Positional Treatment vs Continuous Positive Airway Pressure in Patients With Positional Obstructive Sleep Apnea Syndrome*

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Objectives: **The aim of this study was to compare the relative efficacy of continuous positive airway pressure (CPAP) and positional treatment in the management of positional obstructive sleep apnea (OSA), using objective outcome measures.**
Design: **A prospective, randomized, single blind crossover comparison of CPAP and positional treatment for 2 weeks each.**
Setting: **A university teaching hospital.**
Patients: **Thirteen patients with positional OSA, aged (mean ± SD) 51 ± 9 years, with an apnea-hypopnea index (AHI) of 17 ± 8.**
Measurements: **(1) Daily Epworth Sleepiness Scale scores; (2) overnight polysomnography, an objective assessment of sleep quality and AHI; (3) maintenance of wakefulness testing; (4) psychometric test battery; (5) mood scales; (6) quality-of-life questionnaires; and (7) individual patient's treatment preference.**
Results: **Positional treatment was highly effective in reducing time spent supine (median, 0; range, 0 to 32 min). The AHI was lower (mean difference, 6.1; 95% confidence interval [CI], 2 to 10.2; p = 0.007), and the minimum oxygen saturation was higher (4%; 95% CI, 1% to 8%; p = 0.02) on CPAP as compared with positional treatment. There was no significant difference, however, in sleep architecture, Epworth Sleepiness Scale scores, maintenance of wakefulness testing sleep latency, psychometric test performance, mood scales, or quality-of-life measures.**
Conclusion: **Positional treatment and CPAP have similar efficacy in the treatment of patients with positional OSA.** ***(CHEST 1999; 115:771–781)***

Key words: CPAP; positional treatment; sleep apnea

Abbreviations: AHI = apnea-hypopnea index; ANOVA = analysis of variance; CI = confidence interval; CPAP = continuous positive airway pressure; EMG = electromyogram; MWT = Maintenance of Wakefulness Test; NREM = non-REM; OSA = obstructive sleep apnea; REM = rapid eye movement

Continuous positive airway pressure (CPAP) has become the standard treatment for most patients with obstructive sleep apnea (OSA).[1] Approximately 80% of all newly diagnosed patients with OSA commence treatment with CPAP.[2] CPAP acts as a pneumatic splint to force the upper airway open during sleep, and thus prevents OSA.[1] It is a highly effective treatment for most patients with OSA—improvements in sleep quality, oxygen saturation during sleep, control of systemic hypertension, cognitive performance, and daytime alertness have all been documented with CPAP treatment of OSA.[3–6]

Despite its efficacy in sleep apnea, however, in several ways CPAP performance falls short of an ideal treatment. Patients treated with CPAP report a spectrum of common complaints including a feeling of claustrophobia, skin irritation around the nose, conjunctival irritation from mask leaks, nasal stuffiness, drying of the oropharyngeal mucosa, mouth leaks, noise from the air compressor, chest discom-

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fort, difficulty exhaling against the positive pressure, and conceptual distaste for the long-term use of the system.[7] Indeed, some 30% of patients with OSA reject CPAP treatment as a long-term treatment option.[2,7,8] In addition, among those who accept CPAP, compliance is highly variable and often thought to be suboptimal.[6,9]

Furthermore, despite its beneficial effect on sleep quality and daytime alertness,[3,4] as well as on cognitive performance,[6,10,11] patients with sleep apnea who are treated with CPAP still retain significant functional deficits. Higher executive cognitive skills in patients with OSA on CPAP treatment remain below those of normal subjects.[11] In addition, normal levels of daytime alertness were not restored in patients with OSA treated with CPAP.[6,11] Pretreatment hypoxemia during sleep[12] and noncompliance with CPAP treatment[6] may account for some of these persisting deficits in patients with OSA who are on CPAP treatment. One other intuitively possible explanation for some of these persisting deficits in cognitive performance and alertness is that they may result from residual sleep disruption caused by discomfort from wearing CPAP at night.

From a clinical perspective, given the known benefits of treatment in OSA, few physicians would hesitate to treat patients with objective evidence of positional OSA, presenting with daytime somnolence. Thus, an important clinical issue is the relative efficacy of different treatments for positional OSA.

Simple behavioral treatment modalities, designed to maintain a nonsupine body position at night, have been effective in treating many patients with positional OSA.[13,14] Although the mechanisms by which these postural changes improve sleep-disordered breathing are not fully understood, the lateral position appears to reduce the tendency for the tongue to relapse posteriorly, making collapse of the pharynx less likely, as compared with the supine position.[14] To date, researchers have focused on improvements in apnea-hypopnea index (AHI) with positional treatments, rather than functional outcomes such as sleep quality, daytime alertness, and cognitive performance.

The aim of this study was to compare the relative efficacy of positional treatment and CPAP in the management of positional OSA, using objective outcome measures.

Materials and Methods

Hypothesis

Positional treatment will provide improved sleep quality and daytime performance, as compared with CPAP, in the management of positional OSA.

Study Design

This was a randomized, single blind crossover comparison of CPAP and positional treatment for 2 weeks each.

