Efficacy of Automatic Continuous Positive Airway Pressure Therapy That Uses an Estimated Required Pressure in the Treatment of the Obstructive Sleep Apnea Syndrome

Frédéric Sériès, MD, and Isabelle Marc, MD

Background: Continuous positive airway pressure (CPAP) is effective therapy for the obstructive sleep apnea syndrome (OSAS). Automatic CPAP devices continuously adjust the positive pressure to the required levels.

Objective: To determine the efficacy of an automatic CPAP machine used with an estimated reference pressure value.

Design: A before-and-after, single-blind trial in which patients were randomly allocated to one of three modes of CPAP administration.

Setting: Referral-based sleep center in a public health care institution.

Patients: 36 outpatients with OSAS.

Intervention: Continuous positive airway pressure was given at a conventional fixed pressure (group 1), automatic CPAP was given at a measured reference pressure (group 2), and automatic CPAP was given at an estimated reference pressure (group 3). In group 1, the effective pressure was determined during a titration sleep study. In groups 2 and 3, the pressure interval was allowed to vary from 4 cm H_2O below reference pressure to 3 cm H_2O above reference pressure. In group 3, the estimated value of the reference pressure was determined according to individual anthropometric characteristics.

Measurements: Sleep studies were performed and measurements of diurnal sleepiness were obtained at each visit.

Results: Sleep and breathing disorders and hypersomnolence were alleviated similarly in the three groups. The apnea + hypopnea index remained abnormal in one patient in group 3 for whom the reference pressure had been underestimated. A strong negative correlation was found between the percentage of time spent below reference pressure during CPAP and the difference between the effective and estimated pressures.

Conclusion: Automatic CPAP can be used with an estimated reference pressure without doing a titration sleep study. The positive pressure trend can be used to determine whether treatment failure is caused by an inadequate pressure setting and to determine the amount of pressure to apply.

The obstructive sleep apnea syndrome (OSAS) is highly prevalent; it is found in 4% of middle-aged men and 2% of middle-aged women (1). The increase in the rate of illness and death associated with this disease emphasizes the need for effective treatment (2–5). Approaches to therapy include weight loss (6), upper airway surgery (7), oral appliances (8), and nasal continuous positive airway pressure (CPAP) (9). Therapy with CPAP decreases the rate of illness and death associated with sleep apnea (5, 10–11). Before CPAP therapy is started at home, a polysomnographic study must be done to determine the pressure needed to resolve apnea, hypopnea, snoring, and respiratory-related arousals in all sleep stages and body positions. The CPAP device is then set to this level. Effective pressure can also be estimated by using a regression model that takes into account anthropometric characteristics, neck circumference, and the frequency of nocturnal breathing abnormalities (12). However, the regression model often underestimates the positive pressure requirements. A recent report (13) suggested that effective pressure can be reliably estimated during unattended sleep studies by using automatic adjustments of the positive pressure made on the basis of a record of respiratory variables.

Because the optimal CPAP comprises several factors, the effective pressure can change within one night and from one night to another depending on changes in body position (14, 15), sleep stage (14), neck and mandibular position (16, 17), nasal patency, upper airway edema (18), and other factors. This variability has led to the development of automatic CPAP devices that continuously adapt the positive pressure in response to varying needs. We compared the efficacy of conventional CPAP therapy with that of automatic CPAP therapy administered by an automatic CPAP machine (Morphée Plus, Pierre Médical, Verrières-Le-Buisson, France) for OSAS (19). With this machine, the user must establish a reference pressure that usually corresponds to the effective pressure determined by a conventional titration sleep study. However, the ef-

Ann Intern Med. 1997;127:588-595.

From Hôpital Laval, Sainte-Foy, Québec, Canada. For current author addresses, see end of text.

See related article on pp 581-587.

fective pressure can be estimated by the user's characteristics; we hypothesized that the ability of the automatic CPAP machine to automatically adjust the positive pressure should compensate for the uncertainty in effective pressure determination obtained by the regression model. This procedure may provide effective treatment of OSAS without doing a CPAP titration sleep study.

We compared the efficacy of automatic CPAP given at an estimated reference pressure, automatic CPAP given at a measured reference pressure, and conventional CPAP therapy.

Methods

Patients

The study was conducted between 30 May 1995 and 16 May 1996. Thirty-nine patients were asked to participate, but 3 refused to participate because of their work schedules. Therefore, 36 patients (age range, 36 to 65 years; mean body mass index ± SD, 36.4 ± 7.6 kg/m^2) with previously untreated OSAS were included.

All patients were seen at the sleep clinic at Hôpital Laval. A diagnosis was made on the basis of clinical features and was confirmed by a polysomnographic study (mean apnea + hypopnea index [events per hour] ± SD, 43.6 ± 19.8). After this diagnostic study, all patients who chose to be treated with nasal CPAP and had no exclusion criteria were asked to participate in the study. Patients were excluded if they had life-threatening forms of OSAS (such as severe hypersomnolence) that required immediate treatment, OSAS associated with nonobstructive sleep-related breathing disorders (such as periodic breathing and hypoventilation) or with nonrespiratory sleep disorders (such as narcolepsy and periodic leg movements), or an estimated pressure above 15 cm H_2O.

