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Original article

Slow wave sleep rebound and REM rebound following the first night of treatment with CPAP for sleep apnea: correlation with subjective improvement in sleep quality ☆

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Abstract

Objective: The purpose of this study was to correlate changes in PSG parameters between the diagnostic polysomnogram (dPSG) and the first night of treatment with continuous positive airway pressure (CPAP) (cpapPSG) to subjective improvement in sleep quality.

Background: In patients with obstructive sleep apnea syndrome (OSAS), therapy with CPAP results in reduction of sleep latency, stage 1 sleep, arousal index (Al) and respiratory disturbance index (RDI), and increase in stage 2 sleep, REM sleep and REM density. No data exists on the differences in polysomnographic (PSG) parameters in patients who have subjective improvement in sleep quality and those who do not.

Methods: We retrospectively reviewed PSG studies of 44 patients with OSAS who presented to the Sleep Disorders Center at Duke University Medical Center. Patient's qualitative assessment of sleep was noted using a Likert-type scale administered the morning after the dPSG and cpapPSG. PSG indices of patients noting subjective improvement were compared to those with no improvement.

Results: Patients noting a subjective improvement in sleep quality showed a decrease in the percentages of stage 1 sleep ($P < 0.001$) and an increase in percentages of stages 3 and 4 sleep (slow wave sleep rebound; $P < 0.007$) and stage REM sleep (REM rebound; $P < 0.008$).

Keywords: Obstructive sleep apnea; Continuous positive airway pressure; Symptoms; REM sleep; REM rebound

1. Introduction

Continuous positive airway pressure (CPAP) therapy is a commonly used method of treatment for patients with obstructive sleep apnea syndrome (OSAS). Patients routinely undergo an initial diagnostic overnight polysomnogram (dPSG) to determine the severity of the apnea. The dPSG, depending on the severity of OSAS, typically demonstrates recurrent apneas, hypopneas and oxygen desaturations with accompanying arousals. Compared to normal controls and depending on the severity of apnea, the patients

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spend a majority of the night in lighter stages of sleep (stages 1 and slow wave sleep) with little or no REM sleep. This is a reflection of the sleep fragmentation seen in individuals with OSAS and is considered to be the cause of excessive daytime sleepiness and poor daytime functioning. This is followed by a CPAP titration study (cpapPSG) to determine the optimal CPAP pressure and response to treatment. Following therapy with CPAP, there is a reduction of sleep latency, percentage of stage 1 sleep, arousal index (Al) and respiratory disturbance index (RDI), and increase in percentages of stage 2 sleep, REM sleep and an increase in frequency of eye movements during REM [1,2]. Although most patients report subjective improvement in sleep quality after the CPAP titration study, many do not. No data exists on the differences between these two groups of patients. The purpose of this study was to compare changes in dPSG and cpapPSG parameters in patients who noted an improvement in sleep quality compared to those who did not. We hypothesized that patients reporting an improvement in sleep quality would exhibit less fragmentation, and therefore better restoration of their sleep stages, following CPAP than those reporting no improvement.

2. Materials and methods

2.1. Subjects

We reviewed polysomnography reports of all patients (age $>$ 17 years) that presented to the Duke Sleep Disorders Center for evaluation of OSAS. Only those patients who had two separate studies (dPSG and cpapPSG) performed at our center were considered for enrollment. These patients were diagnosed with OSAS based on the dPSG and only those patients who had a RDI $>$ 10 were eligible for this study. Patients with a movement arousal index $>$ 5 (with no other evidence of sleep apnea) and those with predominantly central sleep apnea were excluded. Forty-four consecutive patients who met the above criteria were entered into the study. The patients were categorized depending on the RDI; mild (RDI 10–20; $N = 12$), moderate (RDI 21–40; $N = 24$) or severe (RDI $\geq$ 40; $N = 8$) OSAS. We noted patient's qualitative assessment of their sleep the morning after each study. This is routinely assessed in our laboratory by means of a Likert-type scale, ranging from 1–10, with 1 representing an extremely poor night's sleep, 5 representing a typical night and 10 representing the best possible sleep [3–5]. Each patient was asked to score the night (independent of any input from their spouses) based on what he/she perceived would be an average night's sleep.

