>
<td>3.42</td>
<td>.025</td>
<td>P7&lt;R</td>
</tr>
<tr>
<td>Fatigue</td>
<td>6.5</td>
<td>7.9</td>
<td>9.4</td>
<td>5.5</td>
<td>7.63</td>
<td>.001</td>
<td>B=R&lt;P7;R&lt;P1</td>
</tr>
<tr>
<td>Confusion</td>
<td>5.9</td>
<td>6.1</td>
<td>7.2</td>
<td>6.2</td>
<td>2.069</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>VAS</td>
<td>5.0</td>
<td>4.8</td>
<td>4.4</td>
<td>5.7</td>
<td>6.716</td>
<td>.03</td>
<td>R&gt;ALL</td>
</tr>
</tbody>
</table>

Poor Sleep 1 = First night of experimentally reduced sleep
Poor Sleep 7 = Last night of experimentally reduced sleep
Recovery 1 = First night of undisturbed recovery sleep
Difference = Statistically significant condition differences: B=Baseline; R=Recovery 1; P1 = Poor Sleep 1; P7-Poor Sleep 7

**Table 3.**—Subjective sleep ratings

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Poor Sleep 1</th>
<th>Poor Sleep 7</th>
<th>Recovery 1</th>
<th>F3,27</th>
<th>P</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep Latency (Min)</td>
<td>49.2</td>
<td>45.1</td>
<td>20.1</td>
<td>24.9</td>
<td>4.441</td>
<td>0.04</td>
<td>NONE</td>
</tr>
<tr>
<td># of Wakes</td>
<td>3.1</td>
<td>4.8</td>
<td>3.3</td>
<td>1.5</td>
<td>4.705</td>
<td>0.01</td>
<td>R&lt;P1</td>
</tr>
<tr>
<td>Time Awake (Min)</td>
<td>47</td>
<td>136</td>
<td>149</td>
<td>26</td>
<td>7.384</td>
<td>0.002</td>
<td>B=R&lt;P1=P7</td>
</tr>
<tr>
<td>Sleep Length (Hr)</td>
<td>6.2</td>
<td>4.4</td>
<td>4.5</td>
<td>6.6</td>
<td>11.81</td>
<td>0.001</td>
<td>B=R&lt;P1=P7</td>
</tr>
<tr>
<td>Sleep Depth</td>
<td>2.3</td>
<td>2.3</td>
<td>1.8</td>
<td>1.8</td>
<td>3.75</td>
<td>0.04</td>
<td>R=P7&lt;B=P1</td>
</tr>
<tr>
<td>Sleep Quality</td>
<td>3.2</td>
<td>3.1</td>
<td>2.7</td>
<td>2.4</td>
<td>2.34</td>
<td>0.1</td>
<td></td>
</tr>
</tbody>
</table>

Poor Sleep 1 = First night of experimentally reduced sleep
Poor Sleep 7 = Last night of experimentally reduced sleep
Recovery 1 = First night of undisturbed recovery sleep
Difference = Statistically significant condition differences: B=Baseline; R=Recovery 1; P1=Poor Sleep 1; P7=Poor Sleep 7
Sleep Depth: Lower scores indicate deeper subjective sleep
Sleep Quality: Lower scores indicate better sleep quality
Specifically, in addition to the wake-related changes, the poor-sleep nights were characterized by significantly decreased stage 2, decreased REM, and an increased arousal index in both studies. SWS was significantly increased and stage 1 decreased on the first recovery night in the study of normal sleepers, but not in the current study with patients with insomnia.

Since wake time after sleep onset was successfully increased by about 80 minutes compared with the screening night, and by 2 hours compared to the baseline night for 7 consecutive nights in these patients with sleep maintenance insomnia, it would be predicted that insomnia symptoms related to this poor sleep should have been significantly exacerbated. Table 5 provides a summary of typical findings in patients with insomnia, and compares those findings with the results of the present study and a previous study in which the sleep distribution of a group of patients with insomnia was reproduced in a group of matched normal sleepers for a week. Changes secondary to the poor sleep produced in the current study clearly do not demonstrate an exacerbation of typical insomnia symptoms. In fact, of the seven common measures reported in Table 5, the only measures that moved significantly in the predicted direction were nighttime metabolic rate and subjective fatigue. MSLT values and daytime metabolic rate moved significantly in the wrong direction— ie, different from that predicted for insomnia.