Treatment with Continuous Positive Airway Pressure

A titration sleep study was conducted within 1 week after the diagnostic sleep study to determine the effective pressure in all patients. Patients were then randomly assigned to one of three treatment groups in a single-blind fashion.

The Morphée Plus automatic CPAP device will soon be distributed in North America by Nellcor Puritan-Bennett (Eden Prairie, Minneapolis, Minnesota) under the name of Cloudnine. The device has not yet been approved by the U.S. Food and Drug Administration. Because the Morphée Plus device can be set to a constant or an automatic CPAP mode, we used this machine to treat the three groups. In the automatic CPAP mode, the positive pressure is allowed to change within a determined range around the reference pressure. The reference pressure and the upper and lower pressure thresholds are chosen separately by the physician. Group 1 was treated with conventional CPAP; that is, a constant pressure was set at the effective pressure. For groups 2 and 3, the machine was used in the automatic CPAP mode; the reference pressure was set at the measured effective pressure for group 2 and at the estimated effective pressure for group 3. The estimated effective pressure was calculated by using the following formula (12):

$$\text{Estimated pressure} = -5.12 + 0.13 \times \text{body mass index} + 0.16 \times \text{neck circumference} + 0.04 \times \text{apnea} + \text{hypopnea index}$$

We set the range of pressure at 3 cm H_2O above the reference pressure to 4 cm H_2O below the reference pressure in accordance with our previous experience with this machine (19) and the level of underestimation reported with the estimated pressure formula (12).

Patients were blinded to treatment assignment. For each block of three patients, the treatment regimen was determined by using a randomization table. To compare patients with similar severity of disease, patients were paired for the estimated pressure value. In each block of three patients, the estimated pressure could not differ by more than 1 cm H_2O from the value of the first patient included in that block. If a candidate did not meet this criterion, he or she was assigned to the first randomized position in the next block of three patients.

Characteristics of Automatic Continuous Positive Airway Pressure

In the automatic CPAP mode, the positive pressure is maintained as long as ventilation remains stable; however, any respiratory disorder results in a progressive increase in the pressure. If a breathing disturbance has not occurred for more than 4 minutes, the positive pressure decreases again. Changes in pressure are regulated by a constant feedback analysis of the patient's ventilation by the nasal CPAP device. The breath-by-breath difference between maximal inspiratory and expiratory flow is estimated by the changes in compressor speed required to maintain a constant positive pressure throughout the respiratory cycle. Apneic and hypopneic events are associated with a decrease in the difference between the inspiratory and expiratory flow; this leads to an increase in the pressure until the flow regimen of the compressor has become stable or the fixed upper limit of pressure has been reached.

Outcome Measures

Sleep and breathing characteristics were measured during polysomnographic studies that consisted of continuous electroencephalography (C_4/A_1, C_3/A_2, O_2/A_1, and O_1/A_2), submental electromyography, electrooculography, and electrocardiography and measurement of 1) nasobuccal airflow by thermistors, 2) thoracoabdominal movements by inductive plethysmography (Respitrace, Ambulatory Monitoring, Inc., Arsdley, New York), 3) arterial oxyhemoglobin saturation (SaO_2) level with an ear oximeter (504 Pulse oximeter, Criticare Systems, Inc., Waukesha, Wisconsin), and 4) breathing noises by two microphones placed at the bedside. All variables were recorded on a computer (Sandman, Melleville Diagnostics, Ottawa, Ontario, Canada). Polysomnographic recordings were manually interpreted in 30-second periods according to established criteria (20). Arousals were scored according to the definition established by the American Sleep Disorders Association (21). Apnea was defined as the cessation of nasal–oral airflow for at least 10 seconds. Hypopnea was defined as a decrease of more than 50% in total thoracoabdominal movements for at least 10 seconds and was associated with a 2% decrease in SaO_2 level or an arousal.

Diurnal somnolence was assessed by using the Epworth Sleepiness Scale (22) and maintenance-of-wakefulness tests (23). For the latter, patients were seated in a comfortable armchair every 2 hours and asked to remain awake as long as possible during four consecutive 40-minute periods in darkness. Each maintenance-of-wakefulness test was stopped after 10 minutes of sleep or after 40 minutes if no sleep was recorded; sleep onset was defined as three continuous periods of stage I sleep, any period of stage II to IV or rapid eye movement sleep. Sleep latency was defined as the mean value of the four tests.

The mean positive pressure, the proportion of time spent at different pressures, and the time at which positive pressure was applied were automatically recorded each night by the CPAP device for 3 weeks. Changes in positive pressure were analyzed during the control sleep study by continuous recording of mask pressure. Compliance with CPAP therapy was assessed by measuring the amount of CPAP used during the study period and was quantified by measuring the percentage of time during which the machine was turned on and positive pressure was applied (effective pressure–time index) and the number of nights during which positive pressure was applied for more than 4 hours and more than 7 hours (24).