Subjects

Fourteen patients with positional OSA were invited to participate in the study. All 14 patients completed the study, but one 59-year-old man was subsequently excluded because he was found on follow-up to have coexisting idiopathic hypersomnolence. Thus, complete data sets on 13 patients (12 men) aged (mean ± SD) 51 ± 9 years, with a body mass index of 30 ± 4 kg/m^2, and a pretreatment mean Epworth Sleepiness Score of 13 ± 1.3, are included in the study. The duration of reported daytime sleepiness preceding the diagnosis of OSA in this study was 7.5 ± 7.1 years. Patients were recruited from outpatients referred to the Sleep Disorders Center, Royal University Hospital, Saskatoon, Canada, for the investigation of daytime hypersomnolence.

Patients with other conditions that might interfere with sleep (respiratory infections, uncontrolled allergies, heart failure, narcolepsy, periodic leg movements) were excluded from the study. All patients gave written informed consent to participate in the study. The University of Saskatchewan Advisory Committee on Ethics in Human Experimentation approved the research protocol and the consent form.

All patients underwent split-night polysomnography, and the raw data were reviewed epoch by epoch the next morning by a clinical polysomnographer.

The criteria for diagnosis of positional OSA in this study were as follows:

AHI during supine sleep that was two or more times the AHI during sleep in the lateral position.

AHI in the lateral position < 15, during a minimum duration of 1 h sleep in the lateral position on overnight polysomnography, and including at least one rapid eye movement (REM) period.

Subjective daytime somnolence.

These criteria for positional OSA are more stringent than those used by others,[13] but this study was designed to assess whether positional treatment would be a viable alternative in clinical practice to CPAP for positional OSA, and it is already clear from the literature that an AHI > 15 is associated with significant morbidity.[15]

The CPAP titration procedure during the diagnostic study was similar to that described by Sanders and colleagues[16]—CPAP was initiated at 4 cm H_2O and was titrated upwards in 2-cm H_2O increments to eliminate gross obstructive apneas and hypopneas, and then 1-cm H_2O adjustments were made with sleep stage and position until the minimum CPAP necessary to eliminate respiratory arousals (obstructive apneas, hypopneas, and repetitive snoring-associated arousals) had been carefully defined both in non-REM (NREM) and REM sleep.[16]

The pretreatment AHI (the number of respiratory arousals per hour of sleep in both supine and nonsupine body positions) was 17 ± 8. There was a marked difference in the AHI and sleep efficiency between the supine and the nonsupine body positions in all subjects, as shown in Table 1.

The protocol of the study is summarized on Figure 1. After diagnosis and CPAP titration, patients were randomized to 2 weeks of treatment with nasal CPAP or positional therapy, followed by crossover to the other modality, with no washout period between. On night 14 of each 2-week study limb overnight polysomnography was performed. On day 15 patients underwent a Maintenance of Wakefulness Test (MWT) and cognitive performance testing, and completed a set of mood scales. Nasal CPAP compliance was monitored using the hour meter readings

Table 1—*Sleep Efficiency and Arousals During the Diagnostic Study Night in Patients With Positional OSA*

	Sleep Efficiency, %		Time in Position, % TST		AHI			Nonrespiratory Arousals		Arousal Index	
Subject	Supine	Lateral	Supine	Lateral	Supine	Lateral	Total	Supine	Lateral	Supine	Lateral
1	77.2	80.0	49.6	50.4	29.8	3.0	16.3	10.0	17.0	33.0	20.0
2	66.1	79.1	21.8	78.2	42.2	1.4	10.3	8.1	2.3	50.3	3.7
3	70.1	88.8	9.7	90.3	56.0	5.0	10.0	9.6	8.4	65.6	13.4
4	65.0	93.0	21.8	78.2	62.3	2.6	15.6	2.3	8.4	64.6	11.0
5	85.3	83.6	20.9	79.1	107.2	8.2	28.9	6.2	12.1	113.8	20.3
6	60.3	82.8	21.8	78.2	127.7	6.4	32.8	7.7	3.6	135.4	12.8
7	72.0	91.2	24.7	75.3	84.4	4.7	24.4	8.9	7.3	93.2	12.0
8	92.0	92.2	61.0	39.0	18.0	2.4	11.9	7.4	7.0	25.4	9.4
9	62.7	89.4	7.3	92.7	149.2	6.8	17.2	0.0	5.1	149.2	11.9
10	91.4	86.7	24.5	75.5	12.0	1.9	4.4*	3.3	8.4	15.3	10.4
11	80.4	79.4	43.4	56.6	37.7	2.2	17.6	9.6	9.6	43.6	11.8
12	83.0	86.1	11.8	88.2	53.6	14.8	19.4	7.7	16.1	61.3	22.5
13	80.0	86.2	14.6	85.4	48.8	4.5	11.0	7.5	10.9	56.3	15.4
Mean	75.8	86.0	25.6	74.4	63.8	4.9	17.9	6.8	8.9	69.8	13.4
SEM	3.0	1.3	39.3	40.3	41.3	4.1	4.7	2.8	4.7	41.3	6.2

*This patient had a clinically significant upper airway resistance syndrome in the supine position with a respiratory arousal index of 17.

on the CPAP units. It was not feasible to objectively monitor compliance with the positional treatment, but patients were asked to indicate on a daily sleep questionnaire whether or not they had used the corresponding treatment (CPAP or positional treatment) on the preceding night.

