Can Patients with Obstructive Sleep Apnea Titrate Their Own Continuous Positive Airway Pressure?

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Manual continuous positive airway pressure (CPAP) titration in a sleep laboratory is costly and limits access for diagnostic studies. Many factors affect CPAP compliance, but education and support, rather than in-laboratory CPAP titration, appear to be pivotal. Self-adjustment of CPAP at home will provide equal or superior efficacy in the treatment of obstructive sleep apnea (OSA) as compared with in-laboratory titration. A randomized, single-blind, two-period crossover trial of CPAP treatment at the in-laboratory–determined optimal pressure versus at-home self-adjustment of CPAP (starting pressure based on prediction equation). Eighteen CPAP-naive patients (16 males, 50 ± 15 years old, apnea hypopnea index 40 ± 20) with a new diagnosis of OSA were tested. Testing was performed before and after CPAP treatment in each of two 5-week study limbs. CPAP, compliance with CPAP treatment, the Sleep Apnea Quality of Life Index, the Functional Outcomes of Sleep Questionnaire score, the Epworth sleepiness scale score, sleep architecture, sleep apnea severity, and maintenance of wakefulness tests were performed. Both modes of CPAP treatment significantly improved objective and subjective measures of OSA, but they did not differ in efficacy. Home self-titration of CPAP is as effective as in-laboratory manual titration in the management of patients with OSA.

Keywords: sleep apnea; continuous positive airway pressure; treatment; outcomes

Obstructive sleep apnea (OSA) is a common condition, affecting 4% adult males and 2% adult females (1). It is associated with significant mortality and morbidity, and untreated OSA imposes a substantial healthcare burden on the economy (2). Since its original description in 1981 (3), continuous positive airway pressure (CPAP) has become the standard treatment for OSA. It is a particularly effective treatment for patients with moderate or severe OSA (4) but also has demonstrable benefits in patients with mild OSA (5, 6). CPAP titration to discern the optimal pressure required to alleviate upper airway obstruction during sleep usually includes a simultaneous recording of sleep, respiration, and oxygen saturation (7) and is typically conducted in a sleep laboratory. This practice is expensive (two overnight sleep laboratory studies per patient with OSA—diagnostic and CPAP titration) and limits access to the sleep laboratory for diagnostic studies. Recent evidence suggests that the use of automated CPAP devices (8) and abbreviated CPAP titrations (9) can improve the efficiency with which CPAP treatment is delivered, as compared with conventional in-laboratory overnight CPAP titration. Given the high disease prevalence and limited healthcare resources, carefully evaluated attempts at greater efficiency in managing patients with OSA are needed. Approximately 15% of patients with OSA refuse CPAP treatment at the outset (10, 11), and compliance among those who accept this treatment is frequently suboptimal (12, 13). More intensive education and support have been documented to improve clinical outcomes in patients with OSA (14), and provision of an abbreviated care regimen resulted in an inferior clinical outcome (15). It is therefore essential to document both compliance with treatment and clinical outcomes in association with any intervention aimed at improving the efficiency with which treatment is delivered to patients with OSA.

An educational model in which the patient is empowered with the understanding and ability to make decisions regarding treatment has been demonstrated to be successful in other medical conditions (16). We reasoned that a similar educational approach might be successful in patients with OSA who require CPAP treatment.

Although the level of educational support, disease severity, treatment response, and other factors have been identified as contributors to CPAP compliance (17, 18), each has accounted for only a small part of the variance in compliance among individuals. The latter fact and the unpredictability of CPAP compliance among patients with OSA have led to the belief that the individual patient's outlook on CPAP treatment may be of paramount importance in determining CPAP compliance (17, 19), which may seem intuitively obvious, given the somewhat cumbersome nature of the device.

We therefore designed an intraindividual crossover trial to compare outcomes between the conventional in-laboratory method of CPAP titration and patient self-titration of CPAP for OSA.

METHODS

Design

A randomized, single-blind, two-period crossover design was employed, with a 1-week wash-in period off CPAP, two 5-week treatment limbs, and a 1-week washout between treatment limbs (Figure 1). On the "fixed limb," patients received CPAP at the pressure predetermined by manual in-laboratory titration and were not permitted to adjust the CPAP. On the "self-adjusting" limb, patients received CPAP preset at an estimated therapeutic pressure based on a prediction formula (20) and were encouraged to adjust the pressure as necessary to maximize comfort and perceived efficacy. Upon entry, patients underwent manual in-laboratory CPAP titration by an experienced registered polysomnographic technologist during full overnight polysomnography but were not informed of the optimal CPAP derived from that study. Pretreatment measurements in each limb were made to facilitate measurement of change in outcomes within each limb and to confirm a comparable degree of disease severity before treatment between limbs.