After 3 weeks of treatment with CPAP, a control sleep study was done by using the CPAP device on the setting that patients had used for the past 3 weeks. The Epworth Sleepiness Scale and maintenance-of-wakefulness tests were completed at baseline and on the day after the third polysomnographic recording. Time and pressure readings were printed from the machine at the end of the study period. Changes in the apnea + hypopnea index and diurnal hypersomnolence after CPAP therapy and differences in compliance with CPAP therapy among the three treatment groups were the main study end points.

The authors designed the study, and Pierre Médical approved the design. Completion of the study and the collection, interpretation, and presentation of data were not subject to further approval by Pierre Médical. The protocol was authorized by the institutional research board of Hôpital Laval, and written informed consent was obtained from each patient.

Statistical Analysis

To compare the results obtained in the automatic CPAP and constant CPAP groups and the effects of treatment, variables were compared by analysis of covariance of the changes from baseline, with baseline data as covariates. Comparisons between groups were performed by using a Tukey test; the α level was set at 0.05, independent of the results of an F

Table 1. Anthropometric, Sleep, and Breathing Characteristics in the Three Treatment Groups at Baseline*

Characteristic	Patients Receiving Conventional CPAP Given at a Measured Effective Pressure (Group 1) (n = 12)	Patients Receiving Automatic CPAP Given at a Measured Effective Pressure (Group 2) (n = 12)	Patients Receiving Automatic CPAP Given at an Estimated Effective Pressure (Group 3) (n = 12)
Body mass index, kg/m^2	36.8 ± 8.3	36.8 ± 7.8	35.8 ± 7.2
Total sleep time, *h*	5.9 ± 1.0	5.5 ± 1.0	5.8 ± 0.9
Apnea + hypopnea index (events per hour)	50.1 ± 14.5	46.8 ± 22.3	61.5 ± 27.9
Arousal index (arousals per hour)	55.4 ± 21.5	49.7 ± 25.5	66.0 ± 23.2
Time spent below 90% SaO_2, *% total sleep time*	9.3 ± 12.2	6.5 ± 6.8	11.8 ± 12.5
Effective pressure, *cm* H_2O	10.7 ± 2.8	10.9 ± 2.8	11.2 ± 2.0
Estimated pressure, *cm* H_2O	9.7 ± 2.4	9.4 ± 2.3	9.7 ± 2.2
Baseline score on the Epworth Sleepiness Scale	16.1 ± 4.5	13.5 ± 4.7	17.0 ± 4.1
Control score on the Epworth Sleepiness Scale	7.8 ± 4.8	7 ± 3.7	7.9 ± 4.0

* All values are given as the mean ± SD. CPAP = continuous positive airway pressure.

Table 2. Confidence Intervals for Group Differences for Measurements Obtained at the Baseline and Control Visits*

Variable	95% CI for Differences between Groups at Baseline Visit			95% CI for Differences between Groups at Control Visit		
	Groups 1 and 2	Groups 1 and 3	Groups 2 and 3	Groups 1 and 2	Groups 1 and 3	Groups 2 and 3
Body mass index, *kg/m²*	6.7 to 8.9	−7.7 to 7.8	−8.8 to 6.7	–	–	–
Estimated pressure, *cm H_2O*	−1.8 to 2.4	−2.0 to 2.2	−2.3 to 1.9	–	–	–
Effective pressure, *cm H_2O*	−2.1 to 3.1	−2.3 to 2.9	−2.8 to 2.4	–	–	–
Time counter, *hours of CPAP/night*	–	–	–	−0.9 to 1.2	−1.0 to 1.2	−1.2 to 1.0
Pressure counter, *hours of CPAP/night*	–	–	–	−0.6 to 1.4	−0.9 to 1.1	−1.2 to 0.8
Effective pressure to time index, %	–	–	–	−0.5 to 12.5	−5.1 to 6.9	−11.7 to 0.4

* CPAP = continuous positive airway pressure.

test. Differences in CPAP use between the different groups were assessed by using one-way analysis of variance. For observance characteristics, a Fisher exact test was used. All data were analyzed by using the SAS statistical package (SAS Institute, Inc., Cary, North Carolina).

Results

Anthropometric characteristics, sleep architecture and fragmentation, severity of nocturnal breathing difficulty, and diurnal sleepiness did not differ substantially at baseline in the three patient groups (**Table 1**). The 95% CIs for pairwise group comparisons of change in values measured at the baseline visit only and the control visit only and values measured both before and after treatment are shown in **Tables 2** and **3**. The groups did not differ greatly in the effective and estimated pressures. In group 3, estimated pressure was significantly lower than the effective pressure (9.7 ± 2.2 cm H_2O compared with 11.2 ± 2.0 cm H_2O; $P = 0.001$). In this group, the largest overestimation was 0.6 cm H_2O and the largest underestimation was 4.6 cm H_2O.

Nasal obstruction was reported during the course of CPAP therapy in four patients (one in group 1, two in group 2, and one in group 3). This side effect was corrected by installing a hot humidifier in the breathing circuit and simultaneously adjusting the reference pressure.