Patients with insomnia typically have difficulty falling asleep both at night and during the MSLT. Both nocturnal sleep latency and MSLT data supported significantly increasing ease of falling asleep as nights of poor sleep increased.

Table 4.—Mean and standard deviation () of MSLT values across the day

<table>
<thead>
<tr>
<th>Time</th>
<th>Baseline</th>
<th>Poor Sleep 1</th>
<th>Poor Sleep 7</th>
<th>Recovery 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>10.00</td>
<td>13.0 (7.7)</td>
<td>10.6 (6.7)</td>
<td>8.6 (7.3)</td>
<td>9.6 (9.0)</td>
</tr>
<tr>
<td>12.00</td>
<td>12.8 (5.9)</td>
<td>13.0 (6.7)</td>
<td>10.8 (7.6)</td>
<td>10.6 (5.9)</td>
</tr>
<tr>
<td>14.00</td>
<td>15.6 (6.3)</td>
<td>13.3 (7.6)</td>
<td>10.8 (5.7)</td>
<td>15.0 (5.4)</td>
</tr>
<tr>
<td>16.00</td>
<td>14.4 (6.4)</td>
<td>13.4 (7.1)</td>
<td>11.6 (7.6)</td>
<td>15.0 (4.8)</td>
</tr>
<tr>
<td>18.00</td>
<td>18.8 (3.3)</td>
<td>13.6 (7.3)</td>
<td>12.7 (6.9)</td>
<td>12.4 (5.9)</td>
</tr>
<tr>
<td>22.00</td>
<td>18.9 (2.3)</td>
<td>17.2 (4.5)</td>
<td>12.3 (6.3)</td>
<td>18.4 (2.3)</td>
</tr>
<tr>
<td>Mean</td>
<td>15.6</td>
<td>13.5</td>
<td>11.1*</td>
<td>13.5</td>
</tr>
</tbody>
</table>

*Differs significantly from Baseline, Poor Sleep 1 and Recovery 1.

Poor Sleep 1 = First night of experimentally reduced sleep
Poor Sleep 7 = Last night of experimentally reduced sleep
Recovery 1 = First night of undisturbed recovery sleep

Table 5.—Changes in measures associated with insomnia in insomnia patients and in two experimental paradigms

<table>
<thead>
<tr>
<th>Patients with Insomnia</th>
<th>Current Study</th>
<th>&quot;Yoked&quot; Insomnia Normals</th>
</tr>
</thead>
<tbody>
<tr>
<td>MSLT</td>
<td>Increased</td>
<td>Decreased**</td>
</tr>
<tr>
<td>Metabolic Rate PM</td>
<td>Increased PM</td>
<td>Increased PM**</td>
</tr>
<tr>
<td>Metabolic Rate AM</td>
<td>Increased AM</td>
<td>Decreased AM**</td>
</tr>
<tr>
<td>Body Temperature</td>
<td>Increased</td>
<td>No Change</td>
</tr>
<tr>
<td>Mood (Tension, Confusion)</td>
<td>Increased</td>
<td>No Change</td>
</tr>
<tr>
<td>Vigor</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Fatigue</td>
<td>Increased</td>
<td>Increased**</td>
</tr>
<tr>
<td>Personality disturbance</td>
<td>Increased</td>
<td>No Change</td>
</tr>
<tr>
<td>Subjective Sleep</td>
<td>Overestimated</td>
<td>No Change or Underestimation</td>
</tr>
</tbody>
</table>

*Significant differences reported in the Bonnet and Arand study in which normal sleepers were given EEG sleep similar to that seen in patients with insomnia.
**Significant differences in the current study.
subjects are awake than when they are asleep, and is also higher in disturbed sleep,\textsuperscript{24} this significant increase can be accounted for by the fact that the patients with insomnia were awake 2 hours longer on their poor-sleep nights. Further, the fact that the metabolic rate was significantly decreased during the daytime observations following the poor-sleep nights, and was also increased during the night in normal subjects being given the poor EEG sleep of patients with insomnia,\textsuperscript{18} offer further support that the nocturnal finding was related to the experimental manipulation and not intrinsic differences in the patients.

