ORIGINAL RESEARCH

The effect of CPAP therapy on blood pressure in the mild obstructive sleep apnea population

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Received: May 24, 2024 **Accepted:** October 16, 2024 **Online Published:** October 22, 2024
DOI: 10.5430/jnep.v15n2p21 **URL:** https://doi.org/10.5430/jnep.v15n2p21

ABSTRACT

This retrospective study uniquely focused on the effects of continuous positive airway pressure (CPAP) on blood pressure in patients with mild obstructive sleep apnea (OSA), a condition characterized by intermittent upper airway collapse. OSA is a well-established risk factor for hypertension, with positive airway pressure (PAP) therapy being the most common treatment. While substantial evidence supports PAP therapy in moderate and severe OSA, the impact of CPAP on mild OSA, which accounts for 50%-70% of all cases, remains underexplored. The primary objective of this study was to investigate whether CPAP therapy, particularly the degree of adherence to treatment, has a measurable impact on blood pressure regulation in patients with mild OSA. Specifically, this research aimed to evaluate whether consistent CPAP use could lower systolic and diastolic blood pressure and assess how varying levels of adherence influence these outcomes. Participants were stratified into two groups based on CPAP adherence: Group A (n = 45) included those who used CPAP for four or more hours per night on at least 70% of nights, while Group B (n = 15) consisted of those with less than four hours of CPAP use per night on 70% of nights. Interestingly, greater systolic and diastolic blood pressure reductions were observed in Group B, suggesting that lower CPAP adherence may still contribute to significant cardiovascular improvements. These findings offer new insights into the management of blood pressure in patients with mild OSA and the role of CPAP adherence.

Key Words: Mild obstructive sleep apnea, Blood pressure, Continuous positive airway pressure

1. INTRODUCTION

Obstructive sleep apnea (OSA), a disorder of increasing recognition, affects 6% to 13% of adults.[1] This alarming prevalence underscores the pressing need to understand and address this condition. OSA is characterized by repeated episodes of collapse or narrowing of the upper airway during sleep, leading to fragmented sleep, blood oxygen desaturations, and sympathetic activation.[2] These respiratory disruptions result in episodes of low oxygen levels, changes in pressure within the chest, and disrupted sleep. These factors reduce quality of life and significantly affect physical and mental health.[3]

OSA is a progressive disease with many cardiovascular effects, one primary effect being hypertension. Previous research indicates a clear association between OSA and hypertension.[4–6] Recent studies suggest that even mild forms of the disease may be correlated with hypertension.[7,8] This study examined the relationship between continuous positive airway pressure (CPAP) therapy and blood pressure in patients with mild OSA.

1.1 Literature review

1.1.1 *Pathophysiology*

OSA is commonly linked to the presence of a narrow and collapsible pharynx as its primary cause. Nonetheless, the size of the upper airway during sleep can also be influenced

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by other elements, thereby heightening the probability of experiencing disordered breathing while asleep.[3] These anatomical and non-anatomical elements culminate in a repeated upper airway collapse. This leads to periodic low oxygen levels in the blood, fluctuations in intrathoracic pressure, disrupted sleep patterns, and subsequent inflammatory response. These are believed to lay the foundation for the negative health effects associated with OSA.[3,9]

The "Great Leap Forward" hypothesis postulates that evolutionary structural changes in the upper respiratory tract predispose humans to obstructive sleep apnea. The hypothesis suggests that the same anatomic features that support the ability to speak are the same features that place the airways in jeopardy during sleep.[3,10] Other factors contributing to this condition include craniofacial abnormalities, advancing age, and obesity.[3]

