Does Sleep Deprivation Worsen Mild Obstructive Sleep Apnea?

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Study Objectives: Sleep deprivation is believed to worsen obstructive sleep apnea (OSA). We assessed the effect of acute sleep deprivation on polysonmography in a cohort of subjects with mild OSA and a cohort of subjects without OSA.

Design: Crossover study in which subjects initially had polysomnography after a normal night’s sleep or after 36 hours of sleep deprivation, followed by a 2- to 4-week interval, after which subjects were restudied under the alternate testing condition.

Setting and Participants: 13 subjects with mild OSA and 16 subjects without OSA were studied in a university teaching hospital sleep laboratory.

Interventions: 36 hours of supervised sleep deprivation.

Measurements: Subjects’ age, body mass index, neck circumference and Epworth Sleepiness Scale scores were measured; actigraphy and sleep diaries were used to estimate prior sleep debt before each sleep study.

Results: Sleep deprivation was found to significantly increase total sleep time, sleep efficiency, and rapid eye movement and slow-wave sleep time. Subjects with OSA showed a lower minimum oxygen saturation after sleep deprivation. However, subjects did not show a significantly different respiratory disturbance index, arousal index, or length of the longest apnea after sleep deprivation.

Conclusions: Acute sleep deprivation did not worsen most OSA parameters as measured by polysomnography. A lower minimum oxygen saturation in mild OSA subjects after sleep deprivation may be important in patients with significant cardiorespiratory disease. More research is needed to assess whether daytime performance and function (eg, driving, sleepiness) is more greatly impaired in OSA subjects who are sleep deprived, compared to normal subjects who are sleep deprived.

Key Words: sleep deprivation, mild obstructive sleep apnea, polysomnography

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INTRODUCTION

OBSTRUCTIVE SLEEP APNEA (OSA) IS VERY COMMON, AFFECTING APPROXIMATELY 25% OF MIDDLE-AGED MEN. Most (approximately 60%) of OSA patients have only mild disease (respiratory disturbance index [RDI] 5-15). Factors that are associated with acute worsening of OSA include alcohol ingestion or nasal obstruction, and conservative management strategies for OSA often include alcohol cessation or relief of nasal obstruction.

Sleep deprivation is another factor that may increase OSA severity. This has been suggested by several studies of sleep-breathing physiology. Firstly, breathing responses to hypoxia may be decreased after sleep deprivation; secondly, sleep deprivation has been shown to have a depressive effect on upper-airway dilator-muscle activity, which is important in maintaining upper-airway patency during sleep; and, finally, sleep deprivation increases rapid eye movement (REM) sleep, where apneas are more likely to occur.

Some of the earliest work on sleep-disordered breathing commented on the potential deleterious effect of sleep deprivation on nocturnal breathing. This has been further supported by a small amount of published work, showing that sleep deprivation worsens OSA. In 1981, Guilleminault and coworkers reported an increase in the mean number of apneic episodes and a lowering of blood oxygen saturation secondary to apneic events following acute sleep deprivation of subjects with mild to moderate OSA. However, in this study the numbers were very small (N = 4).

More recent work has shown that the loss of 1 night’s sleep results in a large increase in the RDI of OSA patients. However, these studies have compared nocturnal polysomnography with daytime polysomnography after sleep deprivation to assess the effect of sleep deprivation on breathing during sleep. One study has investigated the effect of chronic partial sleep deprivation on OSA, revealing an increase in RDI of over 50% and a lowering of the minimum oxygen saturation secondary to apneic events, but subject numbers were very small (N = 6), and limited respiratory monitoring was used to measure OSA (Mesam IV, Madaus Schwarzer Medizin Technik, Munich, Germany, available through Healthdyne Technologies, Marietta, Georgia).

Patients often complain that their snoring and sleep apnea appear to be worsened by sleep loss or sleep deprivation. Given the potential interaction between sleep deprivation and OSA described above, one could easily imagine a “self-feedback loop,” in which sleep deprivation worsens OSA, which in turn has a negative impact on nocturnal sleep quality and sleep hours, worsening sleep deprivation even further. Hence, investigating whether the coincidence of OSA and sleep deprivation has a multiplicative adverse effect on nocturnal breathing disturbances and daytime function is very important and will have important implications, for example, with respect to driving.

The aim of this study was to determine the effect of acute sleep deprivation on measured nocturnal sleep and breathing parameters in a cohort of subjects with mild OSA and a cohort of subjects without OSA.

METHODS

Patients with mild OSA and controls were recruited for this study. Patients with mild OSA were identified in hospital sleep clinics and then invited to participate, or alternatively, they were identified through an audit of recent sleep study results from Royal Prince Alfred Hospital Sleep Investigation Unit. Controls were recruited from these same sources and also from advertising among university students.