Positional treatment consisted of a backpack with a soft ball inside, positioned to prevent the subjects from sleeping supine. The size of the ball in the backpack was 10 × 5.5 inches and it was made of semirigid synthetic foam (Canada Games Company Limited; Toronto, Ontario, Canada). A pilot study, conducted among the authors, of various positional treatments found the backpack and ball to be a comfortable positional treatment. The authors wished to test a simple, inexpensive (cost $20 Cdn), and readily available alternative treatment to CPAP in positional OSA.

A 4-h familiarization session was conducted before the study. During this time, patients were instructed in the use of the backpack and ball and CPAP, and underwent familiarization with the psychometric test battery, to minimize learning effects across subsequent assessments. Patients were instructed to use the CPAP unit or backpack and ball all night, every night during each 2-week study period. The CPAP machine was withdrawn from the home during the positional treatment study limb, and the positional device was removed from the patient during the CPAP treatment limb of the study.

The researchers who scored the sleep studies and conducted the psychometric tests were blinded to the modality of treatment being used by the patient.

Patients were instructed to avoid caffeine during the study period, and decaffeinated coffee was supplied. A urine toxicology screen was performed on day 15 of each study limb to screen for use of any drugs that might affect sleep quality or daytime performance.

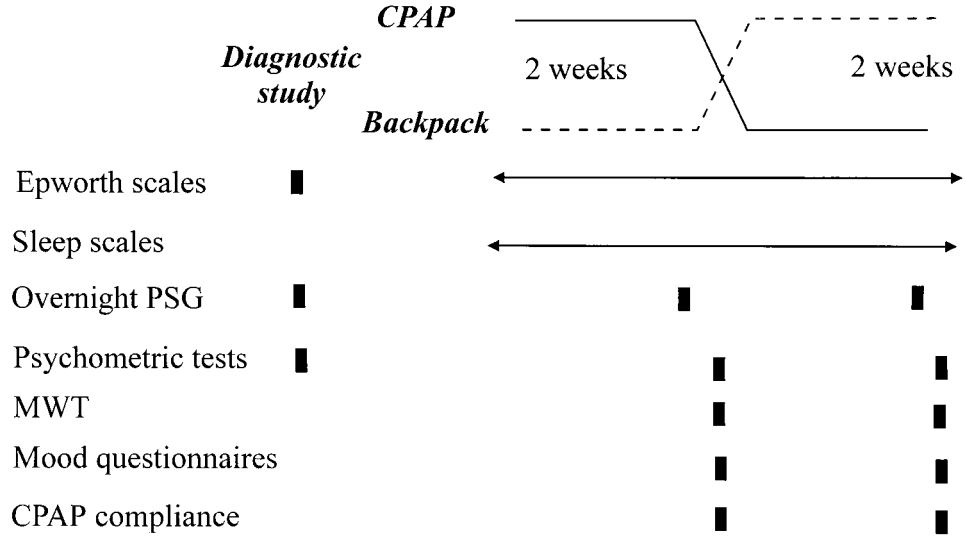

FIGURE 1. Protocol of the study. PSG = polysomnography.

Explanation of Outcome Measures

1. Overnight polysomnography at the end of each 2-week study period to measure objective sleep quality, AHI, number of arousals.
2. Subjective sleep quality, an in-house 5-point scale that patients used daily throughout the study to rate nighttime sleep quality from 1, "no sleep at all," to 5, "perfect night's sleep."
3. Objective daytime sleepiness: MWT,[17,18] on day 15 of each 2-week study limb.
4. Subjective sleepiness: daily Epworth Sleepiness Scale scores.[19]
5. Cognitive performance: 1-h psychometric test battery.
6. Mood and quality-of-life questionnaires.
7. Individual patient treatment preference.

Overnight Sleep Study

Polysomnography included EEG (C4-A1, C3-A2, and O2-A1 derivations), electro-oculogram (2 channels), submental electromyogram (EMG), pulse oximetry, oronasal airflow (oronasal thermistor), chest and abdominal movement (respiratory inductance plethysmography), snore (vibration sensor), intercostal EMG, and anterior tibialis EMG (Melville Diagnostics Inc; Ottawa, Canada). A position sensor was used to monitor position continuously on-line (Rochester Electro-Medical Inc; Tampa, FL). Sleep and arousals were scored according to conventional criteria.[20,21]

Maintenance of Wakefulness Test

Objective daytime sleepiness was measured using the 40-min version of the MWT.[17,18] Sleep-onset latency was defined as the time from lights-out to the first of three consecutive 30-s epochs of stage 1 sleep or any single 30-s epoch of another sleep stage. Four trials were given at 2-h intervals with the first trial beginning 2 h after awakening. The mean sleep-onset latency for the four test periods was taken as the sleep-onset latency result for the MWT.

Psychometric Tests

We measured cognitive performance at baseline, when patient performance had reached a stable plateau on each test (at the end of the familiarization session), and after each 2-week treatment period.

The tests included in the battery were selected to examine vigilance, memory, and higher executive mental function. Several vigilance and memory tests, which had previously been shown to discriminate between sleep apneic subjects and normal subjects, were included.[6,11] Each test was done at the same time of day on both study limbs, after the third MWT trial, to avoid circadian influences on cognitive performance.