2.2. Data collection

The dPSG and cpapPSG studies were performed using Grass instruments model 8 and 9 machines (West Warwick, RI, USA) and Vangard Watchman Digital Systems (Cleveland, OH, USA). Six EEG channels were recorded using Grass disc electrodes (West Warwick, RI, USA) to assess sleep stages (C_3A_2, C_4A_1, 0_1A_2, Fp_10_2, T_3C_Z, C_ZT_4). Nasal and oral airflow was measured using Protech thermocouple airflow sensors (Woodinville, WA, USA) placed into each nares and in front of the mouth respectively. Thoracic and abdominal effort was measured using Grass Piezo Trace respiratory effort transducers (West Warwick, RI, USA). An EPM systems snore sensor (Midlothian, VA, USA) was placed along the trachea in some patients. The majority of patients had a manual assessment of their snoring by the technician. EMG activity was recorded from the mentalis muscle using a pair of Grass disc electrode placed under the chin. Leg movements were recorded using standard Grass disc electrodes placed over the tibialis anterior muscles on both legs. The oxygen saturation was recorded using a finger probe connected to Ohmeda Biox 3700 pulse oxymeter (Helsinki, Finland; averaging time 6 s). CPAP titration was performed using a Respironics Inc. BIPAP airway management system (Pittsburgh, PA, USA). CPAP was routinely started at a pressure of 4 cm of water. The pressure was increased by 1 cm of water if significant snoring, arousals or hypopneas occurred and by 2 cm of water if significant apneas or oxygen desaturations were noted. The dPSG and cpapPSG studies were scored by a technician certified in polysomnography using 30 s epochs using the criteria established by Rechtschaffen and Kales and by definitions established by the International Classification of Sleep Disorders: Diagnostic and Coding

Manual, 1997 [6,7]. In patients who had no REM sleep, the REM latency was taken to be the same as the total sleep time (for statistical purposes). The RDI was calculated as the number of hypopneas (>50% reduction of thermocouple output for 10 s or longer associated with an arousal or a ≥3% reduction of oxygen saturation) and apneas (>90% reduction of thermocouple output lasting 10 s associated with an arousal or a ≥3% reduction in oxygen saturation) per hour [7,8]. The patients were given a questionnaire the morning after the dPSG and cpapPSG studies. In addition to the Likert type scale, they were asked questions regarding the extent of disruption of their sleep and their perception of total sleep time. The patients were not aware of their initial dPSG scores at the time that they were asked to score their sleep following the cpapPSG titration. The morning following the cpapPSG study, the studies were reviewed by a physician who established the optimal CPAP pressure to treat the patient. This was the pressure that best eliminated or diminished the apneas and arousals. On some occasions ($N = 5$) the CPAP titration was considered inadequate because the patients continued to have a significant number of apneas and arousals on the maximum pressure reached during the studies.

2.3. Statistical analysis

A correlation analysis of PSG indices (for the total duration of the study) was performed for both groups; patients noting a subjective improvement with CPAP (cpapPSG scale score−dPSG scale score > 0; Group 1) to those who had no improvement with CPAP (cpapPSG scale score−dPSG scale score ≤ 0; Group 2). We then assessed if there was a difference based on the degree of improvement in subjective sleep quality. Group 1 was divided into two subgroups; mild improvement (cpapPSG scale score−dPSG scale score = 02; Group 1A) and moderate improvement (cpapPSG scale score dPSG scale score ≤3; Group 1B). The PSG indices for these groups were then compared. To assess if improvement in subjective sleep quality was a function of apnea severity, the patients were also divided into three groups based on the dPSG RDI; mild (RDI > 10, <20), moderate (RDI ≥ 20, <40) or severe (RDI ≥ 40) OSAS [7,8]. dPSG and cpapPSG indices between these three groups were analyzed using repeated measures mixed multiple analysis of variance (MANOVA). Although the technicians used a standard protocol to increase the CPAP pressure, the time that it took to achieve an adequate pressure was different for each individual patient. To assess if this could have played a role in the patients symptoms, we also performed a correlation analysis between the change in each patients score to the time it took to achieve the therapeutic pressure and also between the time the patient was on that pressure. Student's t-tests were used for normally distributed data sets and non-parametric testing (Wilcoxon matched pairs tests) was used for skewed data. Only P-values <0.05 were considered significant. Data was expressed as 'mean ± standard deviation.'

