

Efficacy of Auto-CPAP in the Treatment of Obstructive Sleep Apnea/Hypopnea Syndrome

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The auto-CPAP (Morphée Plus) is characterized by its ability to modify the positive-pressure level applied during the night for the presence or absence of sleep-induced respiratory disorders. The aim of the study was to compare the efficacy of this new mode of CPAP therapy with that of conventional constant-CPAP in the treatment of sleep apnea/hypopnea syndrome (SAHS). Sixteen patients with SAHS were randomly allocated to two groups that were paired for age, apnea/hypopnea index, and mean sleep latency. In the auto-CPAP group, the pressure level could change within fixed limits in both directions (+2 to -4 cm H₂O) of the previously determined effective pressure level (Peff). In the constant-CPAP group, patients used the same apparatus (Morphée Plus) in a constant mode at Peff level. At the beginning of the study, the Peff level was determined during a polysomnographic recording. Daytime vigilance was measured subjectively by a standardized questionnaire and objectively by the maintenance of wakefulness test (MWT); Trailmaking tests (TMT) were used to evaluate cognitive functions. After 3 wk of home CPAP therapy, a control sleep study was done with the CPAP machine used in the protocol, and daytime vigilance and cognitive function tests were obtained. Baseline sleep and nocturnal breathing disorders characteristics did not differ between the two groups, and daytime vigilance and cognitive function abnormalities were similarly altered. In both groups, the apnea/hypopnea index was within normal range at the final CPAP sleep study. In the auto-CPAP group, 49.3 ± 14.9% (mean ± SD) of home treatment time was spent at a pressure ≤ Peff. Home amount of use estimated by the number of sleeping hours with a positive pressure applied was 6.5 ± 1.0 h in the auto-CPAP group and 5.1 ± 1.1 h in the constant-CPAP group (p = 0.02). During the control CPAP sleep study, the positive pressure level was significantly lower during Stage III-IV than during the other sleep stages (p = 0.004). The improvement in the MWT and the TMT observed with CPAP therapy was identical in both groups. We conclude that (1) the amount of use during CPAP treatment is higher with auto-CPAP than with constant-CPAP, and (2) Morphée + auto-CPAP is as efficient as conventional CPAP in correcting nocturnal breathing disorders, daytime sleepiness, and cognitive impairment in SAHS.

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Sleep apnea/hypopnea syndrome (SAHS) is an important public health problem since its prevalence is 4% of middle-aged men and 2% of middle-aged women (1). It is associated with an important increase in the morbidity rate related to cardiovascular and cerebrovascular diseases (2) and to neuropsychologic disfunctions (3, 4). These neuropsychologic disturbances can be induced by recurrent SaO₂ drops and arousals that are responsible for sleep fragmentation and disorganization. There is suggestive evidence that SAHS is also associated with an increase in mortality (5). This increase in risk factors emphasizes the need for an effective treatment of SAHS. The usefulness of nasal continuous positive airway pressure (CPAP) in the treatment of

SAHS was first reported by Sullivan and colleagues (6) in 1981. It is still considered as the most effective treatment. CPAP therapy is associated with an improvement in morbidity and mortality (5, 7-10). Its regular use is, however, associated with numerous side effects related to nasal airflow and pressure discomfort (11-15) that reduce CPAP acceptance. Nasal CPAP pattern of use varies from one study to another according to the selected criteria and the mode of evaluation. When evaluated by time counters or by patients' reports, 60 to 75% of patients are considered to use nasal CPAP satisfactorily. Objective measurement using mask pressure analysis has revealed that the estimated amount of use has been overestimated since only 46% of the patients met criteria for regular use (16). On a short-term basis, cognitive performance improves even at low levels of CPAP amount of use (17). However, beneficial effects of CPAP treatment depends on its regular use since the neuropsychologic consequences of SAHS such as excessive daytime sleepiness reappear as soon as it is interrupted (18).