The 1-h psychometric test battery included Wechsler Memory Scale form I and II,[22] tests of short- and medium-term working memory; Symbol Digit Modalities Test,[23] a test of attention, concentration, and speeded visual-motor processing; Concentration Endurance Test,[24] a test of vigilance, concentration, and visual speed-scanning; Consonant Trigram,[25] a test of working memory, concentration, and higher executive function; Trail-Making A and B,[26] tests of higher executive function involving visuospatial organization and visuomotor processing at speed; and Purdue Pegboard,[27] a test of manual dexterity and hand–eye coordination.

Mood and Quality-of-Life Questionnaires

Two questionnaires were used to assess the impact of illness in terms of psychologic distress: Hospital Anxiety and Depression Scale[28] and General Health Questionnaire.[29] The UWIST Mood Adjective Checklist[30] was used to assess the mood dimensions of energetic arousal, tense arousal, and hedonic tone. The Nottingham Health Profile[31] assesses the quality of specific areas of life. They were administered in the early part of the day when endogenous mood symptoms tend to be more severe, and at the same time of day on each treatment limb.

At the end of the study patients were asked to state their preferred treatment (CPAP, positional treatment, or no preference).

Statistical Analysis

Data were analyzed using Excel '97 (Microsoft; Redmond, WA) and the SPSS statistical package (SPSS version 6.0; SPSS Inc; Chicago, IL).

One-way analysis of variance (ANOVA) was used to compare the differences in the outcome measures before and after CPAP or positional treatment. Paired *t* tests were used for most variables, but differences in ordinal scales (sleepiness scale, mood questionnaires) were compared using Wilcoxon signed-rank tests. The Bonferroni correction factor was applied when more than one comparison was made on a single variable.

The statistical power of the study was calculated using the standardized difference (the postulated true difference for each variable divided by the standard deviation of the difference between positional treatment and CPAP treatment values for that variable), applied to a nomogram,[32] with correction for paired comparisons. The statistical power to exclude a β error was estimated using the 5% level of significance ($\alpha = 0.05$).

Results

Sleep data from the diagnostic study night are presented in Table 1.

Sleep Architecture and Arousals

Positional treatment was highly effective in maintaining a nonsupine sleeping position (Table 2). CPAP compliance monitoring during the 2-week CPAP treatment limb demonstrated a mean CPAP run time of 6.3 ± 1.0 h/night. There were no significant differences in objective sleep architecture or sleep efficiency between the two treatments (Table 3).

Sleep apnea severity improved with both positional and CPAP treatments (ANOVA: AHI, $p < 0.001$; arousal index, $p = 0.01$) as shown in Figure 2. The AHI was lower on CPAP (mean [SEM], 3.4 ± 0.5) as compared with positional treatment (9.5 ± 1.9; mean difference, 6.1; 95% confidence interval [CI], 2 to 10.2; $p = 0.007$). There were no differences in the total arousal index between the two treatments (CPAP, 15 ± 1.8; positional treatment, 19.4 ± 1.9; mean difference, 4.4; 95% CI, −0.7 to 9.4; $p = 0.08$).

There was a significant correlation between (1) the severity of OSA (AHI) on the diagnostic study (all body positions) and the severity of OSA with the

Table 2—*Efficacy of the Positional Treatment at Overnight Polysomnography*

Subject	Total Sleep Time, min	Supine,* min
1	308	0
2	394	18
3	389	0
4	297	0
5	337	32
6	398	0
7	336	0
8	401	25
9	367	4
10	376	0
11	340	0
12	292	0
13	388	0
Mean	355	6
Median	367	0

*No patient slept supine, but this time refers to the semisupine position while wearing the backpack device.

positional treatment ($r = 0.78$; $p < 0.005$), and (2) the severity of OSA in the lateral position on the diagnostic study and the severity of OSA while using the positional treatment ($r = 0.75$; $p < 0.005$).

There were no differences in the number of nonrespiratory arousals before and after treatment (ANOVA, $p = 0.28$) or between the two treatments (CPAP, 11.4 ± 0.5; positional treatment, 10.2 ± 1.9; mean difference, −1.1; 95% CI, −3.7 to 1.5; $p = 0.36$). The minimum overnight oxygen saturation was higher on CPAP as compared with positional treatment (mean difference, 4%; 95% CI, 1 to 8%; $p = 0.02$). There was no significant difference in the mean overnight oxygen saturation between the two treatments (CPAP, 94 ± 0.5%; positional treatment, 94 ± 0.5%).

Table 3—*Sleep Architecture: Positional Treatment vs CPAP**

	Positional Treatment	CPAP	p Value
Total sleep time, min	355 (11)	350 (15)	0.44
Wakefulness, % TRT	12 (1.4)	15 (2.4)	0.21
Stage 1, % TST	9 (1.1)	11 (2.2)	0.47
Stage 2, % TST	48 (2.6)	46 (3.7)	0.48
Stage 3, % TST	11 (1.1)	12 (1.7)	0.74
Stage 4, % TST	7 (1.6)	8 (1.8)	0.11
Slow wave sleep, % TST	20 (1.9)	22 (3.1)	0.31
REM sleep, % TST	24 (1.5)	26 (2.8)	0.71
Sleep efficiency, TST/TRT	82 (2.1)	84 (2.5)	0.51

*TRT = total recorded time; TST = total sleep time. Values are mean (SEM).