The contraction of the upper airway muscles ensures that the upper airway remains open during inhalation. Various factors, especially the tone or responsiveness of the upper airway muscles, play a crucial role in determining the critical closing pressure of the pharynx during sleep.[3] At least 20 dilator muscles surround the upper airway, with some playing a crucial role in stabilization and dilation during sleep. The genioglossus muscle is the most critical dilator muscle in the upper airway.[9,11] The genioglossus muscle is an external muscle of the tongue and the most significant among the muscles that expand the pharynx. Its function fluctuates in sync with the breathing process, intensifying during inhalation and diminishing somewhat during exhalation.[12] It has been hypothesized that individuals with OSA have increased genioglossus muscle activation during wakefulness compared to their non-OSA counterparts. The most significant change related to sleep that impacts the respiratory neural drive is the shift from wakefulness to sleep, which reduces the activity of the genioglossus muscle and heightens resistance in the upper airway. During wakefulness, any anatomical shortcomings present in patients with OSA are actively counterbalanced by the upper airway dilator muscles. However, as sleep begins, the activity of the genioglossal muscle decreases more rapidly and to a greater extent in individuals with OSA compared to healthy people, leading to blockage of the upper airway.[12]

The respiratory arousal threshold (AT) is the intensity of breathing effort required to wake someone up at the end of a breathing disturbance. A low AT is associated with the development of OSA and could impact up to one-third of patients with moderate to severe OSA. Those with a low AT often experience shorter breathing disturbances, which hinders the increase of breathing effort needed to reopen the airway during sleep, leading to disrupted sleep.[13]

Several factors can awaken someone from sleep, including respiratory incidents that lead to a drop in oxygen levels and enhance the response of the carotid body.[14] A low arousal threshold is present in 30%–50% of all patients with OSA.[15]

1.1.2 *OSA diagnosis*

Diagnosing OSA is a complex task that often goes unrecognized as a clinical condition. Numerous factors, including limited awareness among patients and providers and organizational and economic barriers, can hinder effective screening.[16] The presence of OSA is frequently initially recognized by the patient's daytime experience (excessive daytime sleepiness) or the nighttime sleep disturbance experienced by the bed partner. Classic nighttime symptoms include nocturnal awakenings, gasping during the night, and loud snoring.[9]

Diagnosis of OSA is based primarily on the number of apnea-hypopnea events per hour an individual has during sleep as measured objectively by an overnight sleep study. An apnea is the cessation of airflow for at least 10 seconds.[17] A hypopnea exists when either there is a 30 percent decrease in airflow from baseline, for a minimum of 10 seconds duration, with at least 3 percent desaturation from baseline or arousal, or there is at least 50 percent decrease in airflow for a minimum of 10 seconds duration with at least a three percent desaturation or arousal.[18] The apnea-hypopnea index (AHI) is based on laboratory findings and is the number of apneas plus hypopneas per hour of documented sleep.

According to the American Academy of Sleep Medicine guidelines, OSA can be diagnosed when the AHI is equal to or greater than 15 or when the AHI is equal to or greater than 5 and accompanied by one or more of the typical associated symptoms of OSA (witnessed apneas, regular snoring, excessive daytime sleepiness, restless sleep, fatigue or insomnia).[19] OSA is further classified as mild, moderate, or severe. The severity of the disease is determined by the respiratory disturbance index (RDI). RDI is the number of apneas, hypopneas, and respiratory effort-related arousals (RERA) per hour of sleep. Mild OSA is determined by an RDI of 5-14, moderate 15-30, and severe >30.[20] Mild OSA is widely prevalent, and it is estimated that 50 to 70% of all OSAS cases are classified as mild.[7] Research suggests that approximately 80%–90% of patients with OSA are undiagnosed due to a lack of awareness among health professionals and the general population.[17]

1.1.3 *OSA and hypertension*

Obstructive sleep apnea (OSA) is a well-established risk factor and cause of hypertension. Although researchers have

previously studied the causal relationship, the specific mechanisms and mediators underlying this association remain uncertain.[21,22] It has been noted that as the apnea-hypopnea index (AHI) rises, so too does the likelihood of developing hypertension.[23] OSA and hypertension appear to have a reciprocal yet bidirectional association, where the presence of one condition elevates the risk of the other.[24,25] While patients may not receive simultaneous diagnoses of both OSA and hypertension, the detection of one condition warrants further investigation into the other.[24]