Twenty-nine subjects (13 with mild OSA, 16 without OSA) underwent polysomnography on 2 occasions: under normal sleeping conditions (baseline testing) and after 36 hours of acute total sleep deprivation.
Subjects were supervised by sleep laboratory staff at all times during their period of sleep deprivation to ensure they did not sleep. Approximately half of each group (mild OSA and controls) were tested under baseline conditions first, with the remaining subjects tested under sleep deprivation conditions first. Generally, both sleep studies were conducted within 2 to 4 weeks of each other. Age, body mass index, neck circumference, and Epworth Sleepiness Scale scores were obtained from the subjects on their first visit. Subjects were instructed to obtain at least 8 hours sleep per night for the 4 nights preceding each occasion of testing to minimize any preexisting sleep debt at the start of a test. Sleep hours for these 4 nights were also measured objectively using sleep diaries and actigraphy.

Given the nature of the testing, it was impossible to blind either the subjects or researchers to the order of testing or to the testing conditions (baseline or sleep deprivation). However, all the sleep studies were scored by an experienced technician who was blinded to the patients’ status. The protocol was approved by the Central Sydney Area Health Service Ethics Committee (RPA zone) and the University of Sydney Ethics Committee, and informed consent was obtained from each patient.

Classification of subjects as having OSA or no OSA (ie, control status) was made on the basis of the results of the baseline weekend’s sleep study. Subjects were regarded as having OSA if their total RDI was at least 5. Controls had a total RDI less than 5.

Nocturnal Polysomnography

A standard sleep study (polysomnography) setup was used for all subjects. The electroencephalogram placement was according to the 10-20 system with 4 electrodes attached to the scalp (C1, O2, A1; and A2), 2 eye electrodes (left and right electrooculogram), and 2 submental leads (electromyography). There were also 2 electrocardiogram leads, 1 near the right shoulder and a second in the sixth intercostal space on the left side of the chest. Sensor leads were placed on each leg over the anterior tibialis for recording leg movements. Breathing was monitored by a nasal pressure transducer in all studies. Inductive respiratory effort bands were placed around the chest and abdomen to monitor thoracic and abdominal wall movement. A position sensor was attached to monitor body position during sleep. A finger probe oximeter continuously recorded the subjects’ oxygen saturation. Specialized computer software (Compumedics, Melbourne) was used to acquire, store, and analyze the collected sleep study data.

Polysomnography Scoring

Sleep stages were scored according to Rechtschaffen and Kales criteria.12 Arousals were scored according to the Atlas Task Force of the Polysomnography Scoring Manual. Arousals were scored according to the Atlas Task Force of the Polysomnography Scoring Manual. Arousals were scored according to the Atlas Task Force of the Polysomnography Scoring Manual.

Table 1—Baseline characteristics* of the study groups

<table>
<thead>
<tr>
<th></th>
<th>Controls (n, 16)</th>
<th>OSA (n, 13)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>38 ± 14.7</td>
<td>45 ± 14.8</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>25.5 ± 5.1</td>
<td>30.3 ± 4.2</td>
</tr>
<tr>
<td>Neck circumference, cm</td>
<td>38.9 ± 2.5</td>
<td>41.4 ± 2.6</td>
</tr>
<tr>
<td>Epworth Sleepiness Scale score</td>
<td>7 ± 4.2</td>
<td>9 ± 6.0</td>
</tr>
<tr>
<td>Sleep time, h</td>
<td>7 ± 0.5</td>
<td>6.6 ± 0.5</td>
</tr>
</tbody>
</table>

*Data are presented as mean ± SD.
†Sleep time is the average number of hours of sleep per night recorded during the 4 nights preceding the study.
BMI refers to body mass index.

RESULTS

Table 1 presents the baseline characteristics of the 2 study groups. Subjects with OSA were more overweight, as indicated by an increased body mass index and neck circumference. The mean Epworth Sleepiness Scale Score for both groups was less than 10. Both groups slept less than 8 hours on average for the 4 nights preceding the testing.

There was no difference in average sleep hours during the preceding 4 nights in the subjects prior to the 2 tests (6.7 hours sleep per night prior to baseline test compared to 6.9 hours sleep prior to sleep deprivation test; difference 0.3 hours, 95% confidence interval -.006 to 0.6).
Baseline Polysomnography

Table 2 presents the baseline polysomnography results of the 2 study groups. Subjects with OSA had a mean total RDI of 12, compared to a mean total RDI of 2 in the control group. Similarly, there was a difference between the groups in other measures of sleep-disordered breathing, especially non-REM RDI, arousal index, and minimum oxygen saturation. Based on these polysomnographic results, OSA subjects had mild disease, whereas controls, by definition, had no OSA.