Daytime Sleepiness

The MWT sleep-onset latencies were similar on CPAP (32.9 ± 1.7 min) and positional treatments (31.2 ± 2.2 min; mean difference, 1.7 min; 95% CI, −1.9 to 5.3; $p = 0.32$).

Epworth Sleepiness Scale scores fell from baseline with both treatments (ANOVA, $p = 0.03$; positional treatment vs baseline, $z = -1.9$; $p = 0.04$; CPAP vs baseline, $z = -2.59$; $p = 0.009$), but were not significantly different between positional treatment (median, 10; range 1 to 19) and CPAP (median, 9; range, 2 to 17; median difference, −1.5; 95% CI, −2.9 to 0.8; $p = 0.2$).

There were no significant differences in subjective sleep quality scores (positional treatment: median, 3; range, 2.0 to 4.0; CPAP, median, 3; range, 2.2 to 3.8; median difference, 0.1; 95% CI, −0.4 to 0.6; $p = 0.7$) between the two treatments (Fig 3).

Cognitive Performance

The psychometric test battery included six tests with 34 subtests for analysis. Table 4 shows differences in cognitive performance between the baseline familiarization session and after 2 weeks of positional or CPAP treatment, and between the two treatment modalities. Although there was a significant improvement in most of the tests of cognitive performance during the 2-week test period with each treatment, no significant difference could be demonstrated on any of the tests or subtests between CPAP and positional treatment.

Mood

Energy level scores on the Nottingham Health Profile were slightly better with CPAP than with positional therapy ($p = 0.04$). However, self-rated levels of energetic arousal or changes in tension or hedonic tone (UWIST Mood Adjective Checklist) were similar with the two treatments, and there were no other significant differences in the mood questionnaire scores between the two treatments (Table 5). Similarly, there were no differences between CPAP and positional treatment on Hospital Anxiety and Depression Scale scores or on the General Health Questionnaire.

Patient Preference

Four patients preferred positional treatment, seven patients preferred CPAP treatment, and two had no preference.

Discussion

This study demonstrates, contrary to our hypothesis, that positional treatment has no clinical advan-

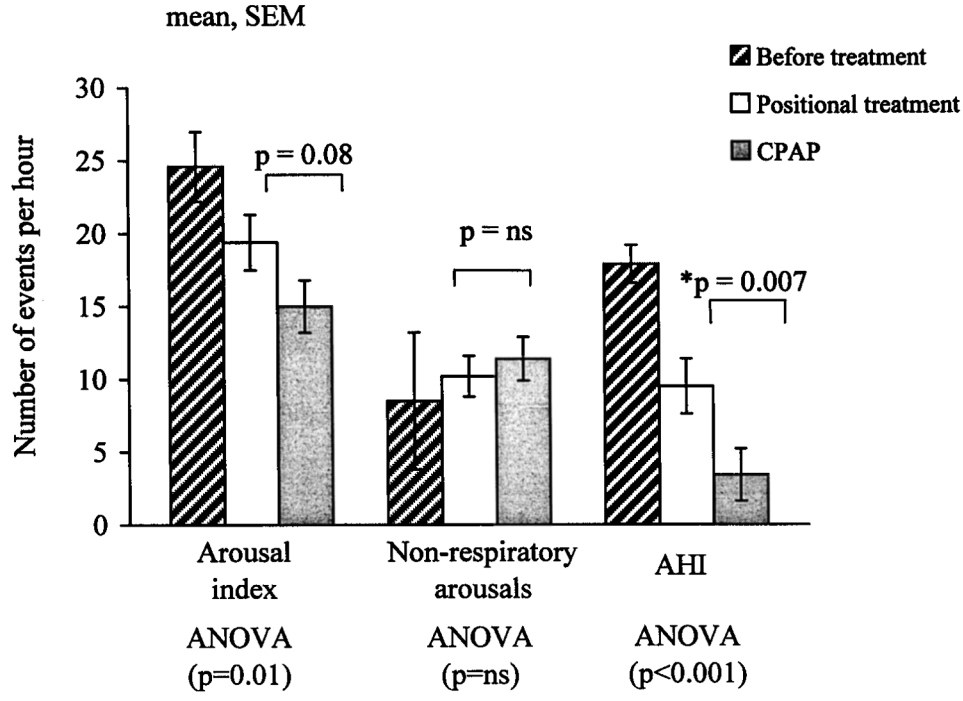

FIGURE 2. Total arousals, nonrespiratory arousals, and respiratory arousals before treatment, with positional treatment, and with CPAP. Data are expressed as mean ± SEM.

tage over CPAP in the treatment of positional OSA. Although our patients found the backpack and ball to be comfortable, there was no improvement in subjective or objective sleep quality or in the arousal index at night with this treatment as compared with CPAP, as we had hypothesized. On the other hand, the study demonstrated that although CPAP was superior to positional treatment in reducing the AHI and severity of desaturation at night in patients with positional OSA, these improvements did not translate into a functional improvement with CPAP treatment. Thus, from the findings in this study, positional treatment appears to be an effective alternative treatment to CPAP, at least in the short term, in positional OSA. This finding is very relevant to clinicians treating OSA because positional OSA is