One of the earliest studies to examine the relationship between OSA and hypertension was Peppard et al.[26] The authors conducted a prospective longitudinal evaluation to examine the association between objective measures of OSA and hypertension (at least 140/90) or the use of antihypertensive medications over a decade ago. The study consisted of 709 participants evaluated at baseline and a four-year follow-up. Of the 709, 184 were followed for an additional four years and reevaluated at eight. The researchers found that persons with few events of apnea or hypopnea (0.1 to 4.9 events per hour) at baseline were 42 percent more likely to have hypertension at follow-up than those with no events of apnea or hypopnea. Persons experiencing 5.0 to 14.9 events of apnea or hypopnea per hour were two times more likely to have hypertension at follow-up, and those with apnea or hypopnea events of 15 or more an hour were three times more likely to have hypertension at follow-up. The authors of this early study suggest that OSA in any form is a risk factor for hypertension. Mild, moderate, and severe OSA are associated with hypertension, underscoring the importance of recognizing even mild OSA as a potential risk factor for hypertension.[27]

1.1.4 *OSA and positive airway pressure*

Positive airway pressure (PAP) therapy is a widely recognized approach for managing obstructive sleep apnea (OSA), providing a steady stream of pressure to keep the airways open during sleep. It is acknowledged as the premier treatment for OSA, with extensive research indicating its effectiveness in decreasing apnea and hypopnea at night and reducing daytime fatigue. The ongoing enhancements in the technology of PAP devices and masks are expected to lead to better patient adherence, overcoming challenges like discomfort and initial negative perceptions.[27]

PAP can be delivered via three modalities: 1) continuous positive airway pressure (CPAP), 2) bilevel positive airway pressure (BiPAP), and 3) automatic self-adjusting positive airway pressure (APAP). CPAP is considered the gold standard treatment for OSA due to being cost-effective, noninvasive, and efficacious.[3] The most significant effect of CPAP on the upper airway is that it prevents the collapse of the oropharynx during sleep by providing continuous pressure to the airway, reduces arterial stiffness and hypertension, and decreases vascular inflammation in those with OSA.[24] However, the effect depends on adherence to therapy, with more significant effects experienced with more hours of nightly use. Typically, adherence is described as utilizing the treatment for a minimum of four hours each night for at least five nights weekly.[9]

The use of CPAP therapy for treating patients with moderate to severe OSA has been validated in the literature as effective in reversing upper airway obstruction during sleep.[28,29] However, the majority (50-70 percent) of individuals with OSA have a milder form of the disease (apnea hypopnea index 5-30).[7] CPAP therapy in persons with mild OSAS has not been widely studied, and additional research is needed in this population.[30]

1.2 Purpose

This study aimed to examine the relationship between continuous positive airway pressure (CPAP) therapy and blood pressure in patients with mild OSA.

2. METHODS

2.1 Design

This study was a retrospective chart review. Institutional Review Board (IRB) approval was obtained for this retrospective chart review through the University of Wisconsin-Milwaukee. A letter of support from the multi-disciplinary medical clinic was obtained. Data were extracted from the EMR and de-identified. No attempt was made to identify study participants.

2.2 Sample selection and setting

Purposive sampling was used to recruit participants from a multi-disciplinary medical clinic specializing in pulmonary diseases and sleep disorders in a state in the southeastern United States. A total of 139 electronic medical records (EMRs) were initially reviewed. The inclusion criterion was a mild obstructive sleep apnea diagnosis as evidenced by an AHI score between 5 and 14, as determined during a complete, supervised, standard polysomnographic study. Participants were excluded if there was no documented blood pressure before CPAP therapy initiation, no documented six-week follow-up blood pressure, no documented data related to CPAP use, or a diagnosis of cognitive impairment of any type, restless legs syndrome, or narcolepsy. After applying these inclusion and exclusion criteria, 79 EMRs were excluded due to incomplete data, leaving 60 EMRs for analysis. Participants were stratified based on CPAP usage: Group A

included those with documented CPAP use for four or more hours per night on 70% of nights (n = 45), and Group B included those with less than four hours of CPAP use per night on 70% of nights (n = 15).