The study was approved by the Research Ethics Board at Queen's University, and written informed consent was obtained before entry.

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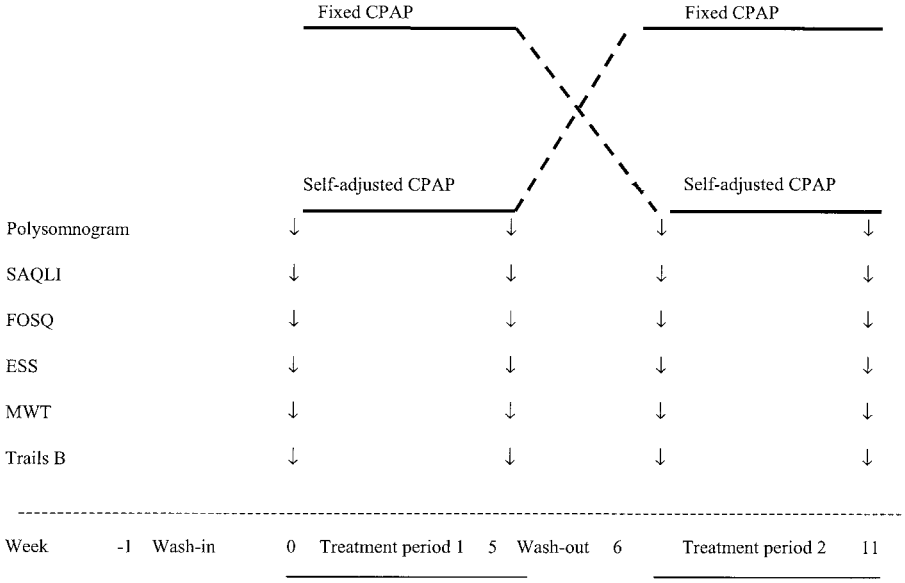

Figure 1. Schematic of study protocol. SAQLI = Sleep Apnea Quality of Life Index (21); FOSQ = Functional Outcomes of Sleep Questionnaire (22); ESS = Epworth Sleepiness Scale score (36); MWT = Maintenance of Wakefulness Test (23); Trails B = Trail-making test, part B (24).

Blinding Procedure

The pressure display on the CPAP unit was concealed throughout the fixed limb of the study with tape and adhesive that could not be removed by the patient. Sleep studies were scored blind by using a montage that excluded the CPAP signal.

Patient Education

A technologist provided 30-minutes of instruction on CPAP treatment for OSA, facial/nasal CPAP appliances, and symptoms that would suggest an incorrect CPAP setting before randomization. Patients were shown how to adjust the CPAP before the self-adjusting CPAP treatment limb.

Outcome Measures

CPAP compliance (mean hours/night), CPAP employed (cm H_2O), Apnea Hypopnea Index (AHI) (21), objective sleep architecture, Epworth Sleepiness Scale Score (22), Sleep Apnea Quality of Life Index score (23), Functional Outcomes of Sleep Questionnaire (FOSQ) score (24), Maintenance of Wakefulness Test (40-minute version) mean sleep onset latency (25), and Trail Making B time(s) (26).

Compliance

Each CPAP unit (Aria; Respironics Inc., Pittsburgh, PA) recorded runtime, time at prescribed pressure, and the CPAP setting daily. The actual CPAP output was measured independently after each limb.

Patients

Of 28 patients approached, 24 agreed to participate in the study. Of the 24 recruited patients, 6 did not complete the study; they were 52 ± 12 years old (mean ± SD, range 39 to 68 years), and had a mean body mass index of 37 ± 9 kg/m^2 (range, 29–53 kg/m^2), an AHI on diagnostic sleep study of 65 ± 31 (range, 28–93), a pretreatment Epworth score of 8.5 ± 3.6 (range, 4–14), and a CPAP requirement (manual titration) of 11 ± 2.2 cm H_2O (range, 8–14 cm H_2O).

Data Analysis

The treatment effect (adjusting limb-fixed limb) estimates for each outcome were calculated using the popular method described by Fleiss (27) and others for two-period crossover studies (*see* the online supplement). This method allows for a possible period effect and is appropriate when there is an imbalance in the number of patients randomized to each sequence. Point estimates of the treatment effects are presented with the corresponding 95% confidence intervals.

