

# Effect of Continuous Positive Airway Pressure vs Placebo Continuous Positive Airway Pressure on Sleep Quality in Obstructive Sleep Apnea\*

José S. Loredo, MD; Sonia Ancoli-Israel, PhD; and Joel E. Dimsdale, MD

**Study objectives:** Continuous positive airway pressure (CPAP) therapy has become the treatment of choice for obstructive sleep apnea (OSA). However, the efficacy of CPAP therapy has not been evaluated against a suitable control. We investigated the effectiveness of CPAP therapy in improving sleep quality in patients with OSA. We hypothesized that CPAP improves sleep quality.

**Patients:** Forty-eight CPAP-naïve OSA patients were evaluated. None were receiving antihypertensive medications, and none had major medical illnesses.

**Design:** Patients were randomized to receive either CPAP or placebo CPAP (CPAP at an ineffective pressure) for 7 days in a double-blind fashion. Forty-one patients completed the protocol. Sleep quality variables, arousals, sleep arterial oxygen saturation (SaO<sub>2</sub>), and respiratory disturbance index (RDI) were assessed at baseline, after 1 day of treatment, and after 7 days of treatment. Repeated measures analysis of variance was used to evaluate the effects of treatment, time, and the interaction of the two.

**Results:** As expected, CPAP lowered RDI and number of arousals, and increased SaO<sub>2</sub> over time ( $p = 0.001$ ). Contrary to expectations, both CPAP and placebo CPAP had comparable effects on sleep quality as assessed by sleep architecture, sleep efficiency, total sleep time, and wake after sleep onset time.

**Conclusions:** This study confirms the effectiveness of CPAP in lowering the number of arousals and the RDI, and in raising SaO<sub>2</sub>. However, our data suggest that short-term CPAP is no different than placebo in improving sleep architecture. Further evaluation of the effectiveness of CPAP using a suitable placebo CPAP in prospective randomized studies is needed

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**Key words:** continuous positive airway pressure; obstructive sleep apnea; placebo continuous positive airway pressure; sleep architecture; sleep quality

**Abbreviations:** BMI = body mass index; CPAP = continuous positive airway pressure; EMG = electromyogram; OSA = obstructive sleep apnea; PSG = polysomnography; RDI = respiratory disturbance index; REM = rapid eye movement; SaO<sub>2</sub> = arterial oxygen saturation; SWS = slow-wave sleep; TST = total sleep time; WASO = wake after sleep onset time.

Continuous positive airway pressure (CPAP) therapy has become the treatment of choice for the control of obstructive sleep apnea (OSA). CPAP had been reported to eliminate snoring, abolish apneas and hypopneas, correct nocturnal oxyhemoglobin desaturation, improve sleep quality, and improve

excessive daytime somnolence.<sup>1–5</sup> Multiple studies also have reported improvements in daytime fatigue, mood, depression, cognitive function, and hypertension with CPAP therapy.<sup>6–9</sup> However, to our knowledge, no randomized, true-placebo-controlled CPAP trials have been performed on any of these outcome measures. Without an appropriate placebo control, it is difficult to understand what role CPAP therapy plays on the clinical improvement noted in OSA patients.

To determine the effectiveness of CPAP in improving sleep quality in patients with OSA, we randomly assigned OSA patients to receive CPAP or placebo CPAP in a double-blind fashion. We hypothesized that CPAP would improve sleep architecture,

\*From the Departments of Medicine (Dr. Loredo) and Psychiatry (Dr. Ancoli-Israel), University of California San Diego, San Diego, CA; and the Department of Psychiatry (Dr. Dimsdale), Veterans Affairs San Diego Healthcare System, San Diego, CA. This research was supported in part by National Institutes of Health grants HL44915, AG02711, AG08415, and RR00827.

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Correspondence to: José S. Loredo, MD, UCSD Medical Center, Department of Medicine, 200 West Arbor Dr, San Diego, CA 92103-8378; e-mail: jloredo@ucsd.edu

respiratory disturbance index (RDI), number of arousals, and arterial oxygen saturation (SaO<sub>2</sub>).