3. Results

3.1. Patient characteristics

A total of 44 (M/F = 36/8; mean age: 51.6 years) patients were enrolled. The mean interval between the dPSG and the cpapPSG was 47 days. Thirty-four patients (M/F: 28/6) noted subjective improvement in sleep quality with CPAP (Group 1; mean patient score changed from 4.3–7.7) and ten patients (M/F: 7/3) did not note an improvement (Group 2; mean patient score changed from 6.65–5.6). The patients who felt subjective improvement were younger (48.9 ± 12.6 years) compared to patients who did not note an improvement (60.9 ± 11.4 years).

3.2. Subjective improvement and changes in PSG indices

All the patients in our study showed a decrease in the Al and RDI (Table 1). However, patients noting a subjective improvement in sleep quality showed an decrease in the percentages of stage 1 sleep ($P < 0.001$) and an increase in stages 3 and 4 sleep ($P < 0.007$) and stage REM sleep ($P < 0.008$). This increase in the percentage of REM sleep has been called REM rebound by other investigators [1,7,9]. We then wanted to assess if there was a difference between the PSG parameters based on the degree of subjective improvement. In patients with mild

Table 1
Changes in PSG indices (mean ± SD) in the patients who noted an improvement (cpapPSG Likert-scale score-dPSG Likert-scale score > 0; Group 1) and those that did not (cpapPSG Likert-scale score-dPSG Likert-scale score ≤ 0; Group 2)[a]

	Group 1			Group 2		
	Improvement ($N = 34$)			No improvement ($N = 10$)		
Age (years)	48.9 ± 12.7			60.9 ± 11.4		
Latency to adequate CPAP pressure (min)	137.4 ± 75.9			127.1 ± 77.5		
PSG indices	dPSG	cpapPSG	*P*-value	dPSG	cpapPSG	*P*-value
Total sleep time (min)	330.5 ± 66.7	333.2 ± 55.3	NS	316.7 ± 64.9	316.4 ± 64.4	NS
Sleep latency (min)	11.4 ± 15.7	8.1 ± 8.6	NS	10.4 ± 9.5	12.9 ± 7.4	<0.04
REM latency (min)	137.6 ± 85.2	102.2 ± 81.5	<0.04	114 ± 79.3	115.9 ± 70.6	NS
Sleep efficiency (%)	82.1 ± 11.6	85.3 ± 9.4	NS	79 ± 14.1	78.1 ± 16.6	NS
Arousal index (per hour)	45.0 ± 19.9	18.4 ± 10.3	<0.001	43.4 ± 16.7	18.8 ± 11.0	<0.005
Respiratory disturbance index (per h)	30.6 ± 19.1	3.1 ± 4.2	<0.001	30.1 ± 14.5	4.6 ± 7.5	<0.006
Stage 1 sleep (%)	15.4 ± 10.9	8.2 ± 4.4	<0.001	9.3 ± 9.3	10.5 ± 8.2	NS
Stage 2 sleep (%)	61.4 ± 11.9	57.6 ± 12.3	NS	65.1 ± 8.8	60.9 ± 10.1	NS
Stage 3 and 4 sleep (%)	12.2 ± 8.3	16.8 ± 12.3	<0.007	12.8 ± 7.5	13.9 ± 11.4	NS
Stage REM sleep (%)	12.1 ± 8.3	17.2 ± 8.1	<0.008	12.7 ± 9.0	15.6 ± 8.6	NS
Patient score	4.4 ± 1.7	7.7 ± 1.4	<0.001	6.6 ± 2.2	5.6 ± 2.4	<0.008

[a] Significant *P*-values are mentioned for the different PSG parameters. NS, not significant.

improvement (Group 1A) there was a significant slow wave rebound following CPAP ($P < 0.005$). Although there was an increase in REM percentage, it was not significant. In patients who noted moderate improvement (Group 1B), there was a significant rebound of REM sleep ($P < 0.03$). There was a trend for an increase in slow wave sleep percentages, but it was not significant.