2.3 Measures

The demographic characteristics of age, race, gender, and marital status were used to describe the sample's demographics. Age is a significant variable due to the existing research that suggests that sleep-related difficulties increase with age.[31] Race was recorded as Caucasian, African American, or other since no other races or ethnicities were represented within this patient population. African Americans have a higher prevalence and severity of OSA than other races.[32] Gender was recorded as male or female. The male gender has been associated with an increased risk of OSA.[31] Marital status was recorded as married, divorced, single/never married, widowed, or separated.

AHI scores were recorded for each participant from the polysomnogram prior to CPAP therapy initiation. OSA was categorized as mild, moderate, or severe (mild OSA AHI – 5-14, moderate OSA AHI – 15-30, severe OSA AHI - >30). Additional information was gathered related to smoking and positive airway pressure use. Current smoking status was recorded as "yes" or "no". Positive airway pressure was recorded as continuous positive airway pressure, automatic positive airway pressure, or bilevel positive airway pressure. The machine manufacturer, pressure setting, and supplemental oxygen with the positive airway pressure were also recorded. The machine pressure setting was measured in centimeters of water pressure, and supplemental oxygen use was recorded as "yes" or "no". CPAP use was documented as the number of days included in the most recent report available to the sleep clinic office, the percent of days during the report when CPAP was used greater than or equal to four hours a night, and the percent of days when CPAP was used less than four hours a night. Information related to current medications was gathered from the EMR. Of specific interest to this study were antihypertensive medications. Antihypertensive medication classifications included angiotensin-converting enzyme inhibitors, angiotensin II receptor antagonists, beta-blockers, diuretics, and calcium channel blockers. Many other medications were gathered from the EMR that were not thought to affect blood pressure. These included, but were not limited to, medications used to treat diabetes, gastrointestinal disorders, hyperlipidemia, and vitamins. Diagnoses related to chronic medical conditions were recorded as follows: hypertension, restless legs syndrome, narcolepsy, diabetes mellitus, depression, cardiovascular disease, obstructive sleep apnea syndrome, and other. Data were collected on chronic medical conditions for descriptive purposes and participant exclusion (i.e., narcolepsy, restless legs syndrome). Blood pressure readings for both systolic and diastolic were recorded.

2.4 Data collection

Data were collected on mild OSA patients from electronic medical records (EMR). Blood pressure values were extracted from the EMR for each participant before the initiation of CPAP therapy and at six weeks follow-up, 1-year follow-up, 2 years follow-up, and 3 years follow-up. The follow-up visit time frames were based on clinic protocol. The 3-year follow-up marks the end of the data collection period, which coincides with the clinic's use of the EMR system.

A total of 139 EMRs were reviewed for this study. The initial review revealed that 79 participants had incomplete data, meaning one or more exclusion criteria were met, such as no CPAP use documented, incomplete or missing data related to blood pressure, or a diagnosis of narcolepsy or restless legs syndrome. The final sample size reviewed for this study was 60.

2.5 Data analysis

Data were analyzed using the Statistical Package for the Social Sciences (SPSS) Version 21.0. Descriptive statistics were used to analyze the demographic data obtained. Frequencies were computed for age, race, gender, and marital status. Descriptive statistics described the research participants.

Each of the 60 participants had pre-CPAP and six-week follow-up visits for blood pressure. This study experienced a significant loss of participants due to a lack of complete EMR data. The data available at the one-, two-, and three-year follow-ups were limited and, therefore, were not included in the final analysis. A t-test was conducted to compare the age between the groups. Additionally, t-tests were conducted at baseline for systolic and diastolic blood pressure to determine if significant differences existed between the groups at baseline.