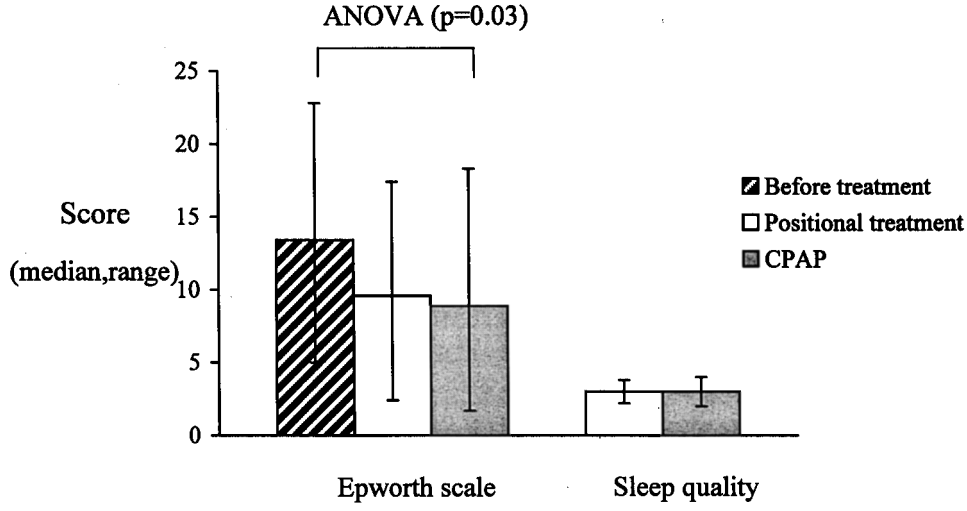

FIGURE 3. Subjective sleepiness and sleep quality before treatment, with positional treatment, and with CPAP. Data are expressed as median and range.

Table 4—*Positional Therapy vs CPAP: Psychometric Tests**

	B	P	C	B:P†	B:C†	P:C†	ANOVA‡
Wechsler Memory Scale							
Orientation		5 (0)	5 (0)			1	
Information		5.9 (0)	5.5 (1.2)			0.14	
Mental control		7.4 (1.2)	7.3 (2.1)			0.90	
Logical memory		12 (3.8)	11.4 (3.3)			0.42	
Associate learning		16.6 (4.2)	14.8 (1.0)			0.06	
Visual reproduction		10.3 (2.0)	10.6 (4.4)			0.72	
Recall							
Logical memory		9.5 (3.5)	9 (5.0)			0.52	
Visual reproduction		9.9 (2.1)	9.2 (4.5)			0.15	
Associate learning		6.3 (3.3)	6.1 (3.1)			0.54	
Digits forward		6.5 (1.5)	6.5 (2.1)			1.00	
Digits backward		5 (1.7)	4.9 (2.1)			0.86	
Memory quotient		122.8 (20.2)	122.2 (24.7)			0.83	
Purdue Pegboard							
Dominant hand	13 (3.6)	14.3 (1.7)	15.3 (1.7)	0.23	0.03	0.25	0.05
Nondominant hand	13 (1.5)	12.8 (2.3)	14.1 (2.3)	2.52	0.43	0.16	0.23
Both hands	10.8 (1.5)	11.5 (1.0)	11.1 (1.0)	0.2	0.1	1.41	0.47
Right + left + both hands	36.8 (6.7)	38.6 (4.9)	39.6 (10.7)	0.61	0.3	1.47	0.44
Assembly	34.6 (5.7)	35.4 (8.1)	35.1 (7.6)	2.05	2.11	2.05	0.96
Trial-Making Test							
Part A	28.1 (2.1)	21.9 (7.9)	20.5 (3.2)	0.02	0.004	1.17	0.04
Part B	73.8 (34.7)	57.7 (6.6)	56.5 (7.0)	0.04	0.09	2.18	0.22
Symbol Digit Modalities							
Written	52.3 (7.4)	60.9 (5.0)	59 (5.5)	0.0032	0.04	0.91	0.22
Oral	60.2 (4.0)	67 (5.0)	65.5 (3.2)	0.01	0.04	1.42	0.43
Consonant Trigram							
3-s delay	12.3 (4.4)	13.4 (1.7)	13.2 (1.5)	0.66	0.64	1.86	0.58
9-s delay	9.8 (2.5)	10.2 (2.6)	10.5 (1.5)	0.47	0.33	1.94	0.83
18-s delay	6.6 (0.6)	8.2 (1.0)	8 (3.6)	0.39	0.69	2.34	0.36
Total correct position	34.3 (7.0)	41.5 (11.6)	40.2 (10.6)	0.4	1.4	2.88	0.82
Total correct sequence	38.9 (9.0)	38.4 (11.1)	37.1 (8.0)	0.21	0.29	0.77	0.63
Concentration Endurance Test							
Total row score	482.3 (38.6)	530 (55.6)	532.3 (73.5)	0.01	0.02	2.36	0.19
Total row score − errors	447.4 (37.1)	506.9 (56)	508.6 (54.6)	0.0006	0.002	2.54	0.11
Omissions	34.4 (14.6)	22.1 (29)	22.8 (31.8)	0.1	0.39	2.42	0.4
Additions	0.6 (0.6)	1.0 (2.6)	0.9 (1.0)	1.19	1.02	2.61	0.65
First four rows	11.3 (3)	7.1 (6.4)	6.1 (5.0)	0.04	0.14	1.50	0.65
Last four rows	8.8 (1.5)	7.1 (12.5)	5.8 (6.7)	0.64	0.16	1.08	0.65
Percentage of errors	7.4 (3.1)	4.5 (6.4)	4.4 (5.4)	0.04	0.11	2.82	0.26
Fluctuation	13.6 (4.7)	9.8 (2.1)	10.1 (2.5)	0.03	0.45	2.56	0.09

*Values are mean (SD). B = baseline study; P = positional treatment; C = CPAP.
†p Values.

not uncommon. Of 184 unselected patients with OSA in one series, 60% had a positional effect that persisted in REM sleep as well as NREM sleep, such that positional treatment was felt to be a viable treatment option.[33] The prevalence of positional OSA among newly diagnosed patients with OSA at our facility was 20% (of some 300 patients who underwent overnight polysomnography during the recruitment period and the course of this study, and according to the commonly used criterion of supine AHI at least twice the nonsupine AHI[13]).

Our results demonstrate no significant difference in objective sleep quality between positional treatment and CPAP in positional OSA. One important factor that likely contributes to this finding in our study is the selection criterion of AHI < 15 in the lateral position, a criterion that was advocated *post hoc* by Cartwright and colleagues.[13] That is, patients with AHI > 15 despite positional therapy may be more likely to remain symptomatic,[15] whereas those with AHI < 15 on positional therapy are more likely to derive significant clinical benefit from positional therapy if the latter proves effective in maintaining the nonsupine posture.

CPAP has been demonstrated to improve sleep quality profoundly in patients with OSA.[3] There is little information available in the literature on the effect of positional treatment on sleep quality in

Table 5—*Positional Treatment vs CPAP: Mood Questionnaires*

	Positional Treatment*	CPAP*	Z	p Value
Nottingham Health Profile				
Emotional reactions	0 (0–2)	0 (0–11)	−0.94	0.35
Sleep	1 (0–2)	0 (0–2)	−0.91	0.36
Social isolation	0 (0–3)	0 (0–4)	0.0	1.00
Physical mobility	1 (0–2)	0 (0–2)	−1.34	0.18
Energy	1 (0–3)	0 (0–21)	−2.02	0.04
Pain	0 (0–6)	0 (0–32)	−1.60	0.11
Hospital Anxiety and Depression Scale				
Depression	3 (0–7)	2 (0–7)	−1.13	0.26
Anxiety	6 (0–14)	5 (0–10)	−0.06	0.95
UWIST Adjective Checklist				
Energetic arousal	23 (12–32)	23 (15–33)	−0.42	0.68
Hedonic tone	32 (22–32)	31 (21–32)	−0.63	0.53
Tension arousal	11 (7–26)	11 (7–18)	−0.05	0.96
General Health Questionnaire	1 (0–14)	0 (0–2)	−0.84	0.40

*Values are median (range).

patients with OSA. McEvoy and colleagues[34] reported a major reduction in the severity of OSA when subjects slept at 60° upright as compared with the horizontal position, but sleep architecture and sleep efficiency were similar in the two postures. Similarly, Braver and colleagues[35] reported an improvement in AHI with the use of the lateral posture combined with a nasal decongestant spray, as compared with control conditions, but no concomitant difference in sleep stage architecture or sleep efficiency.

We have demonstrated in patients with positional OSA that the backpack and ball used as a positional device herein significantly improved sleep apnea severity (as compared with baseline–no treatment) but that CPAP was more effective than positional treatment in alleviating upper airway obstruction during sleep. Nonrespiratory arousals from sleep were similar in frequency with the two treatments. The overall difference in arousal index between the two treatments averaged 4.4. This difference in arousal index did not appear to have any clinical significance during the 2-week course of the current study, but we cannot exclude the possibility that it could be clinically significant during long-term treatment.

Similarly, although the difference in mean overnight oxygen saturation with the two treatment modalities used in this study was not significant, CPAP was more effective than positional treatment in preventing oxygen desaturation at night. However, none of the patients in the current study was severely hypoxemic, and the clinical importance of the observed improvement in the nadir of the oxygen saturation with CPAP vs positional treatment is unclear. There was a strong correlation in the present study between the overall severity of sleep apnea during the diagnostic study night and the severity of respiratory disturbance in the lateral position, a finding that others have also reported previously.[13] This finding implies that patients with mild positional OSA are more likely to benefit from positional treatment than patients with more severe disease.

Subjective sleep quality, which has been documented to improve with CPAP treatment of OSA,[6] was not different between CPAP and positional treatment in this study. There are no previous reports in the literature of the effect of positional treatment on subjective sleep quality in patients with OSA.

Daytime somnolence was measured both subjectively (Epworth Sleepiness Scale scores) and objectively (MWT) in this study. Subjective sleepiness significantly improved with both treatments. The mean values for the Epworth Sleepiness Scale scores after CPAP treatment in this study are very similar to those reported elsewhere.[19]

Daytime alertness, as measured by the MWT, was also similar on both treatments, with mean values of MWT sleep-onset latencies only slightly below the value of 35 ± 5 min found in a group of 59 normal subjects in a recent study.[36]

We performed a series of neuropsychological tests, most of which have been shown to be sensitive to the effects of CPAP treatment, over a wide range of OSA severity.[6,11] Recently, Engelman and colleagues[37] have demonstrated cognitive function deficits in patients with mild OSA (AHI from 5 to 15) that improved with CPAP treatment. Furthermore, even when compliance was suboptimal (mean, 3.4 h per night), CPAP improved cognitive performance substantially in a group of patients with OSA.[6]

There has been no published information on the

efficacy of positional treatment in improving cognitive performance deficits in OSA. Our results demonstrate a significant improvement in cognitive performance from baseline with both CPAP and positional treatment, even in those tests for which there was a clear plateau in performance at baseline (which suggests, but does not prove, that any subsequent learning effect would be small). Nonetheless, the effect on cognitive performance of positional treatment in the current study was similar to that of CPAP, over a wide range of cognitive performance tests—positional treatment conferred no significant advantage vs CPAP, at least in the short term.