The effective positive-pressure level (Peff) generally corresponds to the level of pressure that abolishes apnea, hypopnea,

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and snoring in all sleep stages and in all body positions. However, Peff may vary in different clinical situations such as after the intake of alcohol and sedative medication and after changes in body position (19, 20). The effective positive pressure level is also influenced by sleep stages, upper airway collapsibility being higher in Stages I-II and REM than in slow wave sleep (SWS) (19). Furthermore, recent results from our laboratory have demonstrated that Peff progressively decreases during the course of CPAP therapy (21). This suggests that the positive pressure requirements may change during one night and from one night to another on a short- and long-term basis. It may be anticipated that the continuous adaptation of the pressure settings to the patient's need could improve CPAP amount of use.

This has led to the development of a new positive pressure device that continuously adapts the positive pressure level during the night (auto-CPAP) around a predetermined Peff (22). This nasal CPAP apparatus automatically modifies the pressure level, allowing a decrease in the pressure level when apnea and hypopneas disappear and an increase in this pressure level when the sleep respiratory disorders reappear. In a short-term trial, this apparatus has been found to be as efficient as conventional constant-CPAP in correcting sleep-related breathing disorders, nocturnal desaturations, and sleep fragmentation (23). However, the patients enrolled in this previous study were already treated by CPAP, and the effects of auto-CPAP on the pattern of use to CPAP therapy and on daytime sleepiness were not evaluated. Therefore, the aim of the present study was to analyze prospectively the influence of the auto-CPAP mode on sleep and breathing characteristics, daytime vigilance, and cognitive impairment in patients with previously untreated SAHS and compare it with conventional CPAP.

METHODS

Patients

Sixteen male patients with untreated SAHS (age, 54 ± 11 yr; body mass index, 34.2 ± 5.7 kg/m²; mean \pm SD) were included in this study. The clinical diagnosis of SAHS was confirmed by initial standard polysomnography: apnea/hypopnea index (A/HI) = 43.6 ± 19.8 n/h. Daytime vigilance was assessed at baseline by the maintenance of wakefulness test (MWT) (24) and the psychometric test (Trailmaking test) (25). Subjective evaluation of daytime vigilance was obtained by a clinical score obtained from the Epworth Sleep Questionnaire (26).

Auto-CPAP Characteristics

The "Morphée Plus" apparatus (Laboratoire Pierre Médical, Verrières Le Buisson, France) is characterized by its ability to adapt the level of pressure applied during the night according to the presence or absence of respiratory abnormalities. The positive pressure is allowed to change within a determined pressure range. The upper and the lower thresholds are chosen separately by the physician on each direction of the Peff. Generally (as proposed by the manufacturer) these upper and lower limits are +2 and -4 cm H₂O, respectively, from Peff. During the first 20 min of the night, a constant pressure level equal to Peff is applied. In the absence of apneic/hypopneic events, the pressure progressively decreases below Peff, down to the lower pressure threshold, at a rate of 1 cm H₂O/every 30 s. This pressure level is maintained until respiratory disorders reappear that will result in a progressive increase in the pressure level. The pressure rise varies between 1 and 3 cm H₂O, at a rate of 1 cm/s depending on the severity of the respiratory abnormality (see below). Then, in the absence of a new breathing disorder for more than 4 min, the positive pressure level decreases again as described above.

The pressure adaptation is regulated by a constant feedback analysis of the patient's ventilation by the nasal CPAP apparatus according to the flow regime provided by the CPAP compressor. Because the inspiratory-expiratory pressure changes are automatically compensated by instantaneous electronic pressure regulation, the corresponding changes in the speed of the compressor are used to estimate the difference between maximal inspiratory and expiratory flow. The stability of the

breath-by-breath changes in the speed of the compressor indicates the absence of respiratory disorder and allows the pressure level to decrease. The presence of obstructive breathing abnormalities is associated with a decrease in the inspiratory-expiratory difference in the flow regime, leading to an increase in the pressure level until the stability of the flow regime of the compressor or the fixed upper limit of the pressure level has been reached.