3. RESULTS

The final sample (n = 60) was 56.7 percent male, 43.3 percent female, and 98.3 percent Caucasian (see Table 3). Forty-four participants (73%) were currently taking medication(s) that could be used to treat hypertension, and 96.7 percent had at least one chronic medical condition other than OSA. The three most frequent conditions reported were obesity (55%), hypertension (58.3%), and diabetes mellitus (15%). The mean AHI score was 9.7. CPAP was the only type of positive

airway pressure reported as being used, and 95 percent reported not using supplemental oxygen with the CPAP. Clinic protocol for OSA treatment is a six-week follow-up followed by annual follow-up visits. It is not uncommon for a patient to seek medical treatment for OSA complications outside of the clinic protocol. For the sample above, 9 (15%) sought treatment outside the clinic protocol once, and 2 (3.3%) sought treatment outside the clinic protocol twice. No further exploration of the frequency of follow-up visits outside of clinic protocol was conducted due to limited data.

The demographic characteristics of Groups A and B were similar (see Table 1). The mean age of participants in Group A was 62, and Group B was 59. Both groups were approximately one-half male, predominately Caucasian, married, and non-smokers. A *t*-test was conducted to compare age between the groups and found no statistical difference $t(58) = .770$, $p = .445$ between Group A ($\mu = 62$, SD 11.82) and Group B ($\mu = 59.1$, SD = 14.40). There were 2 (4.4 percent) in Group A and 1 (6.7 percent) in Group B who used supplemental oxygen with their CPAP. The mean age for the two in Group A who received supplemental oxygen with their CPAP was 64 (9.90); they were white, non-smokers, one widowed female, and one married male. The one participant in Group B receiving supplemental oxygen was a 62-year-old white, non-smoking, married female.

Table 1. Demographics, supplemental oxygen use, and CPAP use for both groups

	Group A n = 45	Group B n = 15
Age	62 (11.82)	59.1 (14.40)
Gender		
Male	27 (60)	7 (46.7)
Female	18 (40)	8 (53.3)
Race		
Caucasian American	45 (100)	14 (93.3)
African American		1 (6.7)
Marital Status		
Married	36 (80)	12 (80)
Divorced	2 (4.4)	3 (20)
Single/never married	2 (4.4)	
Widowed	3 (6.7)	
Separated	1 (2.2)	
Smoker		
No	39 (86.7)	14 (93.3)
Yes	6 (13.3)	1 (6.7)
Supplemental oxygen therapy with CPAP		
No	43 (95.6)	14 (93.3)
Yes	2 (4.4)	1 (6.7)

The number of days included in the CPAP use report is highly variable, with a range of 354 days for those who used CPAP four or more hours a night and 104 days for those who used CPAP less than four hours a night (see Table 2). Data on CPAP use included the number of days in the most recent report submitted to the sleep clinic, the percentage of days with usage of four or more hours a night, and the percentage of days with less than four hours a night. The average number of days included in the CPAP use report for Group A was 69.4 (66.90), and the average number of days included for Group B was 53 (34.74). The median number of days included in the CPAP report was 37 (Group A) and 42 (Group B).

Table 2. Days of CPAP use reported

	μ, SD, M
Group A, $n = 45$	69.4 (66.90), 37
Group B, $n = 15$	53 (34.74), 42

Note. Range of days included in CPAP use report for Group A was 11-365. Range of days included in CPAP use report for Group B was 13-117.