Patients with insomnia typically report increased stress, anxiety, or depression.\textsuperscript{2,3} In the current study, significant increases were not found following the poor-sleep nights for the tension/anxiety, confusion, or depression scales of the POMS, although all three scales increased numerically during the course of the study. In contrast, values on all three of these scales decreased (significant for tension/anxiety and depression) when normals were given a sleep pattern of a patient with insomnia for a week.\textsuperscript{18} It is possible that the patients with insomnia responded to the stress of the long laboratory stay or the increased sleep loss in a manner different from the normal sleepers.

In a study in which normal sleepers were given large doses of caffeine for a week to produce hyperarousal,\textsuperscript{17} the anxiety scale of the MMPI (PT) increased significantly after caffeine administration. Patients with insomnia typically have higher MMPI scale t-scores than normals, but no significant changes in the MMPI were found in either the present study or in the study in which normal sleepers were given the sleep pattern of patients with insomnia. Patients with insomnia report increased fatigue and decreased vigor; similar changes, significant for fatigue, were found in the current study. However, these changes are also found during simple sleep deprivation and were also found in normals given the sleep pattern of patients with insomnia. Finally, patients with insomnia overestimate their sleep latency and time spent awake during the night. In the current study, as can be seen in Fig. 1, patients with insomnia consistently underestimated their sleep latency during poor-sleep nights while overestimating their sleep latency on baseline and recovery nights.

It is possible that the wrong variables were manipulated in this study or that the poor sleep was not produced for a period of time long enough to worsen the typical insomnia symptoms. If the results of this study had indicated slight but non-significant increases in symptoms, it might be concluded that not enough nights of exceptionally poor sleep were produced to magnify the disorder. However, as most measures moved in the direction opposite that predicted to be associated with insomnia, it is unlikely that more nights with this sleep pattern would have caused a reversal.

At a more psychological level, it is possible that Ss in this study attributed their very poor sleep to the direct intervention of the experimenter for the discreet period of time covered by the experiment. Patients with insomnia typically do not have either a clear attribution or an end point. Such uncertainty is certainly stressful and may compound an insomnia problem, but such stress almost certainly acts by directly increasing arousal rather than by directly fragmenting sleep. It is of interest that the patients with insomnia did not report a significant change in their sleep quality at any point during this experiment. Normal Ss given poor sleep reported significant decreases in sleep quality when given poor sleep, as would be expected. Such findings may imply that awakenings attributed to the experiment were not counted against sleep quality by the patients with insomnia, or perhaps that they were balanced by better sleep when sleep was allowed.

One criticism of the current study is that the methodology produced increased wake time during the night, but that increased wake time by itself is not necessarily the same as insomnia. It is important to understand that this “criticism” is the point of the experiment. Several years ago, modeling studies in young adults with normal respiration showed that the production of the characteristic sleep disturbance found in patients with sleep apnea resulted in the production of daytime sleepiness characteristic of patients with sleep apnea.\textsuperscript{25} Arousals produced to resume respiration were found to have a direct impact upon the sleep system, and, for this reason, sleep apnea produces a primary sleep disorder. In the current study, as in earlier modeling studies of hyperarousal\textsuperscript{17} and insomnia sleep patterns, a similar question was addressed—ie, if the sleep pattern of insomnia patients is produced\textsuperscript{18} or magnified (as we did when we produced the sleep pattern of patients with sleep apnea), does that produce or magnify other symptoms or complaints commonly given by patients or found in the laboratory? The study which increased arousal level indicated consistent shifts in most of the major symptoms associated with insomnia.\textsuperscript{17} The two studies which have modified EEG parameters either to produce the EEG patterns of insomnia in normals,\textsuperscript{18} or to exacerbate sleep-maintenance insomnia in insomnia patients, have consistently shown that the sleep patterns per se do not produce many of the symptoms that patients normally relate to their sleep. From this, one can conclude that the current study indeed did not worsen insomnia, despite large increases in wakefulness during the night, because insomnia is not a primary sleep disorder—it is a disorder of arousal. As such, manipulations which produce awakenings or increased wake time during the night do not cause insomnia, but instead cause excessive daytime sleepiness.