Sleep Studies

The polysomnographic studies consisted in a continuous acquisition of electroencephalogram (EEG) with surface electrodes placed at C₄/A₁, C₃/A₂, O₂/A₁, and O₁/A₂, submental electromyogram (EMG), electrooculogram (EOG), electrocardiogram (EKG), nasobuccal airflow with thermistors, thoracoabdominal movements by inductive plethysmography (Respirace®; Ambulatory Monitoring, Ardsley, NY), arterial oxygen saturation (SaO₂) with an ear oximeter (504 Pulse oximeter; Criticare Systems, Waukesha, WI), and breathing noises with two microphones placed at the head of the bed (27). All variables were recorded on computer (SomnoStar; SensorMedics, Yorba Linda, CA).

Maintenance of Wakefulness Test

The MWT was administered according to previously published recommendations (24). The patient was seated in an armchair and was asked to remain awake as long as possible during four consecutive 40-min trials every 2 h after the light had been turned off. Each MWT was terminated after 10 min of sleep, sleep onset being defined as three continuous epochs of Stage 1 sleep or any epoch of Stages 2 to 4 or REM sleep. The mean sleep latency upon the four tests was measured.

Trailmaking Test

This test has previously been used to evaluate brain damage (25) and requires alertness and concentration. It consists of two parts, each with 25 circles distributed over a white sheet of paper. In Part A the circles are numbered from 1 to 25. The patient is asked to draw a line connecting the circles in numerical sequence as quickly as possible. Part B includes numbers from 1 to 13 and letters from A to L. The patient is asked to alternate between numbers and letters as he proceeds in an ascending sequence. The score corresponds to the time needed to complete each part, including the additional correction time during each test.

Study Design

At the beginning of the study each patient underwent a first conventional polysomnographic recording to confirm the presence of sleep-related breathing disorders; daytime sleepiness and cognitive functions were quantified with the Epworth Sleep Questionnaire, the MWT, and the Trailmaking test. A second polysomnography was done within 1 wk to determine the level of effective positive pressure that abolished obstructive apnea, hypopnea, and snoring (Peff). Patients were then randomly allocated into two different groups that were paired for age, A/HI, and mean sleep latency. The two groups differed by the CPAP mode used during the study period: in the auto-CPAP group, the CPAP machine was used in the automatic mode, and in the constant-CPAP group, it was used with a constant pressure level equal to Peff. Both groups used the same CPAP apparatus, and the patients were blind to the group they were allocated to. In the auto-CPAP group the pressure limits were 2 cm H₂O above and 4 cm H₂O below Peff. All patients were treated for 3 wk. At the end of the study period, all the patients had a control polysomnographic recording while using the CPAP apparatus in the same mode as during the previous 3 wk. No adjustment in the pressure characteristics was done during this last sleep recording. The Epworth Sleep Questionnaire was completed, and the MWT and the Trailmaking test were realized during the day after the last polysomnographic recording. The protocol was authorized by the Institutional Research Board, and a written informed consent was obtained for each subject.

Data Collection and Statistical Analysis

Polysomnographic recordings were manually interpreted in 30-s epochs by a technician who was blind to the CPAP mode used by the patients. Sleep parameters were determined according to the criteria of Rechtschaffen and Kales (28) with Stages I and II combined in light sleep (LS) and Stages III and IV combined in SWS. The number of arousals per hour (29) was obtained. Apnea was defined as the cessation of nasal

oral airflow for at least 10 s. Hypopnea was defined as a more than 50% decrease in the nasal-oral airflow for at least 10 s associated with a 4% drop in SaO_2 and/or an arousal. Two SaO_2 variables were studied: (1) awake ($w\text{SaO}_2$) and (2) mean nocturnal ($m\text{SaO}_2$). During the period of CPAP therapy, the mean pressure level, the proportion of time spent at the different pressure levels, and the time when a positive pressure was applied were automatically computed by the CPAP apparatus. These pressure characteristics were also measured during the control sleep study by continuous analysis of the mask pressure. The amount of use was estimated by time-counter data and pressure measurements during the study period.