In order to examine the groups more closely for blood pressure changes, baseline, and six-week follow-up blood pressure readings were reviewed based on the American Heart Association hypertension guidelines. Fifty-four participants had blood pressure data at baseline and the six-week follow-up visit (see Table 3). Of the 54, eight were normotensive at baseline, four demonstrated no change in blood pressure at six weeks follow-up (Group A, n = 3; Group B, n = 1), and four demonstrated an increase in blood pressure (prehypertension, stage 1, or stage 2 hypertension) (Group A, n = 4). Twenty-four participants were prehypertensive at baseline. Of the 24 with prehypertension, 13 demonstrated no change in blood pressure at the six-week follow-up (Group A, n = 11; Group B, n = 2), three demonstrated a decrease (normotensive) (all Group A), and eight demonstrated an increase (stage 1 hypertension) (Group A, n = 5, Group B, n = 3). Of the 54 participants with blood pressure data at the six-week follow-up, 16 had stage 1 hypertension (Group A, n = 13, Group B, n = 3), and six had stage 2 hypertension (Group A, n = 2, Group B, n = 4) at baseline. Five with stage 1 hypertension demonstrated no change (Group A, n = 4, Group B, n = 1), and 11 demonstrated a decrease (Group A, n = 9, Group B, n = 2). Of those with stage 2 hypertension, one demonstrated no change (Group B), and five demonstrated a decrease (Group A, n = 1, Group B, n = 4). Eighty-five percent of those who had blood pressure data at baseline and six weeks follow-up were either prehypertensive (44 percent) or hypertensive (41 percent) (stage 1 – 11 percent, stage 2 - 30 percent).

Table 3. Effects on blood pressure, n = 54

	Group A, *n* = 41	Group B, *n* =13
No change	*n* = 18 (44)	*n* = 5 (38.5)
Decrease	n = 14 (34)	*n* = 5 (38.5)
Increase	*n* = 9 (22)	*n* = 3 (23)

There was a decrease in systolic blood pressure of 1.1 mmHg for Group A compared to a decrease of 5.8 mmHg for Group B. Decreases in diastolic blood pressure were similar; Group A demonstrated a decrease of 0.4 mmHg while Group B demonstrated a decrease of 4 mmHg. A more significant decrease in mean systolic and diastolic blood pressures was noted for Group B than for Group A (see Table 4).

T-tests were conducted at baseline for systolic blood pressure ($t[57] = 1.200$, $p = .235$) and diastolic blood pressure ($t[53] = 1.133$, $p = .176$) to determine if significant differences existed between the groups at baseline. It was determined that there were no differences between Groups A and B at baseline. As a result, difference scores were calculated on each variable for each group from baseline to 6 weeks follow-up. Independent samples *t*-tests were conducted on the difference scores to examine the effect of CPAP use on systolic and diastolic blood pressure (see Table 5).

Table 4. Blood pressure measurements (mmHg) at six-week follow-up

	Group A, n = 41 $\bar{x}$ (SD)	Group B, n = 13 $\bar{x}$ (SD)
Pre CPAP systolic	130.6 (15.02)	139.5 (15.71)
6-week systolic	129.5 (13.42)	133.7 912.02)
Pre CPAP diastolic	79.3 (9.26)	84.8 (13.03)
6-week diastolic	78.9 (8.91)	80.8 (9.00)

Table 5. Independent samples *t* test results on difference scores for daytime sleepiness, systolic blood pressure, and diastolic blood pressure

	M	*t* (df)	*p*	CI (95%)
ESS	1.5	.865 (34)	.393	-2.049 – 5.084
Systolic blood pressure	4.7	.911 (52)	.367	-5.653 – 15.053
Diastolic blood pressure	3.6	1.00 (52)	.321	-3.617 – 10.837

Note. Mean reported is the mean difference score for each variable.

4. Discussion

This study examined the relationship of CPAP use to blood pressure outcomes in persons with mild obstructive sleep apnea. Although no significant differences were found between CPAP use and blood pressure, these results can still inform future research and practice directions for nursing.

There was a mean decrease in systolic blood pressure of 1.1 mmHg for Group A and a mean decrease of 5.8 mmHg for Group B. Results were similar for diastolic pressure, where Group A decreased an average of 0.4 mmHg, and Group B decreased 4 mmHg. Group B demonstrated greater systolic and diastolic pressure decreases at six weeks of follow-up than Group A, which was an unexpected finding.

Based on the AHA hypertension guideline definitions of hypertension, 44 percent of Group A demonstrated no change in blood pressure, 34 percent demonstrated a decrease in blood pressure, and 22 percent demonstrated an increase in blood pressure. In Group B, 38 percent demonstrated no change in blood pressure, 38 percent demonstrated a decrease in blood pressure, and 23 percent demonstrated an increase in blood pressure. Although not statistically significant, perhaps due to the small sample, those in Group B demonstrated greater systolic and diastolic blood pressure reductions than those in Group A.