Effect of Sleep Deprivation on Polysomnography

Table 2 illustrates that, after sleep deprivation, subjects slept longer, had greater sleep efficiency, and increased amounts of REM and slow-wave sleep. However, RDI, arousal index, and the length of the longest apnea were not significantly different. The effect of sleep deprivation on minimum oxygen saturation differed according to OSA status. Subjects with OSA showed a lower minimum oxygen saturation after sleep deprivation, whereas control subjects did not.

DISCUSSION

The aim of this study was to determine the effect of acute sleep deprivation on measured nocturnal sleep and breathing parameters in a cohort of subjects with mild OSA and a cohort of subjects without OSA. Sleep deprivation was found to significantly increase total sleep time, sleep efficiency, and REM and slow-wave sleep time. Subjects did not show a significantly different RDI, arousal index, or length of the longest apnea after sleep deprivation. However, subjects with OSA showed a lower minimum oxygen saturation after sleep deprivation.

Because the order of the sleep studies was balanced, with approximately half of the subjects of each group (mild OSA and controls) tested under baseline conditions first, with the remaining subjects tested under sleep deprivation conditions first, the differences in polysomnographic outcomes due to sleep deprivation could not be due to first-night effects (subjects sleeping worse on their first weekend of testing because of unfamiliarity).

This study controlled for prior sleep hours before the 2 polysomnograms. All subjects were instructed to obtain at least 8 hours sleep per night for the 4 nights preceding each occasion of testing, and subjects also completed a sleep diary and wore actigraphs during this period. Analysis of these recordings showed that there was no statistically significant difference in the amount of sleep the subjects had prior to their 2 polysomnograms. Hence, differences in polysomnographic measures between the 2 testing conditions would not be expected to be explained by differences in sleep debt from the days immediately prior to the subjects’ sleep studies.

Sleep measurement by actigraphy and sleep diary showed that our subjects were partially sleep deprived preceding the test nights (6.7 to 6.9 hours of sleep per night, on average). This may have limited the ability to detect an effect of acute sleep deprivation. However, this level of chronic partial sleep deprivation is very frequent in society. A major strength of this study is that we requested subjects avoid sleep deprivation, measured prior sleep hours by 2 different methods, and found this not to differ significantly between groups or testing conditions.

An attempt was made to reduce clinic selection biases in the control group by recruiting university students via an advertisement. However, part of the control group was still recruited from patients seen at hospital sleep clinics or from patients who had had negative laboratory sleep studies. These ‘controls’ are therefore unlikely to be totally disease free. Table 2 shows that although the total RDI was less than 5 in the control group’s baseline polysomnography, their mean REM RDI was 8. As a consequence of selection biases, the presence of simple snoring or mild REM OSA in the control population may have biased the study against finding a difference between the 2 study groups. However, these selection biases would not be expected to affect the response to sleep deprivation in the study subjects. Because a single technician scored all sleep studies, differences in scoring between studies would be very small, eliminating this potential bias.

In this study, subjects with OSA showed a lower minimum oxygen saturation after sleep deprivation. A reduction in minimum oxygen saturation in OSA patients after sleep deprivation has been noted previously. This is likely to be of marginal clinical significance for healthy