Comparisons were performed with a multivariate analysis of variance and Student's t-test, as variance homogeneity and normality were encountered. In order to compare the results obtained in the auto-CPAP and constant-CPAP groups and the effects of treatment, the different variables were compared by analysis of covariance of the changes from baseline with baseline data as covariates. In the auto-CPAP group, the P_{eff} and the mean pressure obtained during the study period and during the control sleep study were compared using Hotelling's T^2 test (30). Statistical significance was inferred for comparisons at $p < 0.05$.

RESULTS

The two groups did not differ in their sleep and respiratory characteristics measured at baseline and with CPAP at the end of the study (Table 1). P_{eff} was identical in the two groups (9.5 ± 3.1 and 9.1 ± 3.1 cm H_2O in the constant- and auto-CPAP groups, respectively, mean \pm SD). The improvement in the A/HI observed at the control sleep study was similar in the two groups (Table 1). Baseline sleep architecture was identical between the two groups and similarly improved after 3 wk of CPAP therapy, as illustrated in Figure 1. The amount of light sleep significantly decreased ($p = 0.0003$), associated with a significant increase in SWS ($p = 0.001$) and REM sleep ($p = 0.01$).

The score of the Epworth Sleep Questionnaire was identical in the two groups at baseline and after CPAP treatment, with a significant improvement in the daytime vigilance at the end of the study in both groups ($p = 0.0001$) (Table 2). The sleep latencies were similar in the two groups before CPAP treatment and at the end of the study period (Table 2). The improvement in daytime vigilance was identical and significant in both groups ($p = 0.01$). Mean Trail making test (TMT A and B) scores were similar in the two groups at the initial and final visits, with a significant improvement in TMT A score with CPAP therapy in both groups ($p = 0.0002$) (Table 2). The changes in TMT B were not significant.

In the auto-CPAP group, the analysis of the pressure behavior during the study period showed that the patients spent $49.3 \pm 15\%$ of the time with CPAP at a pressure level below or equal to P_{eff} . In the auto-CPAP group, we analyzed the influence of

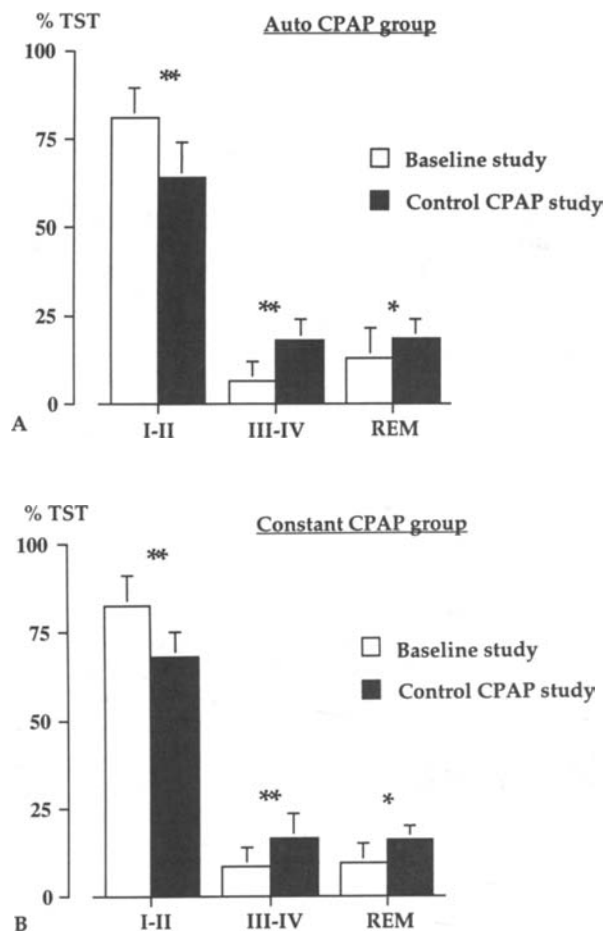


Figure 1. Sleep architecture at baseline and at CPAP control sleep studies in the Auto-CPAP group (A) and in the Constant-CPAP group (B). The values are expressed in percentage of total sleep time (TST) (mean \pm SD). I-II = light sleep (Stages I and II); III-IV = slow wave sleep (Stages III and IV); REM = rapid eye movement sleep (* $p < 0.05$; ** $p < 0.01$).

sleep stage on the positive pressure level during the control CPAP sleep study. The mean positive pressure level significantly decreased ($p = 0.004$) during Stage III-IV compared with those during Stage I-II and REM sleep (Figure 2). The mean P_{mask} measured during the control sleep study was not significantly different from that measured at home during the study period (8.4 ± 2.8 and 8.6 ± 3.2 cm H_2O , respectively).

TABLE 1

SLEEP AND BREATHING CHARACTERISTICS AT BASELINE AND DURING CPAP CONTROL STUDIES IN THE AUTO-CPAP AND CONSTANT-CPAP GROUPS*

	Baseline		Control	
	Auto-CPAP	Constant-CPAP	Auto-CPAP	Constant-CPAP
TST, h	5.7 \pm 1.2	6.2 \pm 0.9	6.2 \pm 1.1	6.6 \pm 1.1
A/HI, n/h	40.5 \pm 17.7	46.8 \pm 22.3	1.7 \pm 1.2	2.6 \pm 3.0
Arousal index, n/h	36.3 \pm 16.4	36.1 \pm 16.9	10.1 \pm 2.5	9.0 \pm 3.0
$w\text{SaO}_2$, %	95.8 \pm 4.0	95.0 \pm 3.4	97.0 \pm 4.0	98.0 \pm 2.0
$m\text{SaO}_2$, %	92.9 \pm 3.4	91.1 \pm 4.0	96.0 \pm 3.0	96.0 \pm 2.0

Definition of abbreviations: TST = total sleep time; A/HI = apnea-hypopnea index; Arousal index = number of arousals per hour; $w\text{SaO}_2$ = awake SaO_2 ; $m\text{SaO}_2$ = mean nocturnal SaO_2 .

* Values are mean \pm SD.

TABLE 2

MAINTENANCE OF WAKEFULNESS TESTS AND TRAILMAKING TESTS AT BASELINE AND POST-CPAP STUDIES IN AUTO-CPAP AND CONSTANT-CPAP GROUPS*

	Baseline		Post-CPAP	
	Auto-CPAP	Constant-CPAP	Auto-CPAP	Constant-CPAP
ESS score	15.2 \pm 4.2	14.4 \pm 6.3	5.6 \pm 3.7†	8.6 \pm 6.9†
MWT, min	18.2 \pm 11.2	19.0 \pm 13.9	26.9 \pm 12.0†	26.1 \pm 14.6†
Trailmaking A, s	41.1 \pm 16.2	36.4 \pm 11.4	32.6 \pm 16.2†	32.6 \pm 10.7†
Trailmaking B, s	85.3 \pm 45.6	93.1 \pm 14.9	73.8 \pm 52.6	79.9 \pm 22.7

Definition of abbreviations: ESS = Epworth Sleepiness Score; MWT = maintenance of wakefulness test.

* Values are mean \pm SD.

† Significantly different between the baseline and the post-CPAP visits ($p < 0.05$).