It is noteworthy to mention that 83 percent of those with either prehypertension or hypertension at baseline demonstrated no change or a decrease in blood pressure, and 17 percent demonstrated an increase in blood pressure at the six-week follow-up. This finding suggests that CPAP use, at varying doses, may effectively reduce or prevent further progression of hypertension in some OSA patients, but further investigation of this relationship is necessary.

The findings of this study are noteworthy since the primary and widely accepted treatment for OSA is CPAP therapy, and the effect of the therapy is dependent on adherence, with more significant effects experienced with more hours of nightly use. Typically, adherence is described as utilizing the treatment for a minimum of four hours each night for at least five nights weekly.[9] However, this treatment can be challenging for users, and underuse has been reported as problematic.[33] Research indicates that adherence to CPAP treatment varies widely, with estimates ranging from 25% to 83% of patients with OSA not adhering to treatment, depending on the specific criteria used to define adherence.[34,35] This study questions the long-standing adherence guidelines accepted for CPAP usage.

Overall, the findings did not reveal significant differences between those who used CPAP four or more hours a night for 70 percent of reported nights (Group A) and those who used CPAP less than four hours a night for 70 percent of reported nights (Group B) from baseline to six-weeks follow-up. However, paradoxically, Group B demonstrated greater decreases at the 6-week follow-up on both variables than Group A.

4.1 Limitations

The generalizability of this study's findings is limited due to a lack of power resulting from the small sample size. Additionally, many participants were excluded from this study (n = 79) for lack of data availability. The lack of data available in the EMR for data collection likely impacts generalizability. The data collected for the EMR are not done for research purposes. Therefore, a lack of standardization related to medical conditions, laboratory tests, disease staging, etc., can hinder the data collection phase of the research process. Overall, current methods of utilizing EMR systems are not sufficient. The quality of care provided and data utilization for research may be compromised due to incomplete or missing data.

A significant concern for this study was the lack of data related to CPAP use in the EMR. It may be presumed that those who use the CPAP machine likely follow up with their sleep medicine provider and that those who do not use the CPAP machine are less likely to follow up with their sleep medicine provider. With that presumption, it is plausible that those without CPAP uses documented are using their CPAP machine but do not have evidence to support the use in the EMR and, thus, were excluded from this study.

4.2 Future research

Future investigations into mild obstructive sleep apnea (OSA) and CPAP therapy should focus on several important areas to deepen our understanding of this treatment's long-term effectiveness and broader implications. Although CPAP therapy is well-recognized for treating moderate to severe OSA, its long-term advantages for individuals with mild OSA have not been thoroughly examined. Upcoming studies need to assess the lasting effects of CPAP therapy on key health outcomes, especially concerning cardiovascular health. Additionally, age may significantly influence how patients respond to CPAP therapy and their challenges in maintaining adherence over time. Research should explore how various age groups, particularly older adults and adolescents, respond to CPAP treatment for mild OSA. Furthermore, gender-specific factors may affect the success of CPAP therapy in this population. Future studies should investigate whether men and women experience different symptom improvement rates, adherence levels, and long-term health benefits from CPAP. Finally, it is crucial to evaluate the effectiveness of existing screening programs in detecting mild OSA among high-risk groups, including individuals with obesity, cardiovascular issues, or a family history of OSA.

Acknowledgements

Not applicable.

Authors contributions

Dr. Michelle L. Nelson is the sole contributor to this work and approved it for publication.

Funding

Not applicable.

Conflicts of Interest Disclosure

The author declares that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Informed consent

Obtained.

Ethics approval

The Publication Ethics Committee of the Sciedu Press. The journal's policies adhere to the Core Practices established by the Committee on Publication Ethics (COPE).

Provenance and peer review

Not commissioned; externally double-blind peer reviewed.

Data availability statement

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

Data sharing statement

No additional data are available.