## MATERIALS AND METHODS

### Subjects

All subjects gave informed consent to the protocol, which was approved by the University of California San Diego Institutional Review Board. Forty-eight CPAP-naïve OSA patients were studied at the University of California San Diego Clinical Research Center. Subjects responded to public service advertisements, were referred from community physicians, or were referred by previous subjects. Subjects ranged in age from 30 to 65 years, and their weight was between 1.0 and 1.7 times the ideal body weight, as determined from actuarial tables (Metropolitan Life Insurance Co; New York, NY).<sup>10</sup> Subjects were excluded if they were receiving medications known to affect sleep or if they had congestive heart failure, symptomatic obstructive pulmonary, coronary, or cerebral vascular disease, history of life-threatening arrhythmias, cardiomyopathy, history of psychosis, narcolepsy, or currently abused alcohol or drugs.

### Experimental Design

Potential OSA subjects were prescreened with an unattended overnight home sleep study using a sleep recording system (NightWatch; Healthdyne Technologies; Marietta, GA). If their RDI was  $\geq 20$ , the subjects then were admitted to the clinical research center at 5 PM for a confirmatory overnight full polysomnography (PSG) sleep recording. If on a preliminary interpretation of the PSG recording the RDI was  $\geq 20$ , subjects were considered to have significant OSA and were admitted into the study.

On the second night of admission, qualifying subjects were randomized to receive either traditional nasal CPAP or placebo CPAP for 7 days in a double-blind fashion.

PSG was repeated on the third night after admission (after 1 day of treatment), and patients then were discharged home for 1 week of continuing CPAP or placebo CPAP home treatment. Research staff were in frequent contact with patients to answer questions about mask placement and to encourage compliance with the therapy. All CPAP units (Horizon, model 7353D; DeVilbiss; Somerset, PA) had a hidden compliance clock, which allowed the measurement of the amount of time the CPAP units were switched on.

On the 11th day of the protocol (after 7 days of treatment) the subjects were readmitted to undergo a fourth sleep study.

### Sleep Recordings

Sleep was recorded using a polysomnograph (Model 4412P; Nihon Koden; Irvine, CA) that recorded central and occipital EEG, bilateral electrooculogram, submental and tibialis anterior electromyogram (EMG), ECG, nasal/oral airflow using a thermistor, and respiratory effort using chest and abdominal inductance belts. SaO<sub>2</sub> was monitored using a pulse oximeter (Biox 3740; Ohmeda; Louisville, CO) and was analyzed using computer software (Profox; Escondido, CA).<sup>11</sup> Sleep records were scored according to the criteria of Rechtschaffen and Kales.<sup>12</sup>

Apneas were defined as decrements in airflow of  $\geq 90\%$  from baseline for a period  $\geq 10$  s. Hypopneas were defined as decrements in airflow of  $\geq 50\%$  but  $< 90\%$  from baseline for a period  $\geq 10$  s. The number of apneas and hypopneas per hour were calculated to obtain RDI.

The definition of an arousal from sleep was based on the criteria published in the 1992 American Sleep Disorders Association Report on EEG arousals<sup>13</sup> with some modifications. An arousal was defined as a shift in EEG frequency to alpha or theta for  $\geq 1.5$  s but  $< 15$  s whether or not it was associated with a rise in chin EMG amplitude, a rise in leg EMG activity, or both. Before an arousal could be scored, the subject had to have been sleeping for a minimum of 10 s in the same or the contiguous sleep epoch. A minimum of 10 s of intervening sleep was necessary to score a second arousal. Shifts in EEG frequency were scored from either or both of the EEG derivations (occipital or central). The abrupt appearance of K-complexes was scored as an arousal only if they were accompanied by an obvious shift in EEG frequency. The total arousal index was calculated by dividing the number of arousals by the total sleep time (TST).