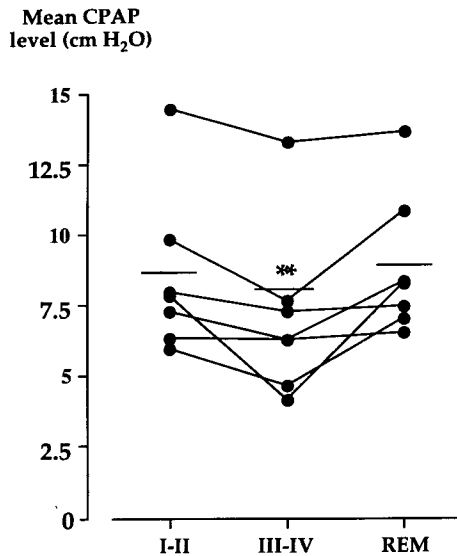


Figure 2. Individual and mean CPAP level measured during the different sleep stages during CPAP control polysomnographic study in the auto-CPAP group. I-II = light sleep (Stages I and II); III-IV = slow wave sleep (Stages III and IV); REM = rapid eye movement sleep (** $p = 0.004$).

The nightly duration of home CPAP utilization given by time counter analysis was 7.1 ± 1.0 h in the auto-CPAP group and 5.7 ± 1.1 h in the constant-CPAP group, the difference being of borderline significance ($p = 0.05$). However, this difference was significant when considering the number of hours per night that a positive pressure was applied during the study period (auto-CPAP group, 6.5 ± 1.0 h; constant-CPAP group, 5.1 ± 1.1 h; $p = 0.02$).

DISCUSSION

The aim of our study was to analyze the efficacy of a new CPAP mode that is able to generate variable pressure levels in correcting nocturnal breathing disorders and also evaluate the consequences on sleep and daytime function in SAHS. We were also interested in comparing these effects with those obtained using a conventional constant-CPAP. The major findings of this study are (1) that the auto-CPAP is as effective as the constant-CPAP in resolving sleep-related breathing disorders, nocturnal desaturations, and sleep fragmentation and in improving daytime sleepiness and cognitive impairment; (2) the amount of use of CPAP therapy is better with auto-CPAP than with constant-CPAP.

As previously reported, we found no difference in the A/HI nor in SaO_2 characteristics between patients treated by auto-CPAP or by constant-CPAP. This suggests that the auto-CPAP apparatus satisfactorily detects respiratory disorders, and that even if some breathing abnormalities may persist because of the adaptation of the positive pressure requirements, their frequency remains minimal. Auto-CPAP was not associated with any increase in the arousal index compared with constant-CPAP, and the improvement in sleep architecture was the same in both groups. This is an important point because it could be questioned if the sudden rises in positive pressure could have theoretically induced arousals or awakenings.

The analysis of the changes in mask pressure during the control polysomnography with nasal CPAP at the end of the study pointed out that the mean positive pressure significantly decreased during Stage III-IV sleep compared with those during the other

sleep stages. This confirms the results observed by Robert and colleagues (22), and it is consistent with the results of Issa and Sullivan (20) who found that the upper airway closing pressure is higher in light sleep and REM than in deep sleep. This sleep stage dependence of the efficient positive pressure level in association with the position dependence of this parameter may have contributed to a better adaptation of the required efficient pressure during the night. This may be an important determinant of the improvement in the amount of use of nasal CPAP since we found that it was significantly increased in the auto-CPAP group compared with that in the constant-CPAP group. It is in accordance with our previous results (23), which showed a subjective enhancement of comfort with the use of auto-CPAP in patients already treated with conventional CPAP.

This study clearly demonstrated that the auto-CPAP mode that we used was as effective as constant-CPAP. Because no difference was found between auto-CPAP and constant-CPAP in sleep and breathing characteristics and in neuropsychologic improvement, it can be asked if the increased amount of use noted with auto-CPAP is of clinical significance. Several arguments suggest that this improved amount of use may benefit the patients. First, our results were obtained after short-term CPAP therapy. Because the initial amount of use may be a reliable indicator of the long-term application of the treatment (16), it is highly probable that this difference in pattern of use between auto- and constant-CPAP modes will be further enhanced on a long-term basis. Second, this study was realized during a period of clinical stability (sleep conditions, absence of nasal obstruction, no medication that could perturb sleep quality), and the patients were aware that they participated in a clinical research study. These conditions were probably associated with an improvement in amount of use. It can be anticipated that in uncontrolled conditions, the treatment pattern of use would be worse. Because during auto-CPAP therapy the level of positive pressure will continue to be adapted to the patients' requirements, the difference in amount of use between auto- and constant-CPAP will probably be further enhanced in clinical practice. Because neuropsychologic abnormalities reappear even during the first day after CPAP interruption (18), an increased amount of use should be accompanied by an improvement in vigilance and cognitive function. Third, on a long-term basis, as the effective pressure level decreases over time (21), the auto-CPAP device will adequately deal with intranight and internight variability of Peff.

In conclusion, we found that auto-CPAP is as effective as constant-CPAP in the treatment of sleep-related breathing disorders. Long-term evaluation of this CPAP mode would be particularly interesting if it confirmed an increase in the amount of use of CPAP in poorly compliant subjects, and if it identified the evolution of mean pressure level with time and the changes in weight.

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References

1. Young, T. E., M. Palta, J. Dempsey, J. Skatrud, S. Weber, and S. Badr. 1993. The occurrence of sleep-disordered breathing among middle-aged adults. *N. Engl. J. Med.* 328:1230-1235.
2. Partinen, M., and C. Guilleminault. 1990. Daytime sleepiness and vascular morbidity at seven-year follow-up in obstructive sleep apnea patients. *Chest* 97:27-32.
3. Kales, A., A. B. Caldwell, R. J. Cadieux, A. Velo-Bueno, L. G. Ruch, and S. D. Mayes. 1985. Severe obstructive sleep apnea: associated psychopathology and psychosocial consequences. *J. Chronic Dis.* 38: 427-434.
4. Millman, R. P., B. S. Fogel, M. E. McNamara, and C. C. Carlisle.

1989. Depression as a manifestation of obstructive sleep apnea: reversal with nasal continuous positive airway pressure. *J. Clin. Psychiatry* 50:348-351.
5. He, J., M. H. Kryger, F. J. Zorcik, W. Conway, and T. Roth. 1988. Mortality and apnea index in obstructive sleep apnea. *Chest* 94:9-14.
 6. Sullivan, C. E., F. Q. Issa, M. Berthon-Jones, and L. Eves. 1981. Reversal of obstructive sleep apnea by continuous positive airway pressure applied through the nares. *Lancet* 1:862-865.
 7. Derderian, S. S., H. Bridenbaugh, and K. R. Rajagopal. 1988. Neuropsychologic symptoms in obstructive sleep apnea improve after treatment with nasal continuous positive airway pressure. *Chest* 94:1023-1027.
 8. Sanders, M. H. 1984. Nasal CPAP effect on patterns of sleep apnea. *Chest* 86:829-844.
 9. Sforza, E., J. Krieger, E. Weitzenblum, M. Apprill, E. Lampert, and J. Ratamaharo. 1990. 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.* 141:866-870.
 10. Leech, J. A., E. Onal, and M. Lopata. 1992. Nasal CPAP continues to improve sleep-disordered breathing and daytime oxygenation over long term follow-up of occlusive sleep apnea syndrome. *Chest* 102:1651-1655.
 11. Sanders, M. H., C. A. Gruendl, and R. M. Rogers. 1986. Patient compliance with nasal CPAP therapy for sleep apnea. *Chest* 90:330-333.
 12. Waldhorn, R. E., T. W. Herrick, M. C. Nguyen, A. E. O'Donnell, J. Sodero, and S. J. Potolicchio. 1990. Long term compliance with nasal continuous positive airway pressure therapy of obstructive sleep apnea. *Chest* 97:33-38.
 13. Rolfe, I., L. G. Olson, and N. A. Saunders. 1991. Long-term acceptance of continuous positive airway pressure in obstructive sleep apnea. *Am. Rev. Respir. Dis.* 144:1130-1133.
 14. Hoffstein, V., S. Viner, S. Mateika, and J. Conway. 1992. Treatment of obstructive sleep apnea with nasal continuous positive airway pressure. *Am. Rev. Respir. Dis.* 145:841-845.
 15. Meurice, J. C., P. Dore, J. Paquereau, J. P. Neau, P. Ingrand, J. J. Chavagnat, and F. Patte. 1994. Predictive factors of long-term compliance with nasal continuous positive airway pressure treatment in sleep apnea syndrome. *Chest* 105:429-433.
 16. Kribbs, N. B., A. I. Pack, L. R. Kline, P. L. Smith, A. R. Schwartz, N. M. Schubert, S. Redline, J. N. Henry, J. E. Getsy, and D. F. Dinges. 1993. Objective measurement of patterns of nasal CPAP use by patients with obstructive sleep apnea. *Am. Rev. Respir. Dis.* 147:887-895.
 17. Engleman, H. M., S. E. Martin, I. J. Deary, and N. J. Douglas. 1994. Effect of continuous positive airway pressure treatment on daytime function in sleep apnoea/hypopnoea syndrome. *Lancet* 343:572-575.
 18. Kribbs, N. B., A. I. Pack, L. R. Kline, J. E. Getsy, J. S. Schuett, J. N. Henry, G. Maislin, and D. F. Dinges. 1993. Effects of one night without nasal CPAP treatment on sleep and sleepiness in patients with obstructive sleep apnea. *Am. Rev. Respir. Dis.* 147:1162-1168.
 19. Issa, F. G., and C. E. Sullivan. 1984. Upper airway closing pressures in snorers. *J. Appl. Physiol.* 57:528-532.
 20. Issa, F. G., and C. E. Sullivan. 1984. Upper airway closing pressures in obstructive sleep apnea. *J. Appl. Physiol.* 57:520-527.
 21. Sériès, F., I. Marc, Y. Cormier, and J. La Forge. 1994. Required levels of nasal continuous positive airway pressure during treatment of obstructive sleep apnoea. *Eur. Respir. J.* 7:1776-1781.
 22. Robert, D., G. Bourdon, B. Langevin, P. Leger, and A. Guez. 1993. Usefulness and efficacy of an autoadjustable CPAP machine in treatment of OSAS (abstract). *Am. Rev. Respir. Dis.* 147(Suppl. 4):A680.
 23. Meurice, J. C., J. Paquereau, D. Recart, F. Series, P. Dore, J. P. Neau, and F. Patte. 1994. Efficiency and tolerance of auto-CPAP in the treatment of sleep apnea/hypopnea syndrome (abstract). *Am. J. Respir. Crit. Care Med.* 149:A497.
 24. Poceta, J. S., R. M. Timms, D. U. Jeong, J. L. Ho, M. K. Erman, and M. M. Mitler. 1992. Maintenance of wakefulness test in obstructive sleep apnea syndrome. *Chest* 101:893-897.
 25. Reitan, R. M. 1958. Validity of the trail making test as an indicator of organic brain damage. *Percept. Mot. Skills* 8:271-276.
 26. Johns, M. W. 1993. Daytime sleepiness, snoring, and obstructive sleep apnea: the Epworth sleepiness scale. *Chest* 103:30-36.
 27. Sériès, F., I. Sériès, and L. Atton. 1993. Comparison of snoring characteristics obtained by polysomnographic studies and home recordings. *Chest* 103:1769-1773.
 28. Rechtschaffen, A., and A. Kales. 1968. A manual of standardized terminology, techniques and snoring system for sleep stages of human subjects. Public Health Service, Washington DC. NIH Publication No. 204.
 29. American Sleep Disorders Association. 1992. EEG arousals: scoring rules and examples. *Sleep* 15:174-183.
 30. Srivastava, M. S., and E. M. Carter. 1983. Repeated measures and profile analysis. In *An Introduction to Applied Multivariate Statistics*. Elsevier Science Publishing Co., Inc., North Holland, New York. 198-230.