RESULTS

The flow of patients through the study is illustrated in Figure 2. Eighteen patients (16 males and 2 females) completed the study. These patients were 50 ± 15 years old (mean ± SD; range, 28–78 years), had a body mass index of 36 ± 9 (range, 28–70 kg/m^2), and an AHI of 40 ± 20 (range, 9–78, using thermistor as airflow signal) (21). Six patients did not complete the study. One patient withdrew for medical reasons (diagnosed with metastatic prostate cancer during the study). One patient was withdrawn when it was discovered that although his diagnostic sleep study and baseline sleep study on limb 1 both showed an AHI of more

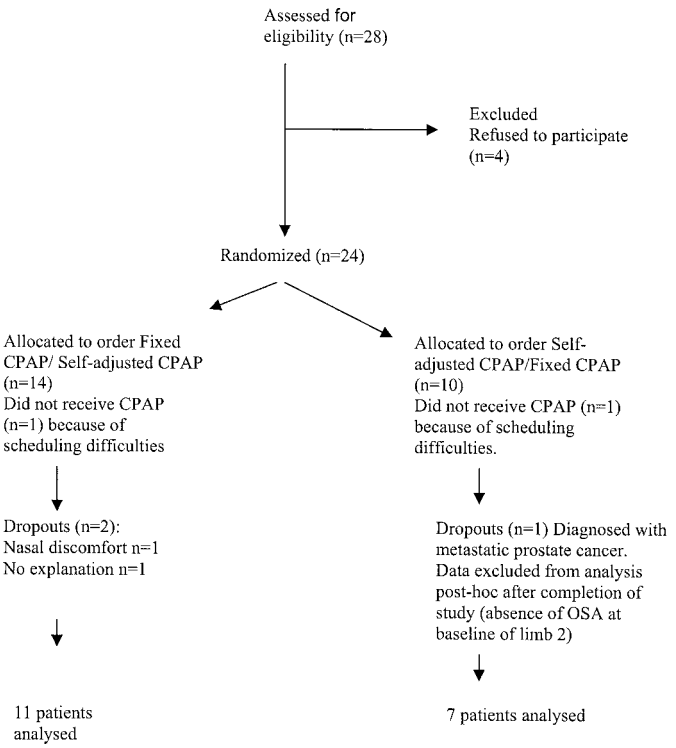

Figure 2. Flow diagram of patients through the study.

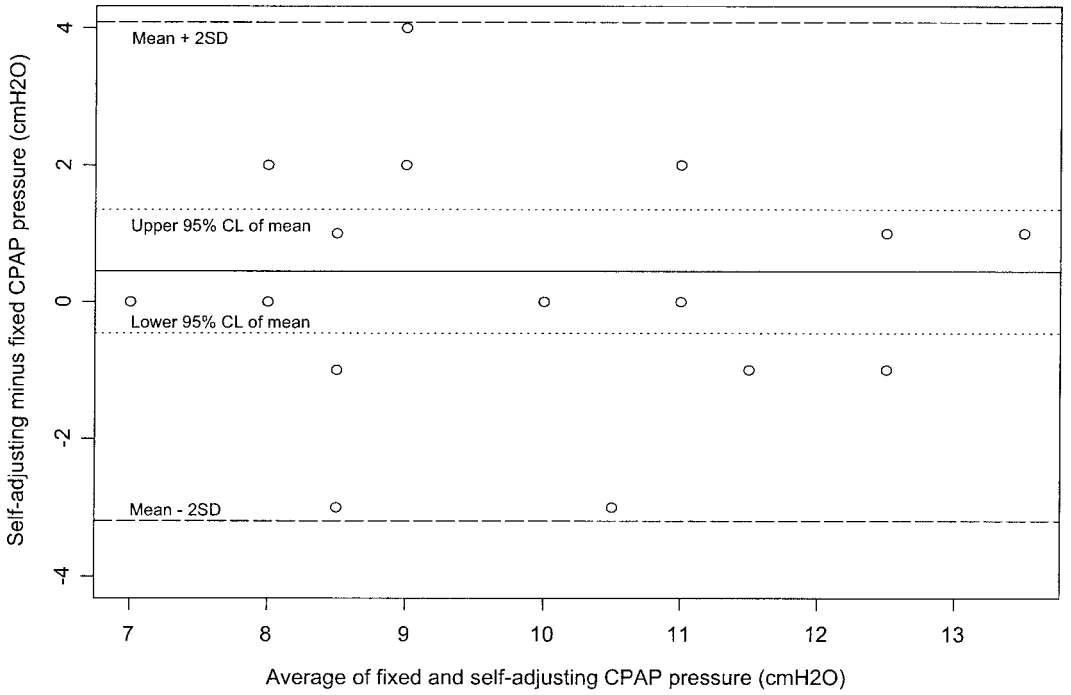

Figure 3. Bland-Altman plot illustrating the level of agreement in optimal CPAP pressure as determined by in-laboratory manual titration and by patient self-titration. CL = confidence limits.