### CPAP and Placebo CPAP Titration

In the CPAP-treated group, optimal effective nasal CPAP pressure to minimize sleep apnea was obtained by conventional manual overnight CPAP titration during monitoring with PSG. After standard PSG hookup, the patient was fitted with an appropriately sized nasal CPAP mask (Respironics; Monroeville, PA). After orientation to the function of nasal CPAP, the patient was allowed to sleep while the CPAP pressure was maintained at 2 cm H<sub>2</sub>O. On the appearance of unequivocal obstructive apneas or hypopneas, the CPAP pressure was increased in increments of 2 cm H<sub>2</sub>O until the respiratory events were abolished or until a CPAP pressure of 8 to 10 cm H<sub>2</sub>O was reached. Further pressure titration then was done in increments of 1 cm H<sub>2</sub>O based on the presence of apneas, hypopneas, or snoring associated with arousals. The titration was considered ended when most respiratory events were controlled with CPAP while the patient was in the supine position and in the second or third rapid eye movement (REM) sleep period, or until a pressure of 20 cm H<sub>2</sub>O had been reached. If the frequency of respiratory events was not reduced by  $> 50\%$ , the CPAP therapy was considered suboptimal and the patient was discharged from the study.

Placebo-CPAP subjects underwent a mock titration night during which CPAP pressure was set at 2 cm H<sub>2</sub>O using a CPAP mask (Respironics) containing three 1/4-inch drill holes to create a large air leak. The pressure at the mask varied from 2 cm H<sub>2</sub>O at end-expiration to 1.5 cm H<sub>2</sub>O during inspiration. During this procedure, sleep was monitored with standard PSG as in the CPAP titration protocol, but the pressure was kept at 2 cm H<sub>2</sub>O.

### Statistical Analysis

Only subjects with a confirmed RDI  $\geq 20$  on overnight PSG were included in the analysis. Differences between and within the two treatment groups over time were assessed using repeated-measures analysis of variance. This analysis allowed us to test for a main effect of treatment CPAP vs placebo CPAP, time effect (prior to treatment, after 1 day of treatment, and after 7 days of treatment), and the interaction of time by treatment. A time effect alone would imply that CPAP itself had no specific effect on the variable of interest. A CPAP-by-time interaction would imply that the CPAP-treated group, in particular, responded to treatment over time with a significant response. Statistical analyses were performed using a statistical computer software package (SPSS for Windows 9.0; SPSS Inc; Chicago, IL).

## RESULTS

Table 1 provides the subjects' characteristics. Of the 48 subjects admitted for testing, 1 was removed

**Table 1—Patient Sample Characteristics\***

Characteristics	CPAP (n = 23)	Placebo CPAP (n = 18)
Sex		
Male	17	16
Female	6	2
Age, yr	46.9 ± 8.3	49.8 ± 9.3
BMI	33.0 ± 5.0†	29.1 ± 5.6
Screening SBP	130 ± 16	125 ± 14
Screening DBP	82 ± 8	79 ± 9

\*Values given as mean ± SD. SBP = systolic BP; DBP = diastolic BP.

†Significantly greater than placebo CPAP group (p = 0.024).

from the analysis due to breach of randomization due to severe OSA and severe hypoxemia when using placebo CPAP. A second patient was removed from the analysis due to inability to sleep with the CPAP equipment. Five subjects were not included in the analysis because their RDI on overnight PSG was < 20. The final sample included 41 subjects with an RDI ≥ 20. The subjects were predominantly (75%) male. The subjects were moderately obese with an average weight of 136% of ideal body weight using Metropolitan Life Insurance Company norms.<sup>10</sup> There were no differences between groups in age, BP, and RDI at baseline. Subjects treated with CPAP had a significantly greater body mass index (BMI; p = 0.024; Table 1), and lower mean sleep SaO<sub>2</sub> at baseline (p = 0.011; Table 2). Both patient groups complied equivalently with CPAP or placebo treatment over the 1-week interval at home (> 5 h per night use for each group). The two treatment groups did not differ at baseline on sleep architecture, TST, wake after sleep onset time (WASO), total arousal index, or sleep efficiency (Table 2).

CPAP as compared with placebo CPAP significantly reduced RDI (p < 0.001) and total arousal index (p = 0.001), and significantly increased mean sleep SaO<sub>2</sub> (p < 0.001) over time (time × CPAP interaction).

There was a significant time effect on sleep latency (p = 0.027), percentage of stage 1 sleep (p < 0.001), percentage of slow-wave sleep (SWS; p = 0.001), percentage of REM sleep (p = 0.008), and sleep efficiency (p = 0.031). TST and WASO did not change with time or treatment. There was no correlation between compliance and the posttreatment sleep architecture variables. Using BMI as a measure of covariance did not alter the results.