### Table 2—Polysomnographic data at baseline and after sleep deprivation.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Total Subjects N, 29</th>
<th>OSA Subjects n, 13</th>
<th>Control Subjects n, 16</th>
<th>Interaction*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep time, min</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>344 (50.6)</td>
<td>53 (15 to 91)</td>
<td>0.01</td>
<td>343 (50.3)</td>
</tr>
<tr>
<td>REM</td>
<td>62 (24.4)</td>
<td>17 (4 to 30)</td>
<td>0.01</td>
<td>62 (24.3)</td>
</tr>
<tr>
<td>S1</td>
<td>22 (12.2)</td>
<td>-8 (-13 to -2)</td>
<td>0.01</td>
<td>22 (12.2)</td>
</tr>
<tr>
<td>S2</td>
<td>195 (28.7)</td>
<td>15 (-9 to 40)</td>
<td>0.20</td>
<td>194 (28.6)</td>
</tr>
<tr>
<td>SWS</td>
<td>63 (21.5)</td>
<td>19 (7 to 32)</td>
<td>0.01</td>
<td>63 (21.4)</td>
</tr>
<tr>
<td>SE, %</td>
<td>82 (11.6)</td>
<td>9 (6 to 13)</td>
<td>0.00</td>
<td>81 (11.6)</td>
</tr>
<tr>
<td>RDI Total</td>
<td>7 (5.4)</td>
<td>1.0 (-2 to 4)</td>
<td>0.50</td>
<td>12 (1.4)</td>
</tr>
<tr>
<td>During REM</td>
<td>14 (12.8)</td>
<td>-1 (-6 to 4)</td>
<td>0.62</td>
<td>20 (12.7)</td>
</tr>
<tr>
<td>During NREM</td>
<td>5 (6.1)</td>
<td>1 (-2 to 4)</td>
<td>0.33</td>
<td>10 (6.1)</td>
</tr>
<tr>
<td>AI</td>
<td>13 (6.1)</td>
<td>-2 (-5 to 1)</td>
<td>0.14</td>
<td>17 (6.0)</td>
</tr>
<tr>
<td>SRE</td>
<td>2 (1.4)</td>
<td>-0.4 (-0.9 to 0.2)</td>
<td>0.23</td>
<td>2 (1.4)</td>
</tr>
<tr>
<td>Longest apnea, s</td>
<td>23 (16.4)</td>
<td>0.0 (-7 to 7)</td>
<td>1.0</td>
<td>29 (18.3)</td>
</tr>
<tr>
<td>Min O₂ sat, %</td>
<td>91 (2.3)</td>
<td>-1.1 (-2.4 to -0.02)</td>
<td>0.047</td>
<td>89 (2.6)</td>
</tr>
</tbody>
</table>

*Interaction, significance test on the difference in the effect of sleep deprivation between patients with mild OSA and control subjects.
†Data are presented as mean (SD).
‡Data, presented as mean (95% CI), are the difference between baseline and sleep deprivation conditions.

OSA refers to obstructive sleep apnea; REM, rapid eye movement sleep; S1, stage 1 sleep; S2, stage 2 sleep; SE, sleep efficiency; RDI, respiratory disturbance index (the number of apneas and hypopneas per hour of sleep); NREM, non-rapid eye movement sleep; AI, arousal index (the number of arousals per hour of sleep); SRE, the number of subcriterion respiratory events per hour of sleep; Min O₂ sat, the minimum oxygen saturation.
patients but may be more relevant in patients with significant cardiorespiratory disease.

This study did not show significant differences in RDI, arousal index, and length of the longest apnea after sleep deprivation in the subjects with OSA. This is in contrast to previous studies with small patient numbers,10,11 which have utilized short periods of daytime polysomnography after 1 night without sleep6,9 or that have used limited respiratory monitoring to measure OSA.10 Although this study has essentially shown that acute sleep deprivation does not worsen OSA severity, the effect of chronic sleep deprivation has not been examined and may differ from that of acute sleep deprivation.

This study showed that normal subjects do not develop OSA acutely as a result of sleep deprivation. Several studies of sleep-breathing physiology3-5 suggest plausible physiologic reasons why this might occur. However, other studies suggest sleep fragmentation has more profound effects on upper-airway collapsibility16 and ventilatory drive17 than total sleep deprivation.

This study was adequately powered to detect clinically important differences in the outcome variables after sleep deprivation (eg, there was an 80% power to detect a difference in total RDI of 5.8 per hour or greater in the OSA subgroup). Smaller differences due to sleep deprivation are unlikely to have been detected in this study. A larger study would be needed to detect smaller differences, especially with respect to the interactive effects of sleep deprivation between subjects with and without OSA.

Subjects with OSA in this study had relatively mild disease, with an average RDI of 12.1 (range, 9.2-15.0). Importantly, these subjects in general did not report excessive daytime sleepiness, as measured by the group’s mean Epworth Sleepiness Scale score (9 ± 6.0). Hence our study subjects with OSA are more representative of those patients with OSA alone (snoring, nocturnal apneas, obstructive respiratory events on polysomnography), rather than those with sleep apnea syndrome (clini-cal combination of OSA and excessive daytime sleepiness).10 It is not known whether these 2 different groups of OSA patients differ in other respects, such as in their response to sleep deprivation. It also is unknown as to whether the results of this study can be extrapolated to subjects with moderate or severe OSA.

Larger studies with a broader range of OSA subjects that address this same research question are needed to further our knowledge of the effect of sleep deprivation on OSA. These studies should also examine whether daytime performance and function (eg, driving, sleepiness) are more severely impaired in OSA subjects who are sleep deprived, compared to normal subjects who are sleep deprived. This will have important clinical implications, especially with regard to advice about sleeping habits for high-risk groups of OSA patients (eg, commercial drivers).

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1041 Does Sleep Deprivation Worsen Mild Obstructive Sleep Apnea?—Desai et al