than 20; his baseline study at the start of limb 2 (off CPAP) showed no evidence of OSA. As per the study protocol, the patient had returned his CPAP unit to the investigators for the duration of the washout period. The disappearance of OSA in his case remains unexplained. However, the patient's spouse had a CPAP unit at home, and it is possible that the patient used this unit during the washout period. One patient withdrew because of nasal discomfort from CPAP treatment. Two patients withdrew because of scheduling conflicts between work and research testing. One patient chose not to provide an explanation for withdrawal from the study.

CPAP

The CPAP determined by patients to be optimal during the self-adjusting limb of the study was 10.1 ± 2.0 cm H_2O (mean ± SD, range, 7 to 14 cm H_2O) compared with 9.7 ± 2 cm H_2O (range, 7 to 13 cm H_2O) derived by manual overnight CPAP titration in the sleep laboratory. The estimated within patient difference between these values was 0.3 (95% confidence interval, −0.6 to 1.3 cm H_2O, $p = 0.45$). The agreement between the optimal CPAP chosen by the patient and that derived by in-laboratory titration ($r = 0.62$, $p = 0.006$) is depicted in Figure 3. The mean prediction equation-derived optimal CPAP (8.5 ± 0.4 cm H_2O, range, 6 to 13 cm H_2O), which was used as the starting pressure for the self-adjusting limb of the study, was significantly different from the mean self-determined optimal CPAP at the end of that treatment limb (mean difference 1.6 ± 1.2 cm H_2O; 95% confidence interval, 1 to 2.2 cm H_2O; $p < 0.0001$), but the two pressures were significantly correlated ($r = 0.82$, $p < 0.001$). Similarly, the mean prediction equation-derived CPAP differed significantly from the mean in-laboratory determined CPAP (mean difference, 1.2 ± 1.8 cm H_2O, $p = 0.012$), but the two were significantly correlated ($r = 0.63$, $p = 0.005$). On the self-adjusting CPAP limb, the average number of CPAP changes made by patients was 5.7 (SEM 1.0; range, 1 to 16). No adjustment of the CPAP occurred during the fixed limb in any patient.

CPAP Compliance

The average duration of CPAP use per night was not significantly different between the fixed (6.4 ± 1.2 hours) and self-adjusting (6.7 ± 1.7 hours) limbs of the study with a mean within patient difference of 0.3 (−0.6 to 1.2 hours, $p = 0.48$). CPAP was not used during an average of 1.9 ± 2.4 days on the fixed limb and 2.3 ± 3.4 days on the self-adjusting limb for a difference of 0.45 (−2.1 to 3.0 days, $p = 0.71$). Analysis of the hours of CPAP use during only those nights when the device was actually applied reveals a mean CPAP use per night on the fixed study limb of 6.7 ± 1.1 hours and on the self-adjusting limb of 7.3 ± 2.2 hours, resulting in a difference of 0.6 (−0.5 to 1.6 hours/night, $p = 0.28$). Patients used CPAP for more than 4 hours on 87 ± 14% of the fixed limb nights and 86 ± 10% of the self-adjusting limb nights.

Subjective Outcome Measures

There were significant improvements in most of the subjective outcome measures during both treatment limbs (Table 1). In particular, substantial improvements in subjective sleepiness and disease-specific quality of life were noted. However, there was no difference in the size of the improvement observed between the two treatment limbs for any of these variables.

Objective Outcome Measures

Table 2 demonstrates the overnight polysomnographic data for the 4 overnight polysomnograms performed on each of the 18 patients. There was no statistically significant difference in the change in any of the sleep variables between the two treatment limbs. The sleep stage architecture did not change significantly with either CPAP treatment limb. As expected, there were profound improvements in minimum oxygen saturation and AHI with both CPAP treatment limbs, but no significant difference between treatment limbs.