In general, the results of this study are very similar to those reported when normals were given a sleep pattern of a patient with insomnia for a week. In both studies, average MSLT latency decreased 4.5-4.9 minutes after a week
of poor sleep. Comparing metabolic rate changes, Fig. 4 in the current study shows almost the exact same shape as the metabolic rate figure from the earlier study.\textsuperscript{18} The figures differ primarily in that metabolic rate was consistently higher in the patients with insomnia by about 20-25 ml/minute, as has been shown in another study.\textsuperscript{2} The similarity in study-related changes in insomnia and normal groups is somewhat surprising in light of the fact that the reduction in sleep for patients with insomnia in this study—to an average of about 4.2 hour per night—was much greater than the 5.8 hours that the normals averaged.

In a study where normal sleepers were allowed 4.8 hours of sleep each night for a week, MSLT values were reduced by 9.7 minutes.\textsuperscript{26} The fact that the insomnia patients in the current study still had MSLT test results that would be classified as well into the normal range (mean of 11.1 minutes) after a week of sleeping little more than 4 hours per night implies that the patients have either a significantly reduced sleep need or significant levels of physiologic arousal blocking sleep drive. In the study with normal sleepers, sleep latencies decreased across the poor-sleep nights, and REM percentage increased despite poor sleep.\textsuperscript{18} In the current study, no trends towards decreasing sleep latency or increasing REM were seen across poor-sleep nights (see Figs. 2 and 3). The patients did sleep well on their first recovery night. In fact, sleep parameters from the insomnia patients on the first recovery night are very similar to those found for the normal sleepers on their first recovery night.\textsuperscript{18} Unfortunately, the poor-sleep pattern had already returned by the second recovery night.

The most parsimonious explanation for the data presented is that poor sleep resulted in partial sleep deprivation. Partial sleep deprivation can account for the significant findings of decreased MSLT, decreased daytime metabolic rate, and increased fatigue. Despite greater sleep loss than that given to normals in a similar study,\textsuperscript{18} the patients with insomnia showed fewer sleep-deprivation-associated deficits.

If very poor sleep in patients with insomnia does not exacerbate their secondary insomnia symptoms, how can the consistent secondary symptoms be explained? Several studies\textsuperscript{17,18,27,28} have concluded that many primary symptoms reported by insomnia patients are actually related to hyperarousal rather than to poor sleep. If the secondary symptoms are related to degree of hyperarousal and not to sleep per se, the lack of an increase in arousal level during the study could explain why the patients with insomnia did not report decreased sleep quality despite large increases in nocturnal wake time.

The current study was different from sleep-restriction studies in that Ss continued to spend the same amount of time in bed each night despite their decreased sleep time. As such, the sleep restriction produced in this study should not have had a noticeable impact on circadian rhythms, and should have maintained unpleasant associations of lying in bed awake.

Together, the current study and several earlier ones\textsuperscript{17,18,27} provide evidence that poor sleep, like fatigue and increased metabolic rate, is a symptom in patients with idiopathic or psychophysiological insomnia, and not the primary disorder. Patients with insomnia do sleep poorly, but the current data suggest that, because worse sleep may actually reverse several symptoms, sleep itself is not the basis of many of the secondary symptoms. Such data imply that many patients with insomnia may respond to treatments which involve relaxation, for example, because relaxation decreases arousal level. Patients may not respond well to other treatments, however, because the wrong problem is being addressed. For example, patients with sleep-state-misperception insomnia are treated as if they have a cognitive disorder. It is now known that sleep-state-misperception insomnia patients have increased whole-body metabolic rate (ie, have evidence of increased arousal level) while presenting normal EEG parameters.\textsuperscript{27} If their primary hyperarousal is treated, then their (real) insomnia should also improve. It is semantically confusing but conceptually important to understand that the complaint of insomnia that is heard may not be directly related to the EEG that is commonly measured, but rather to the level of arousal that is not commonly recorded.

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REFERENCES

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