The three groups had similar improvement in the apnea + hypopnea index in the control sleep study (**Figure 1**, **Tables 3** and **4**). Borderline abnormal index values persisted in two patients in group 1 (14.2 and 13.3) and two patients in group 3 (11.0 and 13.5). The apnea + hypopnea index remained abnormal in one patient in group 3 (19.3). In all three groups, sleep architecture improved substantially after 3 weeks of CPAP therapy, with decreases in stage I and stage II sleep and increases in stage III and stage IV sleep. Sleep fragmentation, measured by the arousal index, improved greatly with CPAP (**Figure 2**). This index remained abnormal in 10 patients (4 in group 1, 4 in group 2, and 2 in group 3). In 1 patient in each group, abnormal scores on the arousal index were explained by frequent periodic leg movements (periodic leg movements index [movements per hour], 38.8, 18.6, and 32.2 for patients in groups 1, 2, and 3, respectively). Sleep fragmentation was not associated with breathing abnormalities or leg movements in the 6 other patients in group 1 and group 2. In group 3, residual obstructive breathing abnormalities persisted in one patient (apnea + hypopnea index, 19.3). This patient had the largest underestimation of estimated pressure.

Diurnal sleepiness was similarly alleviated in the

Table 3. Confidence Intervals for Differences between Changes from Pretreatment to Post-Treatment Values for the Three Treatment Groups

Variable	95% CI for Difference between Change from Pre- to Post-Treatment Values in Group 1 and Change from Pre- to Post-Treatment Values in Group 2	95% CI for Difference between Change from Pre- to Post-Treatment Values in Group 1 and Change from Pre- to Post-Treatment Values in Group 3	95% CI for Difference between Change from Pre- to Post-Treatment Values in Group 2 and Change from Pre- to Post-Treatment Values in Group 3
Total sleep time, *h*	−0.8 to 1.3	−0.8 to 1.3	−1.1 to 1.0
Stage I and II sleep, *% total sleep time*	−14.6 to 5.1	−13.9 to 5.7	−9.3 to 10.4
Stage III and IV sleep, *% total sleep time*	−9.6 to 4.9	−8.3 to 9.0	−6.0 to 8.5
Rapid eye movement sleep, *% total sleep time*	−12.4 to 3.4	−9.5 to 6.3	−5.0 to 10.8
Apnea + hypopnea index (events per hour	−14.4 to 33.0	−19.6 to 27.8	−28.9 to 18.5
Arousal index (arousals per hour	−8.5 to 39.1	−18.6 to 29.0	−33.8 to 13.7
Time spent <90% Sao_2, *% total sleep time*	−10.0 to 6.3	−10.0 to 6.3	−8.1 to 8.2
Sleep latency, *min*	−6.8 to 11.0	−7.2 to 10.7	−9.3 to 8.6
Score on Epworth Sleepiness Scale	−2.2 to 7.4	−3.0 to 6.5	−5.6 to 3.9

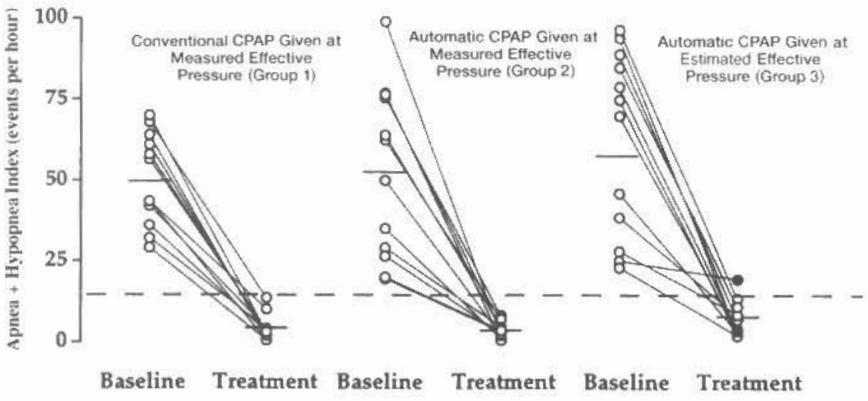

Figure 1. Apnea + hypopnea index values for individual patients observed at baseline and after 3 weeks of continuous positive airway pressure (*CPAP*) therapy in the treatment groups. Each solid line represents one patient. Similar improvement occurred in each group. The black circle indicates one patient in group 3 who had persistent obstructive breathing abnormalities; the black square indicates one patient in group 3 who had residual sleep fragmentation caused by periodic leg movements. Dashed line represents the normal value.

three groups (**Figure 3**, **Table 3**). Sleep latency did not improve in one patient in group 1 and one patient in group 2. The control apnea + hypopnea index was normal in these two patients, but one had unexplained residual sleep fragmentation. Sleep latency remained unchanged in the two patients in group 3 who had persistent sleep fragmentation at the control visit (**Figure 3**). This finding was consistent with the high scores on the Epworth Sleepiness Scale (12 and 17) that were found in these two patients.