Daytime Alertness and Trail-making Performance

CPAP treatment on both limbs of the study was accompanied by a significant improvement in objective daytime alertness, as

TABLE 1. MEAN VALUES FOR OUTCOME VARIABLES ON EACH LIMB

Variable	Prefixed Mean ± SD	Postfixed Mean ± SD	Δ Fixed Mean (*95% CI*) p Value	Preadjusted Mean ± SD	Postadjust Mean ± SD	Δ Adjust Mean (*95% CI*) p Value	Adjusted Δ − Fixed Δ Mean (*95% CI*) p Value
Epworth	10.7 ± 3.8	7.6 ± 3.8	−3.1 (−5.0, −1.1) 0.004	11.1 ± 3.9	6.9 ± 4.0	−4.2 (−6.6, −1.8) 0.002	−1.4 (−3.1, 0.4) 0.11
FOSQ	83.9 ± 15.5	97.7 ± 17.5	13.8 (5.4, 22.3) 0.003	83.4 ± 20.7	95.2 ± 19.8	11.8 (1.8, 21.8) 0.024	−3.0 (−14.8, 8.9) 0.60
SAQLI	4.2 ± 1.0	5.0 ± 1.2	0.8 (0.3, 1.2) 0.002	4.3 ± 1.1	5.4 ± 0.9	1.1 (0.7, 1.6) 0.001	0.5 (0, 0.9) 0.048
MWT, min	16.3 ± 8.3	23.3 ± 10	6.9 (2.3, 11.5) 0.005	15.3 ± 9.0	25.3 ± 9.3	10.0 (5.6, 14.5) < 0.0001	3.1 (−2.2, 8.4) 0.24
Trail-making B, sec	65.7 ± 25	64.4 ± 28	−1.3 (−10.7, 8.1) 0.77	72.6 ± 31	67.3 ± 34	−5.3 (−11.1, 0.6) 0.08	−0.04 (−0.11, 0.04) 0.33

Definition of abbreviations: CI = confidence interval; ESS = Epworth Sleepiness Scale score; FOSQ = Functional Outcomes of Sleep Questionnaire; MWT = Maintenance of Wakefulness Test; SAQLI = Sleep Apnea Quality of Life Index; Trails B = Trail-making Test, Part B.

Mean ± SD values for variables at the start and end of the fixed and self-adjusting CPAP treatment limbs. Δ Fixed refers to the change in the variable between the start and end of the fixed limb. Δ Adjust refers to the change in the variable between the start and end of the self-adjusted limb. Adjusted Δ − Fixed Δ refers to differences between Δ Fixed and Δ Adjust for each variable.

measured by the Maintenance of Wakefulness Test (Table 1). However, there was no significant difference between treatment limbs in the mean improvement in this variable. The Trails B test score did not change significantly with treatment during either of the two study limbs, and there was no difference in the mean change in trail-making performance between the two treatment limbs.

DISCUSSION

This study demonstrates that patients with OSA are capable of effective self-titration of CPAP treatment at home. The optimal CPAPs, defined by self-titration and by manual in-laboratory titration, were similar. Improvements in both subjective and objective outcome measures were fairly consistent and were similar in magnitude between self-titration at home and manual in-laboratory CPAP titration during overnight polysomnography. There was no clinically significant difference in any measured outcome between the two CPAP treatment modalities, and compliance with CPAP treatment was highly satisfactory on both study limbs.

The observed improvements in subjective outcome measures during both treatment limbs were of similar magnitude to those previously documented with CPAP treatment in moderate and severe OSA. Thus, the change in subjective sleepiness as measured by the Epworth Sleepiness Scale score during each treatment limb in this study was similar to that observed in clinical practice (18) and in placebo-controlled trials of CPAP treatment for OSA (5, 28, 29). The Sleep Apnea Quality of Life Index is a useful measure of disease-specific quality of life and, in particular, has the ability to incorporate negative effects of CPAP into the overall pre-to post-CPAP response (23). The size of the mean improvement in the Sleep Apnea Quality of Life Index score with CPAP treatment in both limbs of this study (0.8 units, fixed CPAP limb; 1.1 units, self-adjusted CPAP limb) represents a small but clinically significant improvement (30). There was a slightly greater improvement in Sleep Apnea Quality of Life Index score with self-adjusted CPAP than with in-laboratory