Table 2
Changes in PSG indices (mean ± SD) in the patients based on severity of obstructive sleep apnea (mild OSAS = RDI 10–20; moderate OSAS = RDI 20–40; severe OSAS = RDI > 40) analyzed by repeated measures mixed MANOVA[a]

	Mild OSAS ($N = 12$)		Moderate OSAS ($N = 24$)		Severe OSAS ($N = 8$)		*P*-value
Age	47.8 ± 8.9		55.6 ± 13.5		43.5 ± 13.5		NS
PSG indices	dPSG	cpapPSG	dPSG	cpapPSG	dPSG	cpapPSG	
Total sleep time (min)	335.1 ± 78.4	313.9 ± 59.3	326.7 ± 52.1	332.2 ± 57.8	317.9 ± 88.4	344.1 ± 53.3	NS
Sleep latency (min)	15.4 ± 17.9	10.5 ± 10.3	11.1 ± 14.1	9.6 ± 8.3	5.1 ± 6.5	5.9 ± 6.3	NS
REM latency (min)	151.7 ± 87.1	132.6 ± 68.3	113.4 ± 71.2	92.8 ± 57.9	159.6 ± 107.7	101.6 ± 133.4	<0.0001
Sleep efficiency (%)	81.1 ± 14.0	80.7 ± 10.4	82.0 ± 9.9	82.7 ± 13.0	80.0 ± 16.3	91.0 ± 4.5	NS
Arousal index (per hour)	40.6 ± 14.8	21.8 ± 7.9	42.1 ± 19.0	19.5 ± 11.7	58.6 ± 20.6	10.7 ± 4.1	<0.001
Respiratory disturbance index (per hour)	15.5 ± 3.4	1.9 ± 1.6	27.8 ± 5.9	4.4 ± 6.6	61.2 ± 19.2	2.7 ± 2.1	<0.001
Stage 1 sleep (%)	14.0 ± 1.1	9.8 ± 5.6	11.4 ± 9.1	9.2 ± 5.7	21.9 ± 12.6	5.6 ± 3.4	NS
Stage 2 sleep (%)	57.0 ± 11.0	58.2 ± 13.2	64.4 ± 11.8	58.9 ± 10.8	63.5 ± 8.7	57.2 ± 14.3	NS
Stage 3 and 4 sleep (%)	15.6 ± 12.1	18.9 ± 16.2	10.6 ± 8.3	14.6 ± 9.6	12.8 ± 24.2	16.9 ± 11.7	NS
Stage REM sleep (%)	13.3 ± 9.7	13.2 ± 8.1	13.1 ± 7.4	17.1 ± 6.8	7.9 ± 8.7	21.5 ± 10.2	<0.0001
Patient score	4.4 ± 1.6	7.3 ± 1.8	5.4 ± 2.1	7.6 ± 1.9	4.2 ± 2.2	6.9 ± 2.1	NS

[a] Significant *P*-values are mentioned for the different PSG parameters. NS, not significant.

3.3. Relationship with apnea severity

We then attempted to define the changes in PSG indices with respect to initial apnea severity irrespective of improvement (Table 2). The results of a repeated measures mixed MANOVA indicated a significant effect of group [$F(11,29) = 3.69$, $P < 0.0001$], time [$F(11,29) = 37.97$, $P < 0.0001$), and interaction of group × time [$F(22,58) = 4.36$, $P < 0.0001$). Thus, significant differences between all three levels of apnea were found. All three groups had a significant decrease in the Al and the RDI. Univariate follow-ups indicated significant differences for the Al scores ($P < 0.0001$), RDI ($P < 0.0001$) and for the percentage of REM sleep ($P < 0.0001$). Difference scores were then calculated for posthoc analyses of the significant univariate findings, which was accomplished using the Bonferroni procedure. This analysis revealed that patients with severe apnea had the greatest increase in percentage of REM sleep ($P < 0.0001$) and that patients with mild apnea had no significant change in their percentage of REM sleep ($P < 0.0001$). Patient's with severe apnea demonstrated the greatest decrease in RDI ($P < 0.0001$), while patient's with mild apnea demonstrated the least decrease in RDI ($P < 0.0001$). Posthoc analyses also demonstrated that patient's with severe apnea had the largest decrease in their Al score ($P < 0.0001$) and patient's with mild apnea had the smallest reduction in their Al score ($P < 0.0001$).