During the control CPAP study, we examined the relation among sleep stage, body position, and positive pressure in the two groups that received therapy with automatic CPAP. The mean positive pressure was much higher when patients slept supine (9.3 ± 2.5 cm H_2O) than when they slept on their sides (8.2 ± 2.1 cm H_2O). Compared with sleep stages I and II, the pressure decreased substantially during sleep stages III and IV (8.8 ± 2.8 cm H_2O and 7.4 ± 2.5 cm H_2O, respectively) and increased during rapid eye movement sleep (9.6 ± 2.9 cm H_2O).

Important information was obtained from analyses of the night-by-night variability of the positive pressure. In the 24 patients who received treatment with automatic CPAP, the mean pressure during the 3 weeks of treatment was significantly lower than the effective pressure measured during the baseline titration sleep study (9.5 ± 2.4 cm H_2O and 11.1 ± 2.4 cm H_2O, respectively; $P < 0.001$). In the two automatic CPAP groups, 51.0% ± 7.9% of the CPAP time was spent at a pressure that was lower than the reference pressure. To evaluate the ability of the machine to compensate for the uncertain accuracy of the estimated reference pressure for patients in group 3, we examined the relation between the percentage of CPAP time spent below reference pressure during the treatment period and the difference between effective and estimated pressure. A strong negative correlation between these variables was found ($r = -0.80$; $P = 0.002$) (**Figure 4**). Therefore, the machine was able to decrease the positive pressure to compensate for an underestimation of pressure of less than 4 cm H_2O.

Table 4 shows the nightly duration of home CPAP use by analysis of the time counter and the pressure counter for each group. The effective pressure–time index was significantly higher in groups 2 and 3 than in group 1 ($P = 0.02$) (**Table 2**). One patient with an index of 67% caused the higher index variance in group 1. Without this patient, the difference between the three groups had borderline

Table 4. Characteristics of Sleep and Compliance with Continuous Positive Airway Pressure in the Three Treatment Groups*

Characteristic	Patients Receiving Conventional CPAP Given at a Measured Effective Pressure (Group 1) ($n = 12$)	Patients Receiving Automatic CPAP Given at a Measured Effective Pressure (Group 2) ($n = 12$)	Patients Receiving Automatic CPAP Given at an Estimated Effective Pressure (Group 3) ($n = 12$)
Change in the apnea + hypopnea index (events per hour)	−45.2 ± 11.6	−49.3 ± 26.5	−54.5 ± 29.0
Change in the arousal index (arousals per hour)	−41.4 ± 20.8	−36.1 ± 25.6	−51.4 ± 24.7
Change in the percentage of total sleep time spent below 90% Sao_2	−9.1 ± 12.1	5.9 ± 6.6	6.2 ± 7.6
Change in Epworth Sleepiness Scale score	−8.2 ± 5.3	−6.5 ± 4.2	−9.1 ± 4.9
Time counter, *h/night*	7.1 ± 0.8	7.0 ± 1.1	6.9 ± 1.2
Pressure counter, *h/night*	6.2 ± 1.0	6.5 ± 0.9	6.4 ± 1.1
Effective pressure–time index, %†	86.8 ± 8.6	93.3 ± 4.3	92.5 ± 4.0
Patients with CPAP applied >4 h/d for 5 d/wk, *n*	9	11	12
Patients with CPAP applied >7 h/d for 5 d/wk, *n*	3	6	6

* Values are given as the mean ± SD unless otherwise noted. CPAP = continuous positive airway pressure.
† Significant differences were observed ($P < 0.05$) between group 1 and group 2 and between group 1 and group 3, but not between group 2 and group 3.

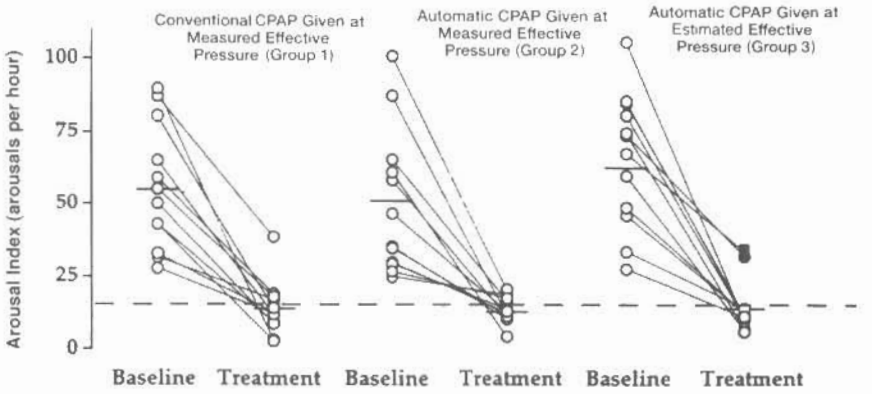

Figure 2. Arousal index values for individual patients obtained at each visit. Sleep fragmentation was similarly alleviated in the three treatment groups. Each line represents one patient. The black circle indicates one patient in group 3 who had persistent obstructive breathing abnormalities; the black square indicates one patient in group 3 who had residual sleep fragmentation caused by periodic leg movements. Dashed line represents the normal value. CPAP = continuous positive airway pressure.

significance ($P = 0.07$). Compliance with CPAP therapy was estimated as the number of patients who received effective treatment for more than 4 or 7 hours per night for at least 5 nights per week. Treatment lasted longer in the two groups that received automatic CPAP therapy than in the group that received conventional CPAP, but this difference was not significant (**Table 4**).