REFERENCES

[1] Hendy RM, Hani BM, Mohammed SH. Hematological parameters as predictors of OSA severity. The Egyptian Journal of Bronchology. 2024; 18(3). https://doi.org/10.1186/s43168-024-00318-6

[2] Wijata A, Wydeheft L, Janiak M, et al. The effect of obstructive sleep apnea on the cardiovascular system. Journal of Education, Health and Sport. 2022; 12(6): 39-50. https://doi.org/10.12775/JEHS.2022.12.06.003

[3] Lee JJ, Sundar KM. Evaluation and management of adults with obstructive sleep apnea syndrome. Lung. 2021; 199: 87-101. https://doi.org/10.1007/s00408-021-00426-w

[4] Visseren FL, Mach F, Smulders YM, et al. 2021 ESC guidelines on cardiovascular disease prevention in clinical practice developed by the task force for cardiovascular disease prevention in clinical practice with representatives of the European Society of Cardiology and 12 medical societies with the special contribution of the European Association of Preventive Cardiology (EAPC). European Heart Journal. 2021; 42(34): 3227–3337. https://doi.org/10.1093/eurheartj/ehab484

[5] Xia W, Jing Y, Yuan C. Correlation between Obstructive Sleep Apnea Syndrome (OSAS) and cognitive dysfunction in elderly patients with hypertension. Journal of Integrative Neuroscience. 2023; 22(4). https://doi.org/10.31083/j.jin2204083

[6] Williams LB, Mancia EG, Spiering YHW, et al. 2018 ESC/ESH guidelines for the management of arterial hypertension: The task force for the management of arterial hypertension of the European Society of Cardiology and the European Society of Hypertension: The task force for the management of arterial hypertension of the European Society of Cardiology and the European Society of Hypertension. Journal of Hypertension. 2018; 36(10): 1953–2041. https://doi.org/10.1097/HJH.00000000000019

[7] Bouloukaki I, Grote L, McNicholas WT, et al. Mild obstructive sleep apnea increases hypertension risk, challenging traditional severity classification. Journal of Clinical Sleep Medicine: JCSM: Official Publication of the American Academy of Sleep Medicine. 2020; 16(6): 889–898. https://doi.org/10.5664/jcsm.8354

[8] Vgontzas AN, Li Y, He F, et al. Mild-to-moderate sleep apnea is associated with incident hypertension: age effect. Sleep. 2019; 42(4). https://doi.org/10.1093/sleep/zsy265

[9] Gottlieb DJ, Punjabi NM. Diagnosis and management of obstructive sleep apnea. JAMA. 2020; 323(14): 1389-1400. https://doi.org/10.1001/jama.2020.3514

[10] Davidson TM. The great leap forward: The anatomic basis for the acquisition of and obstructive sleep apnea. Sleep Medicine. 2003; 4(3): 185-194. https://doi.org/10.1016/S1389-9457(02)00237-X

[11] Mediano O, Romero-Peralta S, Resano P, et al. Obstructive sleep apnea: Emerging treatments targeting the genioglossus muscle. Journal of Clinical Medicine. 2019; 8: 1754. https://doi.org/10.3390/jcm8101754

[12] Cori JM, O-Donoghue F, Jordan AS. Sleeping tongue: current perspectives of genioglossus control in healthy individuals and patients with obstructive sleep apnea. Nature and Science of Sleep. 2018; 10: 169-179. https://doi.org/10.2147/NSS.S143296

[13] El-Solh AA, Lawson Y, Wilding GE. Impact of low arousal threshold on treatment of obstructive sleep apnea in patients with posttraumatic stress disorder. Sleep and Breathing. 2021; 25(2): 597-604. https://doi.org/10.1007/s11325-020-02106-0

[14] Kim LJ, Freire C, Fleury Curado T, et al. The role of animal models in developing pharmacotherapy for obstructive sleep apnea. Journal of Clinical Medicine. 2019. https://doi.org/10.3390/jcm8122049

[15] Eckert DJ. Phenotypic approaches to obