

Sleepiness-Related Accidents in Sleep Apnea Patients

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Abstract: The frequency of motor vehicle and working accidents was analyzed by means of a strictly anonymous questionnaire in 156 patients with sleep apnea syndrome (SAS) and in 160 age-gender matched controls. In the SAS group 12.4% of all drivers had motor vehicle accidents as compared to 2.9% in the control group ($p < 0.005$). The motor vehicle accident rate was 13.0 per million km in patients with more severe SAS (AHI $> 34/h$, $n=78$) as compared to 1.1 in patients with milder SAS (AHI 10-34/h, $n=78$) ($p < 0.05$), and 0.78 in control group ($p < 0.005$), respectively. The accident rates in both patients and the control group were also greater than the rate of 0.02 "accidents due to sleepiness" per one million km in the Swiss driving population as reported by official statistics. During treatment with nasal continuous airway pressure (nCPAP) in 85 SAS patients, the motor vehicle accident rate dropped from 10.6 to 2.7 per million km ($p < 0.05$).

We conclude that patients with moderate to severe SAS have an up to fifteen-fold risk increase of motor vehicle accidents that constitutes a serious and often underestimated hazard on the roads, which can be reduced by adequate treatment.

Key words: Sleepiness; driving; accidents; sleep apnea; nasal continuous positive airway pressure (nCPAP)

Abbreviations: AHI apnea-hypopnea index; SAS sleep apnoea syndrome; nCPAP nasal continuous positive airway pressure; EDS excessive daytime sleepiness; ESS Epworth sleepiness score; km kilometer (s); BMI body mass index

INTRODUCTION

WORKING AND MOTOR VEHICLE ACCIDENTS DUE TO SLEEPINESS HAVE BEEN ANECDOTALLY REPORTED SINCE 1929.¹ Traffic accidents while falling asleep are particularly hazardous because the driver does not decelerate the motor vehicle in face of the collision.²⁻⁵ Sleepiness has also been suggested as a major cause of the so called mega-accidents of Tschernobyl, Exxon Valdez, and Bhopal.⁶ However, it is only within the last 10 years that the true dimension of the problem has been recognized. The estimated percentage of car accidents caused by sleepiness varies over a wide range from 0.4% to 30%.^{2,3} An excellent survey on the relationship between sleepiness and accidents is presented in the Supplement of *J. Sleep Res.* in December 1995.⁷

Numerous risk factors for poor performance and particularly for falling asleep at the wheel have been recognized: Driving during the night hours or during mid-afternoon,^{4,5} adolescent age,⁸ male sex,^{4,5} consumption of drugs or alcohol, driving on motorways or major roads, chronic sleep restriction,^{9,10} shift work,^{11,12} narcolepsy, and snoring as well as sleep apnea syndrome (SAS).¹³⁻¹⁸ Apart from lifestyle-related sleep deprivation SAS is among the most prevalent diseases causing excessive daytime sleepiness

(EDS).¹⁹ Questionnaire surveys on car drivers with suspected SAS revealed greatly divergent estimates of two to tenfold higher risk of car accidents compared to normals.^{17,20-25} Similarly, retrospective analysis of police records revealed a two- to sevenfold elevated accident frequency of SAS patients.²⁶⁻²⁹ On the other hand, SAS patients reported a reduction of accidents under nCPAP treatment³⁰⁻³² or following uvulopalatopharyngoplasty.³³ Many reasons may account for the great variation in suspected accident risk increase in SAS patients: Use of different study parameters, variable severity of the SAS, missing or unsuitable control groups,^{17,20-24,26-28} and lack of consistent polysomnographic confirmation of SAS in some studies.^{17,23,24,26,28,33} Insufficient awareness of the problem and the absence of an entirely accurate objective method to measure sleepiness³⁴ seems to hamper police statistics, and reluctant self-reporting of motor vehicle accidents for fear of losing the driving license²⁰ is a major obstacle when using questionnaires.

We present a retrospective questionnaire study on accidents due to sleepiness in 156 patients with polysomnographically proven SAS and in an age- and sex-matched control group. The overall accident frequencies in both groups were compared with the official federal statistics reporting the annual number of "accidents due to sleepiness" in the Swiss driving population. The aim of the study was twofold: First particular care was taken to use a strictly anonymous questionnaire system to encourage faithful reporting by the patient. Second the comparison of the nor-

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mal controls with federal statistics was undertaken to estimate whether "accidents due to sleepiness" are underestimated by the police statistics. The latter information may be of importance in view of the public financial support for research and prevention.

METHODS

We retrospectively identified all 217 German speaking SAS patients with at least 10 apneas and/or hypopneas per hour of sleep (apnea-hypopnea index (AHI) $\geq 10/h$) during a diagnostic nocturnal polysomnography (method see appendix) performed between January 1993 and May 1996. Patients treated by uvulopalatopharyngoplasty after diagnosis were excluded. A control group of 227 age- and sex-matched subjects examined for low back pain or carpal tunnel syndrome were consecutively selected in our outpatient clinic during the same time period. None of them had any other known neurological illnesses. A questionnaire was sent to all subjects. Eighty-five patients who were already treated by nasal continuous positive airway pressure (nCPAP) filled out two questionnaires, one for the time before treatment and one for the time during treatment.

In order to assure anonymity but not to lose information about the severity of the SAS, the questionnaire sheets were coded with a color according to the degree of SAS. Eleven degrees of severity were defined according to the AHI in steps of five: AHI = 10 to 14/h, 15 to 19/h, ..., 70 to 79/h, and over 80/h. Patients with an AHI of 34 or less were defined as having a mild SAS, those with an AHI of 35 or more as having a moderate to severe SAS. The cut-off value of 34.5 was set in order to get two groups with a similar number of patients.

The questionnaire included information on age, sex, height, body weight, use of sleeping pills, alcohol consumption (glasses per week), and annually driven kilometers (km). To estimate subjective daytime sleepiness, the Epworth sleepiness scale (ESS), which assessed the chance

of dozing off or falling asleep in eight different situations, was used.³⁵ Values from 0 to 10 points are generally assumed to be normal; those above 10 indicate EDS. In addition, the subjects were asked to report the number of motor-bike and car accidents associated with sleepiness during the past three years. All reported traffic accidents were subdivided into those with material damage below 1000 Swiss francs (600 US\$) and into those with material damage above 1000 Swiss francs or personal injury. In addition, any accident in the workplace or at home which the subject remembered as significant and as being caused by falling asleep was considered separately. No attempt was made to further specify or quantify workplace or home accidents in the query, since this is difficult to achieve retrospectively. Also, in order to have an optimal return quota, much care was taken to keep the query short. Because of the attempted absolute anonymity, no information on the subjects occupation or educational background was gathered.

The accidents per one million driven km were calculated for both, mean accidents per individual subject per one million driven km, and number of accidents per one million driven km within each group (total number of accidents in the group divided by sum of driven km in the group).

The Chi-Square test (2x2 contingency table) for categorical data, the Student's t-test for normally distributed parametric, and the Mann-Whitney U-test for non-parametric, continuous variables were used. Each test was performed for a two-tailed hypothesis and p values >0.05 were considered non significant (n.s.). Data from patients treated with nCPAP were analysed with the Wilcoxon Matched-Pairs Signed Rank test.

RESULTS

Questionnaires were sent back by 156 patients (72%) and by 160 controls (70%). Patients and controls as well as the two patient subgroups with a lower AHI (10-34/h,

Table 1—General Characteristics

	controls	all patients	p	low AHI (10-34/h)	high AHI ($>35/h$)	p
N	N	160	156	78	78	
age in y years (SD)	56.2 (12.5)	56.5 (10.4)	n.s.*	58.6 (10.3)	55.5 (10.9)	n.s.*
males in %	92	90	n.s.**	94	87	n.s.**
alcohol in glasses/week	6.7	6.5	n.s.*	6.5	6.4	n.s.*
subjects taking sleep drugs (%)	2 (1.3%)	7 (4.5%)	n.s.**	4 (5.1%)	3 (3.8%)	n.s.**
body mass index (SD)	26.3 (4.7)	31.7 (6.9)	$<0.001^*$	29.4 (5.8)	34.0 (7.1)	$<0.001^*$
Epworth sleepiness score (SD)	7.2 (4.7)	12.9 (5.5)	$<0.001^*$	11.8 (4.9)	13.9 (5.8)	$<0.05^*$
No of drivers (%)	140 (87)	130 (83)	n.s.**	68 (87)	62 (79)	n.s.**
mean km/driver/year	14'160	19'416	$<0.01\text{\$}$	19'742	19'059	n.s.\\$
median km/driver/year	10'000	15'000		15'000	15'000	
median apnea-hypopnea Index				20	50	

*students t-test; **2x2 contingency table Chi-square test; \\$Mann-Whitney U-test

N=78) and higher AHI (>34/h, N=78) were comparable for age, sex distribution, consumption of alcohol and sleeping pills, and for proportion of drivers (Table 1). As expected for an SAS patient group, a strong male predominance was found, which was matched in the controls. Although the range of driven km was similar in patients (15-128'000 km/year) and controls (5-150'000 km/year), the average annual driving distance of the patients was significantly greater than that of the controls ($p<0.001$). The average annual driving distance in the control group of 14'160 km was similar to values obtained from a general male Swiss population, ranging with age dependency from 15'000 to 30'000 km.³⁶ As expected, the body mass index (BMI) as well as the ESS were substantially greater in patients than in controls ($p<0.0001$). An abnormal ESS above 10 was reported by 70% of all patients and by 30% of the control subjects. A greater BMI ($p<0.001$) and a higher ESS ($p<0.05$) were also found in patients with a higher AHI (>34/h) as compared to those with lower AHI (10-34/h).

Patients had significantly more self reported motor vehicle accidents per one million km than controls ($p<0.005$) (Table 2). This difference was due to both a greater number of patients involved in motor vehicle accidents (16 patients = 12.4 % vs. four controls = 2.9 %; $p<0.0005$) as well as the greater frequency of multiple accidents in patients than

in controls during the observation period. Five patients but only one control subject had more than one vehicle accident. The latter control subject had an ESS of 13, indicating an abnormal EDS. The severity of the accidents as estimated from the costs and personal injury was also greater in SAS patients with over half of the accidents causing costs above \$600 as compared to controls with only one of four above \$600. Patients with a moderate to severe SAS (AHI >34/h) were more often involved in motor vehicle accidents (12 patients = 19% vs. 4 patients = 6%; $p<0.05$) and had significantly more motor vehicle accidents per one million driven km than those with a mild SAS (AHI = 10-34/h; vs. 1.1; $p<0.05$). The difference between SAS patients and controls was also significant when only severe vehicle accidents with costs above \$600 or personal injury (Table 2) were considered. It should be noted that the mean of the accident number per one million km per subject (13.0) is different from the number of accidents per one million km calculated for the whole group (5.9), a fact that must be taken into account when comparing between different studies. One could expect that these two values should be identical, but this is not the case in skewed distributions.

No correlation was found between the ESS and the individual accident rate per one million driven km neither in

Table 2—Motor vehicle accidents rates during 3 years

	controls	all patients	p	low AHI (10-34/h)	high AHI (>35/h)	p
No of drivers (%)	140 (87)	130 (83)	n.s.**	68 (87)	62 (79)	n.s.**
1. all motor vehicle accidents						
drivers reporting accidents (%)	4 (2.9%)	16 (12.4%)	< 0.005**	4 (6%)	12 (19%)	< 0.05**
mean accidents/driver/1 Mio km	0.78	6.8	< 0.005§	1.1	13	< 0.05§
accidents per group	5	25		4	21	
accidents/1 Mio km/group	0.8	3.3		0.99	5.9	
2. car accidents >600 \$ and/or personal injury						
drivers reporting accidents (%)	1 (0.7%)	8 (7%)	< 0.05**	2 (3%)	7 (11%)	n.s.**
mean accidents/driver/1 Mio km	0.1	2.5	< 0.01§	0.5	4.7	< 0.05§
accidents/group	1	9		2	7	
accidents/1 Mio km/group	0.2	1.2		0.5	1.97	
3. motor bike accidents						
drivers reporting accidents	2	3		1	2	
accidents/group	3	3		1	2	
4. Working or household accidents						
N	160	156		78	78	
subjects reporting accidents (SD)	2 (1.2%)	12 (7.7%)	< 0.005**	3 (7.6%)	9 (11.5%)	n.s.**
accidents/group	9	42		7	35	

*students t-test; **2x2 contingency table Chi-square test; §Mann-Whitney U-test

the patients nor in the control group. Only when pooling both groups a significant correlation between these two parameters was found (Pearson corr. coeff.=0.218; $p < 0.001$). No independent influence of BMI, alcohol consumption, or use of sleeping pills onto the accident rate was found in any of the study groups. If only persons reporting accidents were compared (Table 3), controls with accidents consumed significantly more alcoholic beverages than patients with accidents ($p < 0.001$).

The mean AHI of those patients reporting at least one accident tended to be greater than the mean AHI of those patients reporting no accident (Mann Whitney U-test; $p = 0.05$) (Table 3).

Accidents at household or work were significantly more frequent in patients than in controls (12 patients = 7.7 % vs. two controls = 1.2 %; $p < 0.005$) (Table 2) and were more frequent in patients with a higher AHI, although this difference did not reach statistical significance.

Under treatment with nCPAP ($n = 85$) the individual mean of motor vehicle accidents per one million km dropped from 10.6 before to 2.7 during treatment ($p < 0.05$) despite the relatively short treatment period of 1 to 40 months (average=15.4 months). A significant reducing effect of the nCPAP treatment was also seen on the ESS ($p < 0.001$) and on the body weight ($p < 0.001$) (Table 4).

In the control group 2.9% of the drivers reported at least one vehicle accident due to sleepiness during the observation period of three years. The overall accident rate of 0.8 motor vehicle accidents per one million km in the control group was clearly greater than the officially reported frequency of 0.02 vehicle accidents per one million km due to "impairment of consciousness, for example, sleepiness", which can be calculated from the official Swiss motor vehicle accident statistics in 1996.³⁷ Severe vehicle accidents with costs above \$600 or personal injury occurred 0.2 per one million km in the control group, which also demonstrates at least a ten fold greater rate in our control group

compared to the Swiss federal statistics values.

DISCUSSION

We performed a retrospective analysis on the rate of motor vehicle and working accidents in patients with sleep apnea. Compared to most previous studies on this topic, this analysis combines several distinct methodological features. First, the diagnosis of SAS was made by full polysomnography, and varying degrees of SAS were considered. Second, we questioned an age- and gender-matched control group recruited from the same outpatient clinic but not referred because of sleep-wake complaints. Third, the accident rate was expressed not only in events/time unit but also in relationship to driven km allowing a comparison with the public statistics. Fourth, the effect of nCPAP on accident rate was assessed in a subset of patients. Finally, and most importantly, a strictly anonymous questionnaire was used.

Our study confirms those previous investigations which found significantly more self-reported motor vehicle accidents in SAS patients than in controls.²⁰⁻²² The main result was an up to fifteen-fold increase of the accident rate per one million driven km in the group with severe SAS compared to normals which is greater than in most earlier studies. Furthermore, the rate of sleepiness induced accidents in our control group is far greater than reported by the official Swiss statistics of traffic accidents under the heading of "accidents due to impairment of consciousness, for example, sleepiness."

Accident Rate in SAS

The mean motor vehicle accidents rate per one million km was 13.0 in the moderate to severe untreated SAS patients and 6.8 over all as compared to 0.78 in the control group. The lower frequency of accidents found in some previous questionnaire studies in SAS patients^{17,23,24} may

Table 3—characteristics of drivers reporting motor vehicle accidents

	subjects reporting at least one accident			no accident		difference in patients (p)
	controls	patients	p	controls	patients	
N	4	16		136	114	
drivers reporting accidents (%)	2.9%	12.4%	< 0.01**	0	0	
vehicle accidents per group	5	25		0	0	
driven kilometers	17'080	10'062	n.s.§	14'074	19'466	n.s.§
alcohol in glasses/week	21.3	5.6	< 0.001§	6.7	7.3	0.01§
drivers taking sleep drugs	0	2		2	5	
body mass index	27.2	35.1	n.s.§	26.3	30.9	0.02§
Epworth sleepiness score	10.5	15.1	n.s.§	7.1	12.9	n.s.§
apnoea-hypopnoea index	n.a.	45		n.a.	36	0.05§

**2x2 contingency table Chi-square test; §Mann-Whitney U-test; n.a. not available

be explained by an unreliably low self-declaration of subjects with higher accident rates.²⁰ A lower response rate of patients with accidents could be another factor leading to a low accident value. Both possibilities cannot not be completely ruled out in the present study either, but are unlikely of major importance due to the trustworthy anonymous procedure used. Also, the response rate of 72% is similar to that in earlier questionnaire studies. Furthermore, other than in some previous studies, this study used a sex and age matched control group not recruited from patients referred to a sleep disorder clinic. This could partly explain the low percentage of controls involved in accidents compared to earlier studies. However direct comparisons are difficult since the observation periods and study parameter also differ between studies. This increase of accident rate is as high as reported by Haraldsson²³ in the most severe group of SAS patients reporting regular sleep spells at the wheel. In contrast, the patients of this study who had only a mild SAS with an AHI of 34/h or less, showed an mean accident rate of 1.1 per one million km, which was not significantly different from the control group. Hence, it should not be concluded from this study that the diagnosis of an untreated SAS should invariably exclude driving a motor vehicle. Findley et al.²⁸ also found an increased accident liability only in patients with "severe SAS" but did not precisely define the degree of sleep induced respiratory impairment. As already reported by others² the severity of the accidents in this study as estimated from the costs and personal injury was found to be greater in SAS patients than in controls corroborating the assumption of motor vehicle accidents caused by falling asleep at the wheel being particularly hazardous.

The greater annual driving distance in SAS patients as compared to controls (Table 1) which was also found by others,^{20,25} is probably due to a diagnostic bias too. Professional drivers are likely to be over-represented in the patient group because they rather seek medical help for their handicap of daytime sleepiness than others. SAS in

patients whose daily activity does not require constant alertness are more likely to go unrecognised. This would, for example, be particularly true for unemployed or retired subjects, characteristics that we did not include in the questionnaires for reasons of confidentiality.

Working accidents were also significantly more frequent in the SAS group than in controls. Of the latter, eight working accidents were caused by one control subject who also reported two sleepiness-induced motor vehicle accidents. This control subject had an abnormal ESS of 13 and therefore was probably suffering from undiagnosed EDS. Since SAS also is suspected of frequently going undiagnosed, there is a good possibility of some SAS patients being included in the control group.

Interestingly, the frequency of 0.78 sleepiness-induced vehicle accidents per one million km in our control group proved much greater than that of the official statistics in Switzerland, which report roughly 1 to 2% of all accidents being due to "impairment of consciousness, for example, sleepiness".³⁷ From this, the calculated relative frequency corresponds to only 0.02 accidents per million km. This suggests a gross underestimation of the vigilance problem by the general public and particularly by the police, who is trained to utterly inquire after possible alcohol or drug influences but seems to neglect sleepiness as an important cause of traffic accidents.

Factors Determining Accident Rate in SAS

Although body mass index and ESS were significantly higher in the patient than in the control group (Table 1), and both parameters also correlated with the AHI within the patient groups, no correlation between the ESS and the accident rate was found within the patient group. This is in agreement with previous studies in a group of mostly severe (AHI=58±3) SAS patients²⁵ or in a population-based sample.²⁹ Only when pooling both groups, patients, and controls in our study, a weak but significant correlation was

Table 4—85 patients treated with nCPAP

	before treatment	during treatment	p
drivers	71	73	n.s.**
drivers with accidents (%)	11 (15.5%)	1 (1.3%)	< 0.005**
drivers with accidents >600\$ (%)	7 (89.9%)	1 (1.3%)	< 0.05**
km/driver/year	17'784	19'282	n.s.§
accidents/1 Mio km/group	4.8	0.7	
mean accidents/driver/1 Mio km	10.6	2.7	< 0.05§
weight in kg (SD)	102 (22)	98 (21)	< 0.001§
Epworth sleepiness score (SD)	13.3 (6.2)	6.7 (4.8)	< 0.001§
alcohol in glasses/week	6.4	5.7	n.s.§
subjects taking sleeping pills (%)	2 (2.3%)	2 (1.2%)	n.s.§

**2x2 contingency table Chi-square test; §Wilcoxon-pairs signed rank test

found between the ESS and the individual accident rate (Pearson corr. coeff.=0.218; $p<0.001$). The different statistical result could be due to the greater number of subjects after pooling both groups and the larger range of the variables. However, whether this result within the pooled groups allows to conclude on a causal relationship between sleepiness and accident rate should be judged cautiously. Other, unknown factors may influence sleepiness and accident rate differentially in the two groups.

A comparison of those patients reporting at least one accident with patients reporting no accidents did not reveal a significant difference in the ESS (Table 3). This is in agreement with the reports of Aldrich et al.¹⁷ and Young et al.²⁹ who found that neither the ESS nor the mean latency of falling asleep in the multiple sleep latency test (MSLT) could predict the accident rate in SAS patients. The subjectively assessing ESS is perhaps not a very good risk predictor because sleepiness tends to be underestimated by SAS patients.^{38,39}

The main question which cannot be answered by this study is why 88% of all patients do not report accidents due to sleepiness although 32% of patients reported an ESS of 15 or above. In this and in previous studies^{40,41} additional specific risk factors for accidents in SAS could not be identified. Hence, the question who should and who should not drive cannot be answered. Since in our study the accident rate in patients with AHI between 10 and 35 was not different from the normal controls, the diagnosis of SAS as such does not seem to be sufficient to predict driving impairment.

The need for further research to establish reliable methods to appropriately assess sleepiness induced impairment of performance is widely recognized.³⁴ The absence of reliable and objective method for evaluating sleepiness itself or its various consequences on performance may explain why the present guidelines to judge driving performance in patients with EDS are so variable in different countries.^{19,36,42}

Alcohol or drug consumption was not different between the control and patient group in this study (Table 1). However, when considering patients and control subjects who were involved in sleepiness-induced motor vehicle accidents (Table 3), alcohol consumption of the four control subjects with accidents was almost four times as high as in control subjects without accidents and as in the 16 SAS patients with accidents. This underlines the important role of alcohol consumption as an alternative cause of sleepiness induced motor vehicle accidents, which is at play independently from SAS.

Finally, treating 85 of the SAS patients with nasal continuous airway pressure (nCPAP) resulted in significant reduction of both the average ESS from 13.3 to 6.7 ($p<0.001$) as well as the mean individual motor vehicle accident rate from 10.6. to 2.7 per million km ($p<0.05$)

which is not different from the control group. Hence, the wide-spread practice of letting SAS patients under successful nCPAP therapy drive again seems justified. However, it is well established that close supervision of the compliance with nCPAP is important. The hours of nCPAP use in our patients were regularly controlled as a part of the routine clinical check, initially after two months and later, once to twice a year.

As is the case in any retrospective questionnaire study, this investigation has several limitations. First, no information could be gathered on individual social and professional habits due to the faithful anonymization of the questionnaires. Therefore it cannot be excluded that SAS patients had more dangerous jobs than controls or that this group included a greater number of professional drivers. However, getting more precise individual information or performing objective individual measurements in addition to the AHI value would seriously jeopardize the main aim of our study—which was to assure the participants on the absolute anonymity of all data. This strategy should minimize the previously recognized problem of underreporting driving accidents.⁴³ Second, no questions were asked on accidents not caused by sleepiness. Patients with sleep disorders tend to underestimate their sleepiness and they may deny sleepiness as a cause of accidents.⁴³ Correcting this bias should increase, rather than decrease the real difference in accident rate between patients and controls. Third, the control group was not a random sample of the general population and, therefore, its rate of accidents might not be representative for the general population. However, since both patient groups were selected from the same outpatient clinic referred either for sleep disorders or for low back pain or carpal tunnel syndrome, there should be no differential report bias between the groups. Fourth, the control patients were not screened for SAS. Exclusion of possible SAS patients among the controls would have most probably increased, rather than decreased, the different accident rate between the groups.

APPENDIX

Method of Polysomnography

Nocturnal polysomnography was performed in the course of the routine diagnostic work-up by using a 12 channel recorder (Neurofax 4400®, Nihon Kohden Corp. Tokyo, Japan) including two EEGs, two electrooculograms (EOG), the electromyograms from the submental muscle and from both anterior tibial muscles. Respiration was monitored by thermocouples (airflow through nose and mouth) and by plethysmography (thorax and abdomen) as well as by transdermal oxymetry (Ohmeda Biox 3700®, Ohmeda Inc. Louisville, CO, USA). All sleep recordings were automatically analysed by a sleep analysing software (Ultrasom®, Nicolet Biomedical Inc. Madison, WI, USA)

and visually corrected when necessary by an experienced physician. Apneas and hypopneas were defined as a reduction of airflow below 25% and 50% of the preceding baseline value respectively for 10 seconds or longer. The apnea-hypopnea index (AHI) was defined as the average number of apneas and hypopneas per hour of sleep.

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