Discussion

We evaluated the feasibility of using the Morphée Plus automatic CPAP machine without doing a titration sleep study in patients with untreated OSAS. Our results suggest that this device can be used effectively with an estimated reference pressure, as shown by the device's ability to compensate for the uncertainty of the estimation and by its effectiveness in normalizing sleep and breathing disorders in most patients.

Estimating the reference pressure can potentially reduce the benefits of automatic CPAP therapy; in our study, however, estimation was accurate enough to allow large pressure decreases in most patients, despite the tendency of the estimation formula to underestimate the reference pressure. Furthermore, estimation did not alter the overall efficacy of the CPAP machine or the potential benefits of the machine with respect to adherence to treatment.

Because the formula tends to underestimate the reference pressure, similar efficiency may be obtained by systematically increasing the interval of pressure changes. However, nonobstructive apneic and hypopneic events and periodic breathing can be seen during CPAP therapy after the obstructive events have resolved (25); it can thus be anticipated that if the increase in pressure is not limited, the positive pressure may increase and interfere with CPAP tolerance and sleep quality. It must be noted that these breathing abnormalities were not observed in our patients with the pressure settings used. Therefore, automatic CPAP therapy relies on highly sensitive detection of breathing disorders and a self-limited pressure increase; our results suggest that these requirements were adequately met with the pressure settings that we used.

We found that the ability of the Morphée Plus device to adjust the pressure to below the reference pressure decreases with the degree of underestimation. Therefore, for a given patient, the difference between the effective and the estimated pressure can be reliably estimated by the percentage of CPAP time spent below reference pressure. This

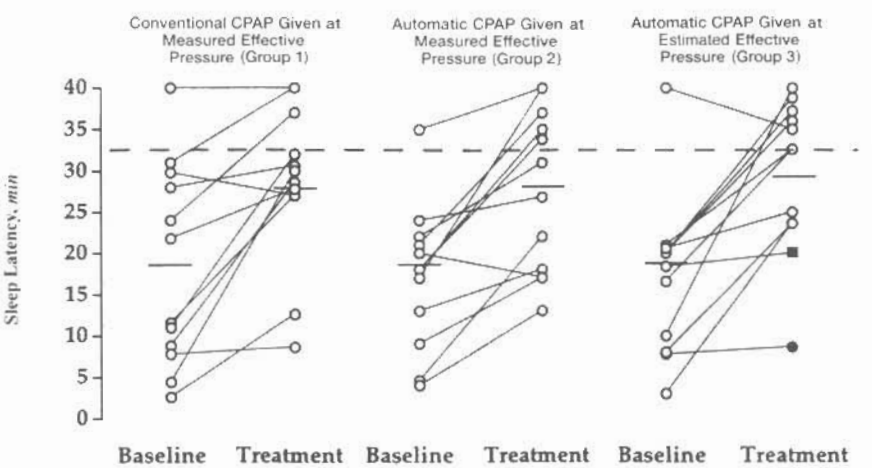

Figure 3. Results of the maintenance-of-wakefulness test for individual patients obtained at each visit. Sleep latency increased greatly by the end of continuous positive airway pressure (CPAP) therapy in the three treatment groups. Each line represents one patient. The black circle indicates one patient in group 3 who had persistent obstructive breathing abnormalities; the black square indicates one patient in group 3 who had residual sleep fragmentation caused by periodic leg movements. Dashed line represents the normal value.

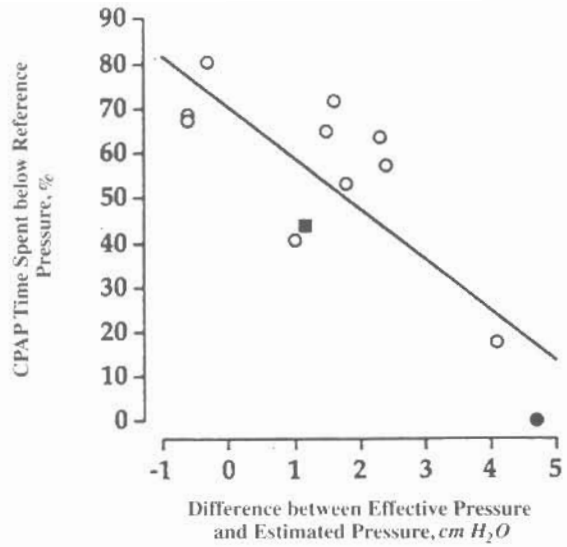

Figure 4. Relation between the values for the percentage of the time during which continuous positive airway pressure (*CPAP*) was applied that was spent below the reference pressure and the difference between the effective and estimated pressure in patients in group 3. A highly significant negative relation was found between these variables. Each symbol represents one patient. The black circle indicates one patient who had persistent obstructive breathing abnormalities, the black square indicates one patient who had residual sleep fragmentation caused by periodic leg movements.

relation can be used to adjust the pressure setting when the clinical response is unsatisfactory. For example, treatment failure was explained by the underestimation of reference pressure in one patient whose nightly pressure data revealed that no CPAP time was spent below the reference pressure (**Figure 4**). This situation can easily be identified and corrected by increasing the reference pressure. Another patient in group 3 who remained hypersomnolent had periodic leg movements, but his breathing abnormalities were effectively corrected by the machine. In this patient, 43.7% of the CPAP time was spent below the reference pressure (**Figure 4**); this finding suggests that the treatment failure was not related to the persistence of sleep-related breathing disorders. If automatic CPAP given without first doing a CPAP titration sleep study is an important step in the treatment of OSAS, this strategy will further justify the need for early clinical follow-up in these patients. In patients with unsatisfactory clinical responses, a control sleep study must be done to evaluate the persistence of respiratory or nonrespiratory sleep disorders. We think that the optimal adaptation of the pressure setting should be determined during the first weeks of treatment because the initial response to CPAP may reliably indicate the need for long-term treatment (24). It is important to understand that reevaluations of CPAP treatment may be indicated not only in patients receiving treatment with automatic CPAP but also in those receiving treatment with conventional CPAP, as suggested by the persistence of diurnal somnolence and sleep fragmentation in some patients in group 1.

It has been proposed that the diagnostic nasal CPAP titration sleep studies can be cond during a single night (split-night study) (26). use of an automatic CPAP machine without titration could replace split-night studies because automatic CPAP is adaptable to the clinical situations that can alter the effective pressure. Also, analysis of the pressure trend can be used to adjust the reference pressure without doing a CPAP titration study. In addition, the effective pressure is underestimated during sleep studies in 49% to 58% of patients with OSAS (26, 27). It is therefore reasonable to hypothesize that conventional CPAP set at a pressure determined during a split-night study may not be as efficient as automatic CPAP initiated without doing a titration study. Because pressure settings, the variables that are analyz and the algorithms of pressure changes may differ dramatically between automatic CPAP machines, caution must taken before the results of our study are extended to other automatic CPAP machines.

Substantial differences in compliance with CPAP therapy were found between the conventional and automatic CPAP groups. Even among patients who received conventional CPAP, compliance was better in our study sample (75%) than in those in other reports (46%) (24). This might be because our patients knew that they were participating in a clinical study. The better adherence to treatment noted with automatic CPAP may improve the effectiveness of treatment for diurnal symptoms, as shown by the reappearance of neuropsychological abnormalities as soon as CPAP therapy is interrupted (28). A similar number of patients in the three groups required the use of a humidifier to correct nasal discomfort. Therefore, we believe that the observed difference in compliance was not related to a reduction in side effects but to an improvement in comfort with automatic CPAP as a consequence of the continuous adaptation of the positive pressure throughout the night. We cannot exclude the possibility that the CPAP titration night might have influenced CPAP compliance, particularly in groups 2 and 3. Therefore, we cannot speculate what CPAP compliance would be in patients who received automatic CPAP and had no previous experience with this treatment. Future studies are required to determine the feasibility and the repercussions of this procedure in clinical practice.

We conclude that automatic CPAP can be used with an estimated reference pressure in patients potentially receiving an initial trial of CPAP therapy at home. The amount of CPAP time spent below the reference pressure can be used to estimate the effective pressure if treatment failure is caused by

an inadequate pressure setting and, if so, to determine the correct pressure.

Acknowledgment: The authors thank Nathalie Dupuis, Andrée Veilleux, Sandra Jolin, Michel Leblond, Isabelle Boucher, and Germain Ethier for technical assistance and Serge Simard for statistical analysis.

Grant Support: In part by Pierre Médical France.

Requests for Reprints: Frédéric Sériès, MD, Centre de pneumologie, Hôpital Laval, 2725 chemin Sainte-Foy, Sainte-Foy, Québec G1V 4G5, Canada.

Current Author Addresses: Drs. Sériès and Marc: Centre de pneumologie, Hôpital Laval, 2725 chemin Sainte-Foy, Sainte-Foy, Québec G1V 4G5, Canada.

References

1. **Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S.** The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med. 1993;328:1230-5.
2. **Partinen M, Guilleminault C.** Daytime sleepiness and vascular morbidity at seven-year follow-up in obstructive sleep apnea patients. Chest. 1990;97:27-32.
3. **Kales A, Caldwell AB, Cadieux RJ, Vela-Bueno A, Ruch LG, Mayes SD.** Severe obstructive sleep apnea—II: Associated psychopathology and psychosocial consequences. J Chronic Dis. 1985;38:427-34.
4. **Millman RP, Fogel BS, McNamara ME, Carlisle CC.** Depression as a manifestation of obstructive sleep apnea: reversal with nasal continuous positive airway pressure. J Clin Psychiatry. 1989;50:348-51.
5. **He J, Kryger MH, Zorick FJ, Conway W, Roth T.** Mortality and apnea index in obstructive sleep apnea: Experience in 385 male patients. Chest. 1988;94:9-14.
6. **Smith PL, Gold AR, Meyers DA, Haponik EF, Bleecker ER.** Weight loss in mildly to moderately obese patients with obstructive sleep apnea. Ann Intern Med. 1985;103:850-5.
7. **Fujita S, Conway WA, Zorick FJ, Sicklesteel JM, Roehrs TA, Wittig RM, et al.** Evaluation of the effectiveness of uvulopalatopharyngoplasty. Laryngoscope. 1985;95:70-4.
8. **O'Sullivan RA, Hillman DR, Mateljan R, Pantin C, Finucane KE.** Mandibular advancement splint: an appliance to treat snoring and obstructive sleep apnea. Am J Respir Crit Care Med. 1995;151:194-8.
9. **Sullivan CE, Issa FG, Berthon-Jones M, Eves L.** Reversal of obstructive sleep apnea by continuous positive airway pressure applied through the nares. Lancet. 1981;1:862-5.
10. **Derderian SS, Bridenbaugh RH, Rajagopal KR.** Neuropsychologic symptoms in obstructive sleep apnea improve after treatment with nasal continuous positive airway pressure. Chest. 1988;94:1023-7.
11. **Sforza E, Krieger J, Weitzenblum E, Apprill M, Lampert E, Ratamaharo J.** Long-term effects of treatment with nasal continuous positive airway pressure on daytime lung function and pulmonary hemodynamics in patients with obstructive sleep apnea. Am Rev Respir Dis. 1990;141:866-70.
12. **Miljeteig H, Hoffstein V.** Determinants of continuous positive airway pressure level for treatment of obstructive sleep apnea. Am Rev Respir Dis. 1993;147(6 Pt 1):1526-30.
13. **Berthon-Jones M.** Feasibility of a self-setting CPAP machine. Sleep. 1993;16(8 Suppl):S120-1.
14. **Issa FG, Sullivan CE.** Upper airway closing pressures in snorers. J Appl Physiol. 1984;57:528-35.
15. **Issa FG, Sullivan CE.** Upper airway closing pressures in obstructive sleep apnea. J Appl Physiol. 1984;57:520-7.
16. **Thut DC, Schwartz AR, Roach D, Wise RA, Permutt S, Smith PL.** Tracheal and neck position influence upper airway airflow dynamics by altering airway length. J Appl Physiol. 1993;75:2084-90.
17. **Meurice JC, Marc I, Carrier G, Sériès F.** Effects of mouth opening on upper airway collapsibility in normal sleeping subjects. Am J Respir Crit Care Med. 1996;153:255-9.
18. **Wasicko MJ, Hutt DA, Parisi RA, Neubauer JA, Mezrich R, Edelman NH.** The role of vascular tone in the control of upper airway collapsibility. Am Rev Respir Dis. 1990;141:1569-77.
19. **Meurice JC, Marc I, Sériès F.** Efficiency of auto-CPAP in the treatment of obstructive sleep apnea/hypopnea syndrome. Am J Respir Crit Care Med. 1996;153:794-8.
20. **Kales A, Rechtschaffen A, eds.** A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects. Bethesda, MD: U.S. National Institute of Neurological Diseases and Blindness, Neurological Information Network; 1968. National Institutes of Health publication no. 204.
21. **American Sleep Disorders Association.** EEG arousals: scoring rules and examples. Sleep. 1992;15:174-84.
22. **Johns MW.** Daytime sleepiness, snoring, and obstructive sleep apnea. The Epworth Sleepiness Scale. Chest. 1993;103:30-6.
23. **Poceta JS, Timms RM, Jeong DU, Ho JL, Erman MK, Mitler MM.** Maintenance of wakefulness test in obstructive sleep apnea syndrome. Chest. 1992;101:893-7.
24. **Kribbs NB, Pack AI, Kline LR, Smith PL, Schwartz AR, Schubert NM, et al.** Objective measurement of patterns of nasal CPAP use by patients with obstructive sleep apnea. Am Rev Respir Dis. 1993;147:887-95.
25. **Marrone O, Stallone A, Salvaggio A, Milone F, Bellia V, Bonsignore G.** Occurrence of breathing disorders during CPAP administration in obstructive sleep apnoea syndrome. Eur Respir J. 1991;4:660-6.
26. **Sanders MH, Kern NB, Costantino JP, Stiller RA, Studnicki K, Coates J, et al.** Adequacy of prescribing positive airway pressure therapy by mask for sleep apnea on the basis of a partial-night trial. Am Rev Respir Dis. 1993;147:1169-74.
27. **Yamashiro Y, Kryger MH.** CPAP titration for sleep apnea using a split-night protocol. Chest. 1995;107:62-6.
28. **Kribbs NB, Pack AI, Kline LR, Getsy JE, Schuett JS, Henry JN, et al.** Effects of one night without nasal CPAP treatment on sleep and sleepiness in patients with obstructive sleep apnea. Am Rev Respir Dis. 1993;147:1162-8.