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Air Leak Is Associated With Poor Adherence to AutoPAP Therapy

Alexandra Valentin, RRT[1]; Shyam Subramanian, MD[2]; Stuart F. Quan, MD[3,4]; Richard B. Berry, MD[5]; Sairam Parthasarathy, MD[1,4]

[1]Southern Arizona Veterans Administration Healthcare System, Tucson, AZ; [2]Baylor College of Medicine, Houston, TX; [3]Division of Sleep Medicine, Harvard Medical School, Boston, MA; [4]Department of Medicine and Arizona Respiratory Center, University of Arizona, Tucson, AZ; [5]University of Florida, Gainesville, FL

Study Objectives: To our knowledge, a systematic study of the effect of air leak on adherence to auto-titrating positive airway pressure (autoPAP) therapy has not been reported. We hypothesized that in patients with obstructive sleep apnea (OSA), greater levels of air leak were associated with poor adherence to autoPAP therapy.

Design: Retrospective cohort study

Setting: Academic Center

Participants: Ninety-six consecutive patients with high probability for OSA.

Interventions: N/A

Measurements: Patients with OSA received 1 week of autoPAP therapy following which both adherence data and air leak information was downloaded from the device. Continuous positive airway pressure (CPAP) therapy was issued for a 5-week period with pressure determined by 90th percentile of that delivered during autoPAP therapy. Adequate adherence was defined as average usage > 4 h per night on all nights.

Results: Forty-three patients were adherent to autoPAP therapy (350 ± 67[SD] min/day), whereas 53 patients were not (122 ± 65 min/day; $P < 0.0001$). Air leak that was adjusted for pressure delivered was greater in non-adherent patients (7.0 ± 3.5 L/min/cm H_2O) than that in adherent patients (4.9 ± 1.7 L/min/cm H_2O; $P < 0.0001$). Greater residual respiratory events (measured as autoPAP-derived hypopnea index) and proportion of time spent at large leak levels were associated with non-adherence. Patients who were adherent to autoPAP therapy received higher average therapeutic pressures from the autoPAP device than non-adherent patients. Multivariate logistic regression revealed that higher levels of air leak were associated with non-adherence to autoPAP therapy (odds ratio 1.43; 95% CI, 1.03, 1.98; $P = 0.03$). Moreover, adherence to autoPAP therapy was strongly correlated with subsequent adherence to CPAP therapy ($R^2 = 0.74$; $P < 0.0001$).

Conclusion: Air leak was associated with poor adherence to autoPAP therapy. We speculate that air leak could be a potential target for future studies aimed at enhancing adherence to autoPAP therapy.

Keywords: Adherence, obstructive sleep apnea, sleep apnea, continuous positive airway pressure, adherence, adult, compliance, artificial respiration, obesity

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INTRODUCTION

The treatment of obstructive sleep apnea (OSA) with auto-titrating positive airway pressure (autoPAP) devices is gaining popularity, and preliminary results from large clinical trials suggest that autoPAP devices are not inferior to conventional polysomnography-based titration of PAP therapy.[1-7] Considering the expense and wait times associated with conventional polysomnography, health care systems are increasingly embracing home-based autoPAP therapy in lieu of the expensive polysomnography-based titration.[7-9] Despite widespread utilization of autoPAP therapy, poor adherence to PAP therapy—be it continuous positive airway pressure (CPAP) therapy or autoPAP therapy—remains a major concern.[10]

In bench experiments, the introduction of air leak causes deterioration in the performance of autoPAP devices as evidenced by an attenuated pressure response to obstructive events of OSA.[11,12] Moreover, in these bench experiments, such air leaks can cause a pressure drop between the device and the artificial upper airway, leading to suboptimal therapeutic pressure.[11,12] In clinical circumstances, air leaks may be either intentional (leaks introduced by the mask "bleeder" valves that prevent rebreathing) or unintentional leaks due to mouth opening, ill fitting, or displaced masks.[13] Although the effect of air leak on bench performance of autoPAP devices is known, to our knowledge, a systematic study of the effect of air leak on adherence to autoPAP therapy has not been reported.[13] We tested the hypothesis that greater levels of air leak are associated with poor adherence to autoPAP therapy. Knowledge derived from such an association could identify a potentially reversible determinant of non-adherence to autoPAP therapy and define a target for future studies aimed at enhancing adherence to such therapy.

METHODS

Ninety-six patients at the Southern Arizona VA Health Care System with a high probability of OSA received home-based autoPAP therapy for one week, followed by 5 weeks of CPAP therapy set at the 90th percentile of pressure derived from the week of autoPAP therapy.[7]

High probability of OSA was based on a clinical prediction rule that required all 3 of the following inclusion criteria: oxygen desaturation (> 4%) index (ODI) > 5/h measured by overnight pulse oximetry in a home setting, history of witnessed apneas, and an Epworth score > 10. In a separate validation set

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Address correspondence to: Sairam Parthasarathy, MD, Research Service Line, SAVAHCS, Associate Professor of Medicine, University of Arizona, 3601 S. Sixth Avenue, Mail Stop 0-151, Tucson, AZ 85723; Tel: (520) 629-1824; Fax: (520) 629-1801; E-mail: spartha@arc.arizona.edu

of 200 patients, the presence of all 3 of these criteria had a 98% positive predictive value for the presence of OSA, based upon the gold standard (polysomnography-derived apnea-hypopnea index ≥ 5/h).[14] Patients with a prior history of receiving home oxygen, suspicion of central sleep apnea (coexistent heart failure or no history of snoring), and a prior diagnosis of OSA were excluded. This study was approved by the Institutional Review Board of the University of Arizona.

AutoPAP Therapy

AutoPAP therapy was initiated by a trained nurse practitioner at the outpatient clinic setting as part of routine clinical care. During the initial visit, patient education regarding OSA and consequences and potential treatment benefits, mask fitting, and device education were accomplished. AutoPAP therapy (RemStar auto, Respironics, Murrysville, PA) was set a pressure range of 4 to 20 cm H_2O with a heated humidifier for all patients. Following a week of autoPAP therapy, during a second office visit, the device was downloaded for information regarding adherence, pressure delivered, air leak levels, and residual events of OSA. Such machine derived residual events of OSA were not derived from polysomnography, but have been shown to compare favorably to the apnea-hypopnea index (AHI) derived from polysomnography.[15] At the same visit, a CPAP device was dispensed, set at the 90th percentile pressure derived from the autoPAP download.[7] Subsequently, after 5 weeks of CPAP therapy, during a third visit, the CPAP device was again downloaded for adherence information. Adequate adherence during autoPAP therapy was strictly defined as device usage > 4 h/night on all nights during the first week (240 min per day for the entire 7-day treatment period).[16] Alternatively, other definitions were also used for sensitivity analysis: ≥ 4 h/d for 5 days a week (Medicare definition); ≥ 3 h on all day; or ≥ 5 h on all days. Patients who failed to achieve adequate adherence or refused therapy at the end of the 5 weeks of CPAP therapy were referred to a sleep specialist and underwent a conventional (laboratory-based) polysomnography.

Device Download

Adherence levels were measured as time; air leak levels were measured as average or 90th percentile leak levels (liters per minute [L/min]) and were adjusted for pressure levels, because leak levels are proportional to pressure delivered. Adjustment for pressure was performed by simple division by the appropriate pressure variable—90th percentile pressure or average pressure level. Events indicative of OSA were identified as apneas, hypopneas, vibratory snore index (a measure of snoring derived from pressure oscillations), and flow limitation index (episodes of inspiratory flow limitation).

Statistics

Group comparisons of continuous variables were made by unpaired *t*-tests or nonparametric equivalents. Proportions were compared using χ^2 test. Adherence to autoPAP therapy was treated as a binary dependent variable. Simple logistic regression with adherence as a dependent variable and the independent determining variables of interest (air leak levels, residual events, age, gender, race) were performed. Subsequently, multiple logistic regression of the significant determinants ($P < 0.05$) with adherence to autoPAP therapy as the dependent variable were performed. Receiver operating characteristics (ROC) curves for air leak were constructed to determine if air leak levels could be used to discriminate adherent and non-adherent patients. P values < 0.05 were considered significant. All data are shown as mean and standard deviation (SD) or median and interquartile range (IQR). SPSS v12.01 (SPSS Inc., Chicago IL) was used for statistical analysis.