TABLE 2. CHANGES IN SLEEP VARIABLES

	Prefixed	Postfixed	Preadjust	Postadjust	Adjusted Δ − Fixed Δ Mean (*95% CI*)
Total recording time, min	453 ± 34	465 ± 26	448 ± 35	460 ± 31	−1 (−26, 24)
Total sleep time, min	367 ± 61	385 ± 53	360 ± 73	386 ± 53	10 (−33, 52)
Sleep efficiency, %	81 ± 13	83 ± 10	80 ± 14	84 ± 9	3 (−4, 10)
Sleep onset latency, min	7.2 ± 7.4	7.4 ± 5.7	7.9 ± 6.5	7.9 ± 7.4	−0.9 (−7.8, 5.9)
REM latency, min	123 ± 73	118 ± 47	112 ± 35	106 ± 60	−13 (−31, 44)
Stage 1, %	12 ± 8	11 ± 6	11 ± 5.7	9 ± 5.7	−1.4 (−6.6, 3.9)
Stage 2, %	60 ± 11	61 ± 10	67 ± 13	62 ± 9	−4 (−11, 4)
Stages 3/4, %	4.0 ± 7.4	6.2 ± 8.6	3.1 ± 5.6	4.9 ± 7.5	−7.4 (−5.1, 3.6)
Stage REM, %	24 ± 7	22 ± 6	19 ± 8	24 ± 7	6 (−1, 13)
Time supine, % TST	41 ± 30	47 ± 28	39 ± 28	50 ± 29	0 (−30, 30)
Time right lateral, % TST	20 ± 22	20 ± 24	31 ± 26	17 ± 15	−10 (−30, 11)
Time left lateral, % TST	39 ± 32	33 ± 27	32 ± 25	32 ± 31	−10 (−30, 11)
Apnea–Hypopnea index*	43 ± 25	6 ± 6	46 ± 20	5 ± 5	−4 (−14, 7)
Minimum Sa_{O_2}*	75 ± 13	91 ± 4	76 ± 12	91 ± 6	1 (−7, 8)
RDI*	36 ± 19	6 ± 5	40 ± 17	5 ± 4	−4 (−13, 6)

Definition of abbreviations: CI = confidence interval; RDI = respiratory disturbance index; REM = rapid eye movement sleep; Sa_{O_2} = oxygen saturation; TST = total sleep time.

Mean ± SD values for objective sleep variables at the start and end of the fixed and self-adjusted CPAP treatment limbs. Adjusted Δ − Fixed Δ estimates the difference between the changes in the fixed and adjusting limbs. None of these differences were significant at $\alpha = 0.05$.

* $p < 0.01$ for the change from pre to post for both fixed and self adjusting limbs.

titrated CPAP, but the difference between CPAP treatment modes was not clinically important. The CPAP treatment-associated change in the FOSQ (24) score observed during each limb of the current study was significant and consistent with the findings reported in other trials of CPAP treatment for OSA (28, 29). Thus, patients with mild OSA had fewer symptoms (a higher baseline pretreatment FOSQ score, mean 101 ± SD) (18) and a smaller response to CPAP treatment (mean post-CPAP treatment FOSQ score, 106 ± 18) than that observed during either CPAP treatment limb in this study (29), whereas patients with more severe OSA tended to have a more symptoms (a lower pre-CPAP FOSQ score, mean ± SEM, 84.5 ± 4.63) and a larger pre- to post-CPAP treatment response in FOSQ score (post-CPAP score, 109.4 ± 2.6) (28) than that observed in this study. The magnitude of the improvement in subjective outcome measures with either CPAP treatment limb in this study, therefore, was at least as great as that which might have been predicted, based on published literature in similar patient groups with OSA, suggesting that both treatment limbs provided effective symptomatic treatment to this patient group. However, it is important to understand that this patient group was not selected on the basis of symptom severity and included several patients who had few daytime symptoms; if subjective outcomes had been the most important outcome measures, then the presence of significant daytime symptoms related to OSA would have been an essential inclusion criterion.

Most objective outcome measures also improved significantly with either method of CPAP treatment. The Maintenance of Wakefulness Test (31, 32) was employed as a measure of daytime alertness in this study, as evidence suggests that it is a more valid measure of sleepiness/alertness in OSA than the Multiple Sleep Latency Test (31–33). Using the one-epoch criterion for sleep onset and the 40-minute version of the test, patients were clearly objectively somnolent pretreatment on both limbs of the study, but voluntary alertness improved into the normal range (25) with treatment, as expected. Trail-making B performance, a test of higher executive function, has demonstrated sensitivity in some previous studies to the effects of sleep apnea (26) and to CPAP treatment versus placebo (34) but did not show any significant change with CPAP treatment in this study. However, the finding of improved trail-making performance with treatment of OSA has not been a consistent one (35), with several studies demonstrating no change in Trail-making performance with treatment of OSA, despite unequivocal treatment-related improvements in several other domains. Therefore, it would be unreasonable to extrapolate from the negative trail-making performance response to CPAP treatment in this study to other domains of cognitive performance that were not measured in this study.

The disparity between the aforementioned improvements in subjective and objective outcome measures and the lack of change in any objective measures of sleep architecture with CPAP treatment in this study is surprising. There was a marked improvement in sleep continuity associated with reduction in the AHI and also a marked improvement in the nadir of the oxygen saturation during sleep with treatment. One might have expected a coincident increase in slow wave sleep and rapid eye movement sleep with this magnitude of improvement in sleep-disordered breathing (21). The lack of improvement in objectively measured sleep stage architecture with CPAP treatment in this study is not easy to explain. The noise to signal ratio of in-laboratory recordings could potentially have interfered with the ability to detect changes in sleep architecture, but the recording equipment and environment used in this study were standard for clinical sleep studies and were similar to those employed in other studies that have demonstrated improvements in objective sleep architecture in patients with OSA with CPAP treatment. Others have described improvements in symptoms and daytime performance in patients with OSA treated with CPAP, in the absence of any objective improvement in sleep architecture (6), but this finding is not typical. This study was not specifically designed to examine differences in sleep stage architecture with treatment (sleep stage data were required to calculate the pretreatment and post-treatment AHIs and were therefore reported). McArdle and Douglas, in a placebo-controlled crossover study of 22 patients, designed to analyze the sleep architectural changes associated with treatment of OSA, demonstrated a doubling of slow wave sleep, halving of stage 1 sleep, and a nonsignificant increase in REM sleep with CPAP treatment (36). *Post hoc* statistical power analysis reveals that this study had 90% power ($\alpha = 0.05$) to detect a change of 5% in the proportion of total sleep time spent in slow wave sleep pre- to post-CPAP on either treatment limb or between the two post-CPAP nights in this study (an amount similar to the mean difference observed by McArdle and Douglas between CPAP and placebo). However, there was more variability in the percentage of time spent in stage 1 sleep pre- to post-CPAP treatment in this study. The *post hoc* power estimate suggests that a difference of 8% in the proportion of total sleep time spent in stage 1 sleep comparing pre- to post-CPAP and comparing the two post-CPAP nights would be required to provide 90% statistical power ($\alpha = 0.05$) in this study; this is a larger change than that observed by McArdle and Douglas. Hence, an inadequate sample size may underlie the apparent lack of improvement in some aspects of sleep architecture in this study.

The use of a crossover design increased statistical power to detect differences in the primary outcome variables of this study, but the major motivation for this design was to eliminate the effect of interindividual differences on study outcomes, particularly CPAP compliance. CPAP compliance among patients with OSA is a complex issue. Although severity of disease and improvement in daytime somnolence with treatment have been demonstrated to be important factors in determining CPAP compliance (37), interindividual attitudes and preferences appear to be even more predominant in this regard (19). Thus, despite a wealth of literature on factors associated with either satisfactory or poor CPAP compliance among patients with OSA, it remains very difficult to predict CPAP compliance in a given patient with OSA. This fact has fuelled enthusiasm for a purely pragmatic approach to CPAP treatment in OSA: A short individual clinical trial of CPAP has been advocated as the best way of determining the likelihood of acceptable CPAP compliance in a given individual (38). Whereas an intraindividual crossover design eliminated concerns about dominant interindividual differences in attitude to and acceptance of CPAP treatment, it also opened up other potential sources of bias in this study. In particular, acclimatization to CPAP treatment in the first study limb could have biased toward a greater treatment effect in the second study limb, and a carryover effect between treatment limbs could have reduced the treatment effect on the second treatment limb. We attempted to address these potential sources of bias by (*1*) designing a washout period between treatment limbs that would eliminate any likelihood of a carryover effect, (*2*) randomizing the treatment order between patients, (*3*) including possible order effects in the data analysis when comparing outcomes between treatment limbs, (*4*) making baseline pretreatment measurements at the start of each treatment limb to document any change in pretreatment disease severity between study limbs, and (*5*) comparing the pretreatment to post-treatment change in relevant variables rather than simply comparing the post-treatment values for each variable. There are, of course, other cost-efficient methods of introducing CPAP treatment to patients with OSA. For

patients with more severe OSA, split-night studies (where the overnight study is partitioned into an initial diagnostic part and then, after the diagnosis of OSA has been objectively confirmed, CPAP titration is performed) have proven feasible (9, 39). CPAP titration can also be undertaken by automated CPAP treatment devices, and those devices that base the change in CPAP on changing airflow contour may provide a satisfactory estimation of the therapeutic CPAP in some patients (40–42) Automated CPAP titration devices do not appear to have any advantage over conventional fixed CPAP in the routine treatment of OSA (43–45) and may not compensate appropriately for changes in nasal resistance (8). A strategy that empowers the patient with OSA with the freedom to alter CPAP appropriately in response to altered upper airway physiology is inexpensive and may prove advantageous in the latter situation and in the long-term management of the patient. To date, detailed objective measures of daytime performance in patients with OSA after automated CPAP titration have not been reported, and there is no available information, of course, on the relative merits of self-titration of CPAP versus automated CPAP titration.

In considering the findings of this study, it is important to appreciate that the protocol required each patient to undergo a manual in-laboratory CPAP titration after randomization. It is likely that patients derived some benefit from the presence of a sleep technologist during this initial exposure to CPAP. Indeed, there is very clear evidence that even minor initial efforts at encouragement and education of the patient with sleep apnea may influence subsequent CPAP compliance (46, 47). Patients in this study also received 30 minutes of education about sleep apnea and CPAP treatment and a phone call from a research assistant on each study limb. The latter level of patient education and support, which is equivalent to the routine allotment of time for education of each patient with a new diagnosis of OSA in our clinical practice, may nonetheless account for the superior CPAP compliance observed during both limbs of this study as compared with other similar controlled trials of CPAP treatment in OSA (4, 6, 48, 49). Hence, it is important not to misconstrue the findings of this study as obviating the need for education and support of the patient with OSA undergoing CPAP treatment. Rather, the study demonstrates that in combination with a modest amount of educational support (a time commitment from a clinical assistant that would, hopefully, be feasible in routine clinical practice), the patient with OSA is just as capable of performing an effective CPAP self-titration as a technologist during overnight polysomnography. Although the self-determined optimal CPAP sometimes varied widely from the prediction equation–derived CPAP and the two mean pressures were significantly different in this study, the prediction equation may have provided a useful starting point for patient self-titration because it tended on average to slightly underestimate the optimal CPAP. One could speculate that there may be some advantage to starting with a slightly lower than optimal CPAP rather than too high of a CPAP in that the patient is protected from the discomfort associated with higher CPAPs, and this may have contributed to the high degree of patient compliance with CPAP during the self-adjusted CPAP treatment period in this study.

The AHI measured in this study was based on a thermistor measure of airflow. This measure, although it yields a useful threshold (AHI of 5) between minimum clinically relevant disease and normal (21), is less sensitive in the detection of airflow limitation than the nasal cannula pressure transducer (50). For that reason, the thermistor may underestimate disease severity; it is important to bear this fact in mind when extrapolating the findings of this study to patients whose sleep apnea diagnosis is based on a nasal cannula pressure transducer airflow signal and who may have milder OSA than the patients in this study. The study protocol empowered each patient with the knowledge and capability of directing his or her own CPAP treatment during the self-adjusted CPAP treatment limb. This strategy has not previously been employed in CPAP treatment of OSA, but systematic evaluations of similar management approaches for other medical disorders have generated very positive findings and have been demonstrated to facilitate cost-effective treatment of those conditions (16, 51–53). Because CPAP compliance is already known to be sensitive to patient education, it would be unethical and clinically unhelpful to have conducted a placebo-controlled study of this educational intervention. The very satisfactory CPAP compliance rate with the conventional approach to CPAP prescription in this study provided a suitably high treatment standard against which to evaluate self-directed CPAP therapy and provided information that will, hopefully, be useful in clinical practice. The study was adequately powered to detect clinically meaningful differences between the two CPAP treatment strategies. One shortcoming of this study is the relatively short duration of the treatment protocol; although the results are promising, they cannot be extrapolated to long-term clinical outcomes. A randomized parallel group study with a longer treatment duration, and with both clinical and health–economic outcomes, would be required to assess whether such a treatment strategy can provide significant economic advantages without compromise of the standard of care for patients with OSA.

In summary, this study demonstrates that self-titration of CPAP in patients with OSA is as efficacious as manual titration in a sleep laboratory, with similar subjective and objective outcomes, and CPAP compliance. Clearly, for this strategy to be successful, the patient must understand when and how to change the CPAP. Although the patient population studied did include a wide age range, this strategy would not be feasible for intellectually disadvantaged patients and those with physical handicaps that would severely limit vision and/or manual dexterity. Nonetheless, the findings from this study imply that routine overnight polysomnography is unnecessary for the purpose of CPAP titration in many patients with OSA, provided that the patient is given some basic education and support. Resources currently allocated to manual in-laboratory CPAP titration might be better spent on specific attention to patient education and support rather than pressure titration. A treatment algorithm that focuses on such ambulatory patient education and support rather than in-laboratory CPAP titration may realize significant efficiencies in the management of OSA without loss of treatment efficacy.

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