3.4. Effect of differences in CPAP titration

Despite using a standard protocol for increasing the CPAP pressure for the cpapPSG's, the time it took to achieve an adequate pressure to eliminate the apneas, hypopneas and arousals was different for each patient. The most appropriate pressure to treat a particular patient was deemed to be one that either completely or adequately eliminated the apneas. A physician reviewing the study the following morning determined this. There were several patients ($N = 5$) in whom the CPAP titration was considered to be inadequate since there were still a significant number of apneas (>5–10/h) seen on the highest pressure reached for that patient. The latency to achieve an adequate pressure was determined for the remaining 39 patients (158.4 ± 96.8 min). No statistically significant correlation was seen between the latency to adequate CPAP pressure and change in patient score or degree of slow wave rebound or REM rebound. Conversely there was no correlation between the time the patients spent after reaching the optimal pressure and subjective improvement or slow wave or REM rebound. Of those patients who were considered to have had an inadequate CPAP titration ($N = 5$), all felt subjective improvement (cpapPSG scale score −dPSG scale score = 3.4 ± 1.1%), four had REM rebound (8.6 ± 8.0%), and three had slow wave sleep rebound (9.0 ± 12.4%). There was also no statistically significant difference in latency to adequate CPAP pressure in patients who had an improvement in subjective sleep quality versus those who did not.

4. Discussion

Overnight PSG studies in patients with OSAS usually demonstrate sleep disruption by frequent obstructive respiratory events (apneas and hypopneas) and arousals [9]. The result is non-restorative sleep and daytime hypersomnolence. In addition to cardiopulmonary disease, other complications of OSAS include depression, memory deficits, nocturnal panic disorder and chronic fatigue [10–14]. Treatment options were initially limited to surgery and attempting to eliminate coexisting conditions. It was not until the mid 1980s that CPAP became a proven and preferred method of treatment [15]. CPAP has been shown to eliminate apneas and arousals and results in more sustained and less disrupted sleep [2]. There is also an increase of stages 3 and 4 and REM sleep that has been described as slow wave sleep rebound and REM rebound respectively [1,2].

Several authors have attempted to evaluate the effects of long-term CPAP therapy on symptoms. Psychological symptoms were assessed using a profile of Mood States questionnaire in seven patients with OSAS and showed a general improvement in mood at the end of 2 months of treatment [16]. The effects of CPAP were studied on a group of 32 patients with OSAS and was found to improve daytime functioning, mood, vigilance, mental flexibility and attention

compared to placebo [17,18]. Others have performed more detailed neuropsychological testing before and after CPAP therapy and have found improvements in vigilance and affect [19–21]. CPAP not only affects these individual parameters but also results in an overall improved quality of life [22–24]. The bed partners of these patients also appreciate improvements in symptoms and personal relationships [25]. Recent work also shows that treatment with therapeutic levels of CPAP, compared with sub-therapeutic levels, reduces excessive daytime sleepiness and self-reported heath status [26]. The goal of this study was to identify PSG correlates of subjective improvement in sleep quality following the first night of treatment with CPAP. This had previously never been studied.

Although CPAP clearly benefits the majority of patients with OSAS, there are some who feel little or no improvement. The objective of our study was to attempt to define the differences in PSG characteristics between patients who felt subjective improvement after the first night of therapy with CPAP and those who did not. We decided to use a Likert-type scale to determine subjective improvement. Likert-type scales are used routinely in a variety of clinical circumstances and have been validated for other conditions [3–5].However, no previous work to evaluate this phenomenon exists and therefore there is no validated scale available. Our objective was to simply assess how patients felt the morning after their studies compared to what they perceive, as should be a typical night's sleep. We used a Likert-type scale that is routinely in our laboratory to assess how patients feel the night after their PSG studies. We believe that we were able to get a good estimate of improvement following the cpapPSG by subtracting the cpapPSG score from the dPSG score (probably comparable to an average night of sleep at home). There are some drawbacks. Several other factors may play a role on how a patient rates the quality of his/her sleep which include, but are not limited to, difficulty sleeping in an alien environment (the so called first night effect), aggravation of using a CPAP mask and external noise during the study. However, untill the time that a scale is validated this was the only method available to us.

All patients in our study had a decrease in the Al and RDI with CPAP. However, only patients that noted subjective improvement with CPAP had significantly reduced percentages of stage 1 sleep, shortened REM sleep latency and an increase in the percentages of stages 3 and 4 and REM sleep. This was despite the observation that the arousal indices were similar between the two groups. This is consistent with studies dealing with sleep fragmentation. Sleep fragmentation results in increased daytime somnolence, confusion, poor memory and attention deficits [27–32]. Recovery of performance following restoration of sleep patterns with near normal sleep stage percentages has been reported even after a single night of treatment [33]. Studies of selective deprivation of slow wave and REM sleep have failed to conclusively demonstrate which of these are more responsible for producing cognitive and attention deficits [31,34–36]. It seems that the major predictor of performance improvement is related to decrease in fragmentation and an increase in total sleep time. All the patients in our series showed a decrease in the Al. However, there was no significant increase in the total sleep time. This is probably because the PSG studies are performed during fixed hours in our sleep lab and the patients are awakened at the end of the allotted time.

Although patient perceptions of their sleep quality has not been directly evaluated, it is reasonable to assume that they would feel symptomatically better following restoration of a near normal sleep state and improvement in sleep efficiency. This is what we observed in our group of patients. All patients had a decrease in the Al and RDI. However, only patients who had a significant increase in the amount of slow wave and REM sleep felt an improvement following CPAP. During the cpapPSGs, pressures are slowly increased to eliminate apneas. It is reasonable to assume that if the adequate CPAP pressure were reached earlier during a study, there would be less sleep fragmentation during the remaining period. This was our observation individually for each patient. We found that the latency of reaching an adequate pressure (i.e. one that eliminated all or most apneas) was independent to subjective improvement in sleep quality noticed by patients. We found also that there was no correlation between the time patients spent after reaching the optimal pressure and improvements in sleep quality or slow wave or REM rebound. Also amongst the patients who had an

inadequate CPAP titration, three and four patients ($N = 5$) experienced slow wave and REM rebound respectively. This supports the hypothesis that slow wave and REM rebound are independent of other factors in causing subjective improvement. REM rebound has been shown to correlate with oxygen desaturations during the cpapPSG and to a lesser extent with apnea severity [1]. Although we did not evaluate the oxygen desaturations, we also found a correlation between apnea severity and the degree of REM rebound.

The patients who felt no different or worse did not have a significant rebound effect. The reasons why these patients did not have a rebound of sleep stages are unclear. We noticed some difference between the two groups based on their dPSG parameters. The patients who noted subjective improvement had a greater percentage of stage 1 sleep during the dPSG. They also had a lower score on the dPSG compared to those who did note an improvement. This implies that there were some pre-existing differences in subjective sleep quality during the dPSG. We attempted to determine if some patients cited a reason for not feeling an improvement. These included problems tolerating the mask, irritation of the eyes, venting through the mouth and an unsatisfactory laboratory environment. Although all these patients agreed to use CPAP, it is likely that they would be more skeptical of the benefits despite a clear decrease in the Al and RDI. If these patients do not feel a benefit immediately following the first time use of CPAP, they are maybe less likely to continue to use it over a long-term period. It is now well established that there is a subset of patients who eventually stop using CPAP. It may be that a lack of rebound of slow wave and/or REM sleep may be able to define that subset of patients. Focusing more attention on this subset of patients may potentially help increase long-term compliance rates. Our group collected preliminary data but we were able to establish contact with only 28 of 44 patients [37]. Contact was established with 22 patients in Group 1 and six patients in Group 2. Patients in Group 1 (68.2%) and Group 2 (66.7%) were using CPAP at least four nights per week and 2 years following the initial evaluation. Further studies to evaluate this issue are required. There is recent work suggesting the use of Modafinil for use in treating daytime sleepiness in patients with obstructive sleep apnea [38]. The subset of patients who did not respond may be ideal candidates for treatment with medication and can potentially be identified early using this information.

There are several questions that we did not address. Our objective was to evaluate the PSG correlates of subjective improvement the day after first time treatment with CPAP. We did not correlate this to any objective measures of improvement in symptoms such as hypersomnolence by performing multiple sleep latency tests. There is data to suggest that long-term use of CPAP results in continued improvement in physiological and PSG parameters [39,40]. Our study did not address the question of improvement in subjective sleep quality with continued use of CPAP. Also, each of our patients had a single dPSG and cpapPSG. The use of single night recordings has been supported by several authors [41]. Others have, however, argued for performing multiple studies based on the 'first night effect' [42]. This is related to sleeping in an alien environment and several authors have demonstrated longer REM latencies, higher arousal indices and decreased sleep efficiency compared to PSG's performed subsequently over consecutive nights. Since our data lacks a control group, we can neither support nor disprove the existence of this phenomenon.

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References

[1] Aldrich M, Eiser A, Lee M, Shipley J. Effects of continuous positive airway pressure on phasic events of REM sleep in patients with obstructive sleep apnea. Sleep 1989;12:413–419.

[2] Issa FG, Sullivan CE. The immediate effects of nasal continuous positive airway pressure treatment on sleep pattern in patients with obstructive sleep apnea. Electroenceph Clin Neurophysiol 1986;63:10–17.

[3] Ahearn EP. The use of visual analog scales in mood disorders: a critical review. J Psychiatr Res 1997;31(5):569–579.

[4] Bond A, Lader M. The use of analogue scales in rating subjective feelings. Br J Med Psychol 1974;47:211.

[5] Likert R. A technique for the measurement of attitudes. Arch of Psychol 1932;22:1–54.

[6] Rechtschaffen A, Kales A. A manual of standardized termi-

nology, techniques and scoring system for sleep stages in human subjects. Los Angeles CA: UCLA Brain Information Service/Brain Research Institute, 1968.

[7] American Sleep Disorders Association. The international classification of sleep disorders: diagnostic and coding manual. American Sleep Disorders Association. Rochester, MN, 1991.

[8] Radtke RA. Sleep disorders: laboratory evaluation. In: Daly DD, Pedley TA, editors. Current practice of clinical electroencephalography, New York: Raven Press, 1990. pp. 561–592.

[9] Eiser AS, Aldrich MS, Lee ML, Shipley JE. REM rebound in successful treatment of obstructive sleep apnea with nasal CPAP. Sleep Res 1988;17:173.

[10] Buchwaid D, Pascualy R, Bombardier C, Kith P. Sleep disorders in patients with chronic fatigue. Clin Infect Dis 1994;18(Supp 1):S68 72–77.

[11] Edlund MJ, McNamara ME, Millman RP. Sleep apnea and panic attacks. Compr Psychiatry 1991;32(2):1302.

[12] Kaplan R. Obstructive sleep apnoea and depression–diagnostic and treatment implications. Aust New Zealand J Psychiatry 1992;26(4):586–591.

[13] Kelly DA, Claypoole KH, Coppel DB. Sleep apnea syndrome: symptomatology, associated features, and neurocognitive correlates. Neuropsychol Rev 1990;1(4):323–342.

[14] Mosko S, Zetin M, Glen S, Garber D, et al. Self-reported depressive symptomatology, mood ratings, and treatment outcomes in sleep disorders patients. J Clin Psychol 1989;45:51–60.

[15] Sullivan CE, Lssa FG, Berthon-Jones M, Eves L. Reversal of obstructive sleep apneaby continuous positive airway pressure applied through the nares. Lancet 1981:8625.

[16] Derderian SS, Bridenbaugh RH, Rajagopal KR. Neuropsychologic symptoms in obstructive sleep apnea improve after treatment with nasal continuous positive airway pressure. Chest 1988;94(5):1023–1027.

[17] Engleman HM, Cheshire KE, Deary IJ, Douglas NJ. Daytime sleepiness, cognitive performance and mood after continuous positive airway pressure for the sleep apnoea/hypopnoea syndrome. Thorax 1993;48(9):911–914.

[18] Engleman HM, Martin SE, Deary IJ, Douglas NJ. Effect of CPAP therapy on daytime function in patients with sleep apnoea/hypopnoea syndrome. Lancet 1994;343(8897):572–575.

[19] Millman RP, Fogel BS, McNamara ME, Carlisle CC. Depression as a manifestation of obstructive sleep apnea: reversal with nasal continuous positive airway pressure. J Clin Psychiatry 1989;50(9):348–351.

[20] Montplaisir J, Bedard MA, Richer F, Rouleau L. Neurobehavioral manifestations in obstructive sleep apnea syndrome before and after treatment with continuous positive airway pressure. Sleep 1992;15(Supp 6):S1 7–9.

[21] Ramos Platon MJ, Espinar Sierra J. Changes in psychopathological symptoms in sleep apnea patients after treatment with nasal continuous positive airway pressure. Int J Neurosci 1992;62(34):173–195.

[22] Flemons WW, Tsai W. Quality of life consequences of sleep-disordered breathing. J Allergy Clin Immunol 1997;99(2):S750–S756.

[23] Jenkinson C, Stradling J, Petersen S. Comparison of three measures of quality of life outcome in the evaluation of continuous positive airways pressure therapy for sleep apnoea. J Sleep Res 1997;6(3):199–204.

[24] Tousignant P, Cosio MG, Levy RD, Groome PA. Quality adjusted life years added by treatment of obstructive sleep apnea. Sleep 1994;17(1):52–60.

[25] Kiely JL, McNicholas WT. Bed partners' assessment of nasal continuous positive airwaypressure therapy in obstructive sleep apnea. Chest 1997;111(5):1261–1265.

[26] Jenkinson C, Davies RJ, Mullins R, Stradling JR. Comparison of therapeutic and subtherapeutic nasal continuous positive airway pressure for obstructive sleep apnea: a randomized prospective parallel trial. Lancet 1999;353(9170):2100–2105.

[27] Bonnet MH. Effect of sleep deprivation on sleep, performance and mood. Sleep 1985;8:11–19.

[28] Bonnet MH. Performance and sleepiness as a function of frequency and placement of sleep disruption. Psychophysiology 1986;23:263–271.

[29] Bonnet MH. Performance and sleepiness following moderate sleep disruption and slow wave sleep disruption. Physiol Behav 1986;37:915–918.

[30] Bonnet MH. The effect of sleep fragmentation on sleep and performance in younger and older people. Neurobiol Aging 1989;10:21–25.

[31] Bonnet MH. Sleep Deprivation. In: Kryger MH, Roth T, Dement WC, editors. second edition. Principles and practice of sleep medicine. WB Saunders, New York, 1994. pp. 50–67.

[32] Downey R, Bonnet MH. Performance during frequent sleep disruption. Sleep 1987;10:354–363.

[33] Fenz WD, Graig JG. Autonomic arousal and performance during sixty hours of sleep deprivation. Percept Mot Skills 1972;34:543–553.

[34] Agnew HWJ, Webb WB, Williams RL. Comparison of stage four and REM sleep deprivation. Percept Mot Skills; 1967;24:818–851.

[35] Johnson LC, Naitoh P, Moses JM, Lubin A. Interaction of REM deprivation and stage 4 deprivation with sleep loss. Experiment 2. Psychophysiology 1974;11:147–159.

[36] Lubin A, Moses JM, Johnson LC, Naitoh P. The recuperative effects of REM sleep and stage 4 sleep on human performance after complete sleep loss. Experiment 1. Psychophysiology 1974;11:133–146.

[37] Verma A, VanLandingham KE, Husain AM, Radtke RA. Compliance with CPAP for sleep apnea is independent of subjective improvement after the first night of treatment. J Clin Neurophysiol 1999;16(2):181.

[38] Arnulf I, Homeyer P, Garma L, Whitelaw WA, et al. Modafinil in obstructive sleep apnea-hypopnea syndrome: a pilot study in 6 patients. Respiration 1997;64(2):159–161.

[39] Chaouat A, Weitzenblum E, Kessler R, Oswald M, et al. Five-year effects of nasal continuous positive airway pressure in obstructive sleep apnea syndrome. Eur Respir J 1997;10(1):2578–2582.

[40] Sforza E, Lugaresi E. Daytime sleepiness and nasal continuous positive airway pressure therapy in obstructive sleep apnea syndrome patients: effect of chronic treatment and 1 night therapy withdrawal. Sleep 1995;18(3):195–202.
[41] Mendelson WB. Use of the sleep laboratory in suspected sleep apnea syndrome: is one night enough? Cleve Clin J Med 1994;61(4):299–303.
[42] Toussaint M, Luthringer R, Schaltenbrand N, Carelli G, et al. First night effect in normal subjects and psychiatric inpatients. Sleep 1995;18(6):463–469.