RESULTS

Ninety-six patients (age 59 ± 10 years; 93% men) with a body mass index of 37.6 ± 6 kg/m^2 underwent autoPAP and CPAP therapy. Average Epworth score was 15.0 ± 3.1, and therapeutic CPAP pressure based upon 90th percentile of autoPAP download was 10.3 ± 3.2 cm H_2O. Adherence download of autoPAP therapy at the end of one week was available in all 96 patients. However, the CPAP download at the 5-week timeline was available in only 68 patients: 8 patients were lost to follow-up; 8 patients missed their 5-week return appointment (although they were adherent on subsequent visits); and 12 patients cancelled their 5-week appointment in favor of referral to sleep physician and polysomnography because of persistent symptoms, difficulty using CPAP, or simply upon patient request.

Adherence to AutoPAP therapy

At the end of one week, the adherence to autoPAP therapy for the entire cohort was 224 ± 131 min (range 6-501 min). By definition, the adherent and non-adherent groups had the expected distinct difference in adherence information (Table 1). After 1 week of autoPAP therapy, based on a strict definition of adherence (i.e., ≥ 240 min per day on all days), 43% of the cohort was adherent to autoPAP therapy; whereas based on Medicare criteria of 4 h/day on 5 days a week, 62% were considered adherent to autoPAP therapy.

Therapeutic Pressures

During the week of autoPAP therapy, for the entire cohort, the average pressure delivered by the device was 7.8 ± 2.4 cm H_2O and the 90th percentile pressure was 10.3 ± 3.2 cm H_2O. The therapeutic pressures delivered by the autoPAP device were lower in non-adherent than that in adherent patients (Table 1). Similarly, the 90th percentile pressures derived from 1 week of autoPAP therapy were lower in non-adherent than that in adherent patients (Table 1). However, the severity of sleep disordered breathing measured as ODI was similar in patients who were adherent (median of 23.2, interquartile range [IQR]; 10.6, 38.2) versus those who were non-adherent (median of 24.8, IQR; 9.8, 42.1; P = 0.8) to autoPAP therapy.

Residual Events Detected by AutoPAP Device

During the week of autoPAP therapy, the median residual AHI was 7/h (IQR; 4.3, 12.1) for the entire cohort. The residual apnea index and hypopnea index of adherent patients were lower than those of non-adherent patients (Table 1). The vibratory snore index—a measure of snoring based upon pressure oscillations—was greater in non-adherent than adherent patients (Table 1). The flow limitation index—which requires the device to sense flattening of the inspiratory flow tracing measured by

Table 1—Demographics and autoPAP download information

Variable	Adherent (n = 43)	Non-adherent (n = 53)
Demographics		
Age (years)	59 ± 10	59 ± 10
Gender, n (%)	3 (7%)	3 (6%)
Race* n (%)		
Caucasian	23 (53%)	37 (69%)
African American	4 (9%)	3 (6%)
Hispanic	3 (7%)	7 (13%)
Body mass index (kg/m^2)	38 ± 6	37 ± 6
ODI (events/h)	23.2 (10.6, 38.2)	24.8 (9.8, 42.1)
Epworth score	14.9 ± 3.1	15.1 ± 3.0
Mask interface		
Nasal mask#	32 (74%)	31 (58%)
Nasal pillows	2 (5%)	4 (8%)
Full face mask	9 (21%)	18 (34%)
Device download data§		
Adherence (all days; min)	350 ± 67	122 ± 65**
Adherence (days used; min)	363 ± 65	144 ± 71**
Pressure$_{avg}$ (cm H_2O)	8.4 ± 2.6	7.4 ± 2.1*
Pressure$_{90th}$ (cm H_2O)	11.1 ± 3.3	9.7 ± 2.9*
Apnea index†	1.6 (1.2, 3.7)	3.3 (1.7, 7.1)**
Hypopnea index†	3.0 (1.9, 5.1)	4.3 (2.9, 6.8)**
Vibratory snore index	14 (7, 20)	21 (14, 47)**
Flow limitation index	1.0 (1.0, 1.3)	0.7 (0.4, 1.2)**

n, sample size; kg/m^2, kilogram per meter^2; autoPAP, auto-titrating positive airway pressure device; min, minutes; Pressure$_{avg}$, average pressure delivered by autoPAP; Pressure$_{90}$, 90th percentile pressure delivered by autoPAP; vibratory snore index, pressure oscillations similar to snoring detected by device per hour; flow limitation index, events compatible with inspiratory flow limitation detected by device per hour of use.
*Missing race classification in 19 subjects (2 × 3 χ^2; P = 0.51); §Derived from autoPAP download 7 days after initiation of therapy. †Apneas and hypopneas were derived from autoPAP device download and not by polysomnography. Variables are shown as mean ± SD or median and inter-quartile range. *P < 0.05; **P < 0.01.

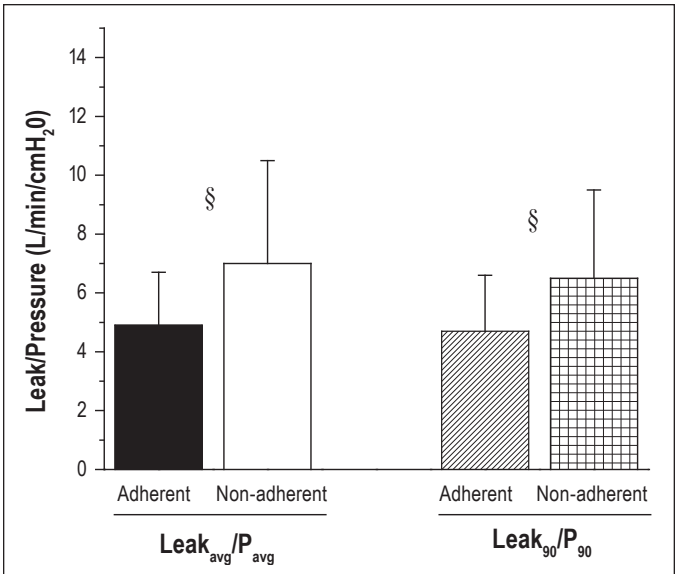

Figure 1—Average air leak levels adjusted for pressure level (Leak$_{avg}$/P$_{avg}$) derived from the auto-titrating positive airway pressure (autoPAP) device downloaded at the end of one week of therapy are shown for adherent (solid column) and non-adherent patients (open columns; *left panel*). On the right panel, 90th percentile air leak levels adjusted for pressure level (Leak$_{90}$/P$_{90}$) derived from the autoPAP device download at the end of one week of therapy are shown for adherent (slope hatched column) and non-adherent patients (cross-hatched column). Average air leak levels adjusted for pressure in adherent patients were lower than those in non-adherent patients (P < 0.0001). Similarly, 90th percentile air leak levels adjusted for pressure were lower in adherent patients than in non-adherent patients (P = 0.001). § P < 0.01; L/min = liters per minute.

the pneumotachograph of the device—was lower in non-adherent than adherent patients (Table 1).

Air Leak Levels

During the week of autoPAP therapy, average leak levels recorded by the device were greater in non-adherent patients (48.7 ± 21.0 L/min) than adherent patients (39.7 ± 16.6 L/min; P = 0.02). Similarly, the 90th percentile air leak levels were greater in non-adherent (67.0 ± 27.5 L/min) than adherent patients (55.3 ± 23.8; P = 0.03). Because levels of air leak are a function of the pressure level, when the average air-leak levels were adjusted for pressure delivered, the adjusted average leak levels in non-adherent patients (7.0 ± 3.5 L/min/cm H_2O) were still greater than those of adherent patients (4.9 ± 1.7 L/min/cm H_2O; P < 0.0001; Figure 1). Similarly, when the 90th percentile air leak levels were adjusted for pressure delivered, the adjusted 90th percentile leak levels in non-adherent patients (6.5 ± 3.0 L/min/cm H_2O) were still greater than those of adherent patients (4.7 ± 1.9 L/min/cm H_2O; P = 0.001; Figure 1). The proportion of time spent in large leak—i.e., time spent in large leak expressed as a percentage of time that the device was used—was greater in non-adherent patients (median 5%; IQR 0.3%, 35.7%) than adherent patients (median 1.2%; IQR 0%, 7%; P = 0.027; Mann-Whitney test).

ROC curves for adjusted average air leak and adjusted 90th percentile leak are shown in Figure 2. The accuracy of air leak levels for predicting non-adherence was fair (ROC area under curve of 0.72 and 0.69). A threshold adjusted leak level of 4.9 L/min/cm H_2O was associated with a sensitivity of 0.62 and specificity of 0.65 for discriminating adherent and non-adherent patients.

Determinants of Non-Adherence to autoPAP Therapy

Univariate logistic regression identified both magnitude of average air leak adjusted for pressure, proportion of time spent with large leak, and residual events of OSA (hypopneas and snoring detected by device) as being associated with non-adherence (dependent variable) (Table 2; P < 0.05). Mask type, race, gender, and age were not associated with non-adherence to autoPAP therapy in our study (Table 2). Because hypopnea index and vibratory snore index (VSI; measure of snoring) were collinear, multiple logistic regressions were performed with the hypopnea index variable, which demonstrated a stronger association with non-adherence than VSI. Multiple regressions identified average leak adjusted for pressure to be associated with non-adherence to autoPAP therapy (Table 2). We performed sensitivity analysis pertaining to our definition of adherence to autoPAP therapy. The final model was analyzed using 3 alter-

nate definitions of adherence: ≥ 4 h per day for 5 days a week (Medicare definition); ≥ 3 h per day on all days; and ≥ 5 h per day on all days. There was no substantial difference in the results among the 4 definitions of adherence (Medicare definition, 3 h, 4 h, or 5 h thresholds; results not shown). Multiple linear regression with minutes of adherence as dependent variable and average leak adjusted for pressure and hypopnea index revealed a significant inverse relationship between adherence and magnitude of air leak (R = −0.23; P = 0.038).

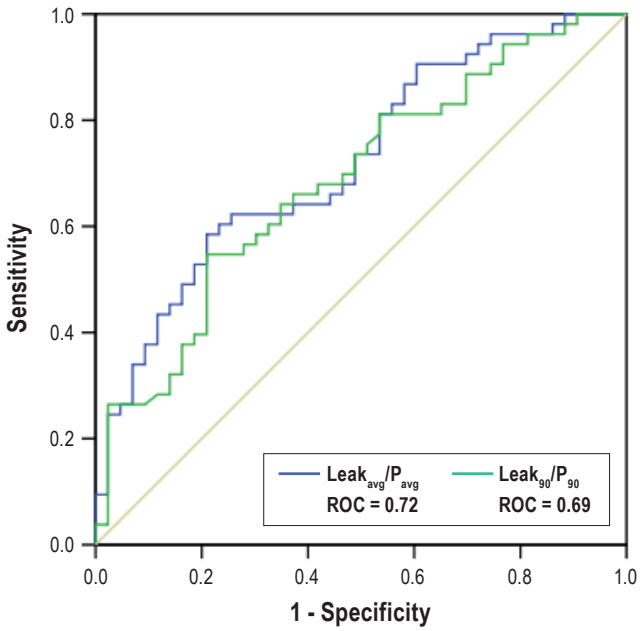

Figure 2—Receiver operating characteristics (ROC) for average leak adjusted for pressure (Leak$_{avg}$/P$_{avg}$) in predicting non-adherence to auto-titrating positive airway pressure (autoPAP) therapy is shown as a blue line. ROC for 90th percentile leak adjusted for pressure (Leak$_{90}$/P$_{90}$) in predicting non-adherence to auto-titrating positive airway pressure (autoPAP) therapy is shown as a green line. ROC areas under the curve for both these measures are shown in the panel on the right side of the figure. The ROC area under the curve for accurately predicting non-adherence (defined as > 240 min per day of usage on all days) was fair.

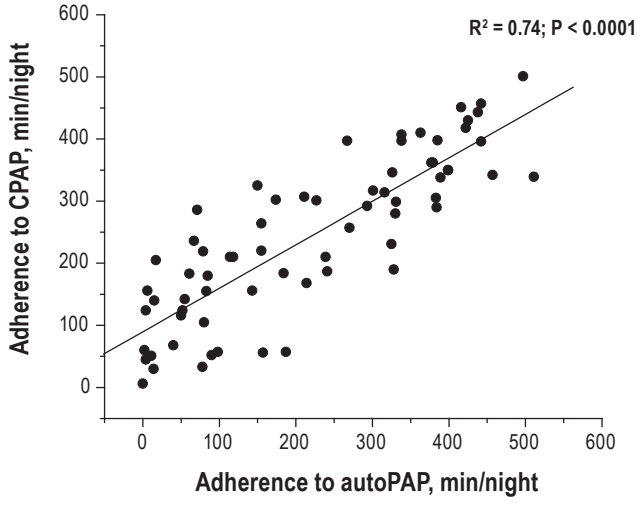

Figure 3—Adherence to autotitrating positive airway pressure (autoPAP) therapy downloaded from autoPAP device after one week of therapy is plotted against adherence to continuous positive airway pressure (CPAP) therapy downloaded from the CPAP device at the end of 5 weeks of CPAP therapy. All adherence information is represented as minutes of use per day on all days of the week. Adherence to autoPAP therapy was closely related to subsequent adherence to CPAP therapy (R^2 = 0.74; P < 0.0001).

Adherence to CPAP Therapy

Adherence to CPAP therapy, during the 5-week period that followed the week of autoPAP therapy was closely related to adherence to autoPAP therapy (R^2 = 0.74; P < 0.0001; n = 68; Figure 3).

DISCUSSION

In this retrospective cohort study, greater air leak levels during autoPAP therapy were associated with poor adherence to such therapy. Moreover, greater levels of air leak during autoPAP therapy were associated with lower mean pressure delivered and higher residual events of OSA. Lastly, adherence to autoPAP therapy was closely related to subsequent adherence to CPAP therapy.

In our study, higher levels of air leak during autoPAP therapy were associated with non-adherence (Table 2). Such an association although implied in day-to-day clinical practice

Table 2—Univariate and multivariate logistic regression of determinants of nonadherence to autoPAP therapy

Variable	OR, 95% CI	P Value
Univariate regressions		
Age	0.9 (0.96, 1.04)	0.9
Gender	1.0 (1.0, 1.0)	1.0
Race (versus Caucasian)		
African American	2.1 (0.4, 10.4)	0.4
Hispanic	0.7 (0.1, 2.9)	0.7
Body mass index	0.9 (0.9, 1.03)	0.9
Epworth score	0.9 (0.9, 1.09)	0.9
Nasal mask#	0.48 (0.19, 1.24)	0.13
Nasal pillows#	1.0 (0.15, 6.5)	1.0
Leak$_{avg}$/P$_{avg}$	1.4 (1.1, 1.8)	0.003*
Log proportion of time at large leak	2.57 (1.28, 5.15)	0.008*
Vibratory snore index	1.01 (1.0, 1.04)	0.045*
Apnea index	1.1 (0.99, 1.2)	0.07
Hypopnea index	1.25 (1.05, 1.49)	0.01*
Multivariate regression (Model R^2 = 0.22; Leak$_{avg}$/P$_{avg}$, log proportion of time spent in large leak, and hypopnea Index)		
Leak$_{avg}$/P$_{avg}$	1.43 (1.03, 1.98)	0.03
Hypopnea index	1.16 (0.96, 1.42)	0.13
Log proportion of time at large leak	0.83 (0.29, 2.40)	0.73

OR, odds ratio; CI, confidence interval; Leak$_{avg}$/P$_{avg}$, average leak adjusted for pressure level downloaded from autoPAP at end of one week of therapy; vibratory snore index, pressure oscillations similar to snoring detected by device per hour; hypopnea and apnea index were derived from autoPAP device. #subjects with nasal pillows and nasal mask were referenced against subjects with full face mask. †Apneas and hypopneas were derived from autoPAP device download and not by polysomnography. *P < 0.05.

has, to our knowledge, not been systematically studied.[13] Several mechanisms may be responsible for this association. For example, mouth opening could lead to large air leaks, which, in turn, can cause drying of the oronasal passageway, eye irritation, increased noise, and consequent intolerance of PAP therapy. Moreover, autoPAP devices may perform suboptimally in the presence of air leak.[11,12] Specifically, in the presence of air leak, the autoPAP device may fail to detect the events of OSA, and thereby either fail to respond or respond in a suboptimal fashion leading to lower levels of delivered (therapeutic) pressures.[12] In a bench study, we demonstrated that the pressure response of an autoPAP device was reduced by 56% in the presence of air leak of 30 L/min.[11] In line with such bench study findings, in the current clinical study, the average pressure delivered was indeed lower in patients who were non-adherent to therapy (and manifested larger leak levels) than adherent patients (Table 1). Such lower levels of therapeutic pressure may have led to the greater number of residual events of OSA as detected by autoPAP device (Table 1). Presence of residual events of OSA may, in turn, have also contributed to the lack of perceived benefit and consequent non-adherence to therapy. In line with such reasoning, a prior study suggested that unresolved air leaks were associated with persistent events of OSA that were verified by polysomnography, but the same study did not explore the relationship of non-adherence and PAP therapy.[17] In our study, the observed differences in residual events were statistically significant, but the effect size was rather small, and residual events did not remain significant after adjusting for leak levels (multiple regression; Table 2). Our findings are based upon associations. Future research on interventions aimed at reducing air leaks—better mask interfaces or devising leak-resistant device algorithms—are warranted.

The type of mask (nasal mask, nasal pillow, or full face), however, was not associated with non-adherence to autoPAP therapy. Conceivably, although the intentional leak levels may be greater with the full-face mask (due to larger bleeder valves), patients with nasal masks may have experience equally large leaks due to mouth opening (unintentional leak). We attempted to identify a threshold level of air leak that discriminated adherent versus non-adherent patients by constructing ROC curves (Figure 2). A well-defined threshold that is adjusted for the pressure level could conceivably guide the sleep technician during manual titration, as currently there is no clearly defined air leak threshold available.[13] However, the identified threshold (4.9 L/min/cm H_2O) was only modest in accurately discriminating adherent versus non-adherent subjects. Such modest discriminating ability of the air leak threshold would suggest that other variables besides mechanical air leak may contribute to treatment non-adherence.[10,18-21]

The tight correlation between adherence to autoPAP and CPAP therapy (Figure 3) also suggests that adherence to PAP therapy was not influenced by the type of device. Such a finding is in agreement with a recent Cochrane database review that concluded that device type (auto, bilevel, or CPAP) and pressure contour modification may not play a significant role in influencing adherence to PAP therapy.[22]

In our study, the severity of OSA (measured as ODI) was similar in patients who were adherent and those who were non-adherent to autoPAP therapy. However, in a recent study by Kohler and colleagues, greater severity of OSA (measured by higher ODI) was associated with better long-term adherence.[23] We suspect that the differential effect of severity of sleep disordered breathing on CPAP adherence reported in these two studies may be due to difference in duration and mode of therapy. Specifically, while we studied short-term adherence (7 days) to autoPAP therapy, the aforementioned study looked at long-term adherence (median 3.9 years) to CPAP therapy.

Limitations

There are limitations to our study. First, OSA was diagnosed by a clinical prediction rule associated with high probability of OSA rather than polysomnography. However, many healthcare systems are adopting such practices to decrease wait times and expenses, and a recent study involved a clinical prediction rule not dissimilar from our study.[8] In that study by Mulgrew and colleagues, a high sleep apnea clinical score, Epworth score > 10, and ODI > 15 per hour was associated with a 95% probability of OSA for their population.[8] In our population, we validated our clinical prediction rule (witnessed apnea, Epworth score > 10, and ODI > 5 per hour) against polysomnography in 200 consecutive patients and reported a positive predictive value of 98%.[14] Moreover, we used autoPAP device-derived apnea, hypopnea, snoring, and flow-limitation detection rather than polysomnography-verified events. Although PSG-verified events would have been preferable, recent studies have indicated that autoPAP estimates of AHIs may be used to estimate residual AHI in patients with OSA of varying severity while being treated with autoPAP.[6,15] Interestingly, unlike the apnea and hypopnea indexes and the vibratory snore index, the flow limitation index was less in non-adherent patients (who manifested greater levels of air leak) than adherent patients. We suspect that the detection of flow-limitation requires a good flow tracing from the device pneumotachograph, and that the presence of air leak may have caused deterioration of the autoPAP device's ability to "sense" such events. However, this is speculation on our part. Lastly, the current study involved only one particular kind of device. As with all other device-related studies, autoPAP devices made by other manufacturers and newer modifications and enhancements to existing devices affect the generalizability of our study findings.[24] Devices made by different manufacturers perform differently and are variably susceptible to the effects of air leak.[11,25]

CONCLUSION

In conclusion, during autoPAP therapy, air leak was associated with poor adherence. Future research in interventions aimed at reducing air leaks—such as better mask interfaces or devising leak-resistant device algorithms—is warranted to determine if this relationship is causal and not an epiphenomenon. Moreover, adherence to autoPAP therapy at one week was strongly correlated with subsequent adherence to CPAP therapy. Treatment paradigms should consider triaging patients with poor adherence to autoPAP therapy after one week to polysomnography rather than awaiting failure of ensuing CPAP therapy.

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