The demonstrated neuropsychological deficits in patients with OSA include changes in attention, vigilance, hand–eye coordination, immediate and delayed memory, intellectual efficiency, and higher executive functions.[6,10,11,37–40] The cause of these neuropsychological deficits has not been fully elucidated. Cognitive deficits in OSA are probably multifactorial in origin, with evidence pointing to both severity of sleep fragmentation at night[41,42] and severity of nocturnal hypoxemia.[39,40]

Even though normative data for all of the psychometric tests performed were not available, review of the data from the baseline (pretreatment) psychometric familiarization session revealed some pretreatment cognitive impairment in our patients. After a 4-h familiarization session with repeated attempts at the Purdue Pegboard, and a stable plateau in the performance scores on this sensitive test of higher executive function, 9 of our 13 patients had a performance below the normative values for this test.[24] In addition, 7 of 13 patients had scores below normative values for the Consonant Trigram Test.

Trail-Making Test B is the most widely recognized test of higher executive function in our psychometric battery. Although the mean baseline values for the group were within the normative range (73.8 ± 34.7 s)[23] and similar to those reported in patients with mild OSA on placebo,[37] there was a clear improvement in performance with either treatment during the course of the study (CPAP, 56.6 ± 7 s; positional treatment, 57.7 ± 6.6 s). Although learning effects have been documented on some cognitive performance tests (trails A and B, symbol digit modalities, concentration endurance test), we attempted to control for these effects, between treatments, by balancing the treatment order in the study.

Adverse mood changes such as irritability, impatience, or depressive manifestations are common in patients with OSA.[43] Most of our patients with positional OSA presented with increased self-rated depression and anxiety scores on the Hospital Anxiety and Depression Score, indicating psychologic distress. However, this study showed no difference in mood assessments or quality of life between CPAP and positional treatment for positional OSA. Although the Nottingham Health Profile showed higher energy levels with CPAP, other questionnaires that include similar subscales did not confirm these findings. There is no previous published information on mood changes with positional treatment for OSA. These manifestations appear to be related to the severity of OSA, contribute to the observed cognitive impairment, and improve with CPAP treatment.[6,43]

At recruitment, most patients expressed their hope that the positional treatment would prove more beneficial. However, after the study more patients preferred CPAP than positional treatment. This raises the question as to whether the positional treatment used in this study was optimal or not. Certainly, it was effective in preventing patients from sleeping in the supine position, its primary purpose. No patient volunteered a complaint of discomfort about the positional device used in this study, either during or after completion of the protocol. Others have used different strategies to treat positional sleep apnea, such as a posture alarm,[14] a tongue retaining device designed to prevent the tongue moving posteriorly when the patient sleeps in the supine position,[13] or sleeping at a 60° incline.[34] These studies demonstrated a very favorable effect of treatment on severity of OSA, regardless of which treatment or combination of treatments was used, in patients with positional OSA. However, 30% of patients using the posture alarm continued to have significant sleep apnea. The latter studies[13,14] did not report patient preference or patient satisfaction with the treatments used. In addition, despite improvement in OSA, sleep quality was not improved with the posture alarm.[14] For these reasons, we believed there would be some merit in searching for a comfortable, inexpensive, and readily accessible form of positional treatment to compare with the standard treatment for sleep apnea, rather than using the posture alarm.

It should be pointed out that many positionally trained individuals may learn to stay off the supine position after 8 weeks and can discard the device.[44] This may further enhance the comfort and efficacy of positional treatment for OSA in the longer term. However, there are as yet no reported studies that have examined the efficacy of, or patient preference for, positional treatment of OSA for a prolonged period.

The statistical power of this study to discriminate differences in outcome between the two treatments for several important variables is shown in Table 6. Although the between-treatment differences listed are significantly smaller than those previously noted

Table 6—*Power Analysis*

Variable	Estimated True Difference	Standardized Difference	Statistical Power
% Total sleep time[4]	10% TST	1.05	95%
Trails B[11]	10 s	0.91	90%
Wechsler Memory Scale[11]	10	0.96	95%
Epworth Sleepiness Scale score[19]	3.5	1.3	95%
MWT sleep latency[18]	5 min	0.84	85%

between untreated and CPAP-treated patients with OSA,[32] the current study does not have the statistical power to exclude very small treatment effects that could potentially have clinical impact during a more prolonged treatment period.

We conclude that a simple positional treatment (backpack and ball) provides similar clinical efficacy to CPAP in the treatment of patients with positional OSA, at least in the short term. Patient preference rather than any medical priority should dictate which of these treatments is used in a given patient with positional OSA. In addition, positional treatment is a very reasonable consideration for management of positional OSA in those patients who are intolerant of CPAP.

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