## DISCUSSION

A wide range of beneficial physiologic, psychological, and cognitive changes in the OSA patient have

**Table 2—Sleep Quality at Baseline and After Treatment\***

Variable	Sleep Quality		
	Baseline	After 1 Day	After 7 Days
Sleep latency, min			
CPAP	9.4 ± 10.0	8.0 ± 6.0	5.3 ± 6.2
Placebo	10.8 ± 12.6	3.5 ± 3.8	5.1 ± 4.9
Stage 1 sleep, %			
CPAP	23.3 ± 24.2	9.5 ± 7.3	9.8 ± 9.3
Placebo	21.9 ± 20.2	13.4 ± 16.0	15.4 ± 17.3
SWS, %			
CPAP	7.4 ± 8.7	16.1 ± 12.2	12.5 ± 10.6
Placebo	6.3 ± 8.6	10.6 ± 10.0	13.0 ± 14.5
REM sleep, %			
CPAP	19.0 ± 7.9	25.8 ± 7.8	26.2 ± 7.0
Placebo	17.7 ± 8.0	22.3 ± 10.2	19.2 ± 8.3
WASO, min			
CPAP	42.6 ± 33.6	26.0 ± 22.9	32.0 ± 18.2
Placebo	49.2 ± 32.7	36.9 ± 26.2	43.0 ± 41.1
TST, min			
CPAP	319.9 ± 50.5	336.2 ± 50.8	334.0 ± 42.6
Placebo	317.1 ± 46.1	318.6 ± 60.4	307.4 ± 83.0
Sleep efficiency, %			
CPAP	83.7 ± 10.7	88.3 ± 9.4	88.1 ± 8.6
Placebo	82.2 ± 11.8	87.9 ± 6.1	83.7 ± 12.9
SaO <sub>2</sub> †			
CPAP	89.1 ± 6.6	95.4 ± 1.4	95.6 ± 1.3
Placebo	93.5 ± 1.7	93.9 ± 2.6	94.1 ± 2.1
Total arousal index†			
CPAP	55.1 ± 24.4	11.6 ± 5.5	14.4 ± 9.5
Placebo	48.6 ± 28.2	33.3 ± 21.2	33.5 ± 23.2
RDI†			
CPAP	56.4 ± 24.1	5.1 ± 5.6	3.3 ± 3.8
Placebo	44.2 ± 25.3	26.1 ± 19.5	28.3 ± 22.7

\*Values given as mean ± SD.

†Significant CPAP × time interaction (p = 0.001).

been attributed to the effects of treatment with nasal CPAP.<sup>6–8</sup> Nasal CPAP was primarily designed to reverse by means of a pneumatic splint the experience of a repetitive collapse of the upper airway by the OSA patient.<sup>14,15</sup> Thus, it has been well documented that CPAP is effective in abolishing the resultant apneas, hypopneas, and associated hypoxia and sleep fragmentation.<sup>4,5</sup> What is not clear is whether CPAP is able to correct the poor sleep quality that is found in OSA patients.

As expected, we found that CPAP effectively reduced RDI and increased mean nocturnal SaO<sub>2</sub>. CPAP also, as expected, effectively abolished arousals (Table 2).

Contrary to our expectations, CPAP was not different than placebo CPAP in improving sleep quality as assessed by sleep architecture, sleep efficiency, TST, and WASO. The significant time effect noted in sleep architecture parameters suggests that the benefits noted in both treatment groups were not spe-

cific to a CPAP effect. Also, compliance was similar between the CPAP and placebo CPAP groups and was not related to sleep architecture.

Our findings differ from those reported in the literature regarding the effect of CPAP on sleep quality. Uncontrolled sleep studies have reported significant reductions in stage 1 sleep and increases in REM sleep and SWS in OSA patients treated with CPAP.<sup>1,2</sup> Improvement in sleep architecture also has been reported in OSA patients using both standard CPAP units and auto-CPAP technology.<sup>3</sup> Had we not used a placebo version of CPAP, we would have concluded that nasal CPAP had a robust effect in improving SWS and REM sleep and in reducing stage 1 sleep. That conclusion is not fully supported by our data.

There are two possible confounding factors affecting our findings<sup>1</sup>: Laboratory-based sleep studies are known to have a first-night effect,<sup>16,17</sup> in which sleep quality tends to be worse than usual during the first night due to the unfamiliar surroundings and equipment. This was suspected in our study, as noted by the high baseline sleep latencies in a population with moderate to severe OSA syndrome. Therefore, an acclimatization effect could have accounted for some of the changes noted in sleep architecture for both the CPAP- and placebo CPAP-treated subjects.<sup>2</sup> Another possibility is that the small CPAP pressure (1.5 to 2 cm H<sub>2</sub>O) used in our placebo CPAP group was having a partial therapeutic effect, as suggested by a significant drop in RDI from baseline to day 1. However, CPAP in the range used in our placebo CPAP is much lower than would be expected to significantly reduce the RDI. Berry and Block<sup>18</sup> showed that a minimum CPAP of 4 to 6 cm H<sub>2</sub>O was required to control the RDI in sleep apnea subjects of comparable weight to ours. Previous reports have demonstrated that a mean CPAP of 4 cm H<sub>2</sub>O (range, 2.0 to 6.0 cm H<sub>2</sub>O) is required just to prevent snoring in nonapneic subjects.<sup>19</sup> In contrast, the average CPAP required to control sleep apnea in our CPAP-treated group was 10 cm H<sub>2</sub>O. Also, in a recent abstract publication, Stradling et al<sup>20</sup> noted a significant placebo CPAP effect on quality of life using CPAP pressures of < 1 cm H<sub>2</sub>O.

Despite the intuition that wearing a CPAP mask with ineffective pressure and extra holes in the mask itself is sleep disruptive, placebo CPAP did not adversely affect sleep latencies, RDI, mean sleep SaO<sub>2</sub>, arousals, sleep architecture, TST, or WASO.

We found six studies in the literature examining the effects of CPAP on OSA that employed some form of placebo control. Four of these studies utilized an oral placebo,<sup>6,21–23</sup> and one randomized patients to receive nasal cannula air as placebo.<sup>24</sup> Only the report by Jenkinson et al<sup>20</sup> used a placebo CPAP set-up that provided ineffective levels of

CPAP (< 1 cm H<sub>2</sub>O). In our opinion, the literature pertaining to the effectiveness of nasal CPAP in the treatment of OSA suffers from a lack of appropriate placebo CPAP control trials. Despite the confounding effects of the first-night effect acclimatization and the possible partial treatment by the placebo CPAP set-up, our findings suggest that CPAP, when compared with a suitable placebo, may not be as effective as has been reported in improving sleep architecture abnormalities of the OSA patient.

This study has a number of limitations. We treated patients for only 1 week because we were concerned about exposing OSA patients to ineffective CPAP pressure for prolonged periods of time. It is possible that a longer trial of CPAP could have demonstrated a clear advantage over placebo CPAP in improving sleep architecture. Although the mean RDI was > 40, indicating moderate to severe OSA, we excluded individuals with major medical comorbidity and patients with severe obesity. Perhaps CPAP would have shown a clearer advantage over placebo CPAP in patients with more severe derangement of sleep architecture. As it turned out, subjects randomized to CPAP were more obese and had lower mean nocturnal SaO<sub>2</sub>, which could imply that their OSA was more severe.

Our null findings concerning CPAP efficacy could reflect insufficient sample size. However, we doubt that sample size accounts for our findings for a number of reasons: First, our sample size was comparable to or better than many studies of sleep architecture response to CPAP. Second, the p values (range, 0.125 to 0.85) for the null findings were far from significant. Third, if in fact we are committing a type II error that would be corrected by a larger sample size, we estimate that we would need to study many more subjects to perceive a significant CPAP by time interaction (sample size, approximately 80 to 330, depending on the variable in question). Such large sample size requirements suggest to us that the magnitude of the short-term effect of CPAP on sleep architecture, if significant, must be small.

Nevertheless, our study confirms the effectiveness of nasal CPAP in correcting OSA and the associated arousals and hypoxia. However, our results suggest that CPAP may not be as effective in improving sleep architecture in the OSA patient during the first 7 days of treatment. Further evaluation of the effectiveness of CPAP using a suitable placebo CPAP in prospective randomized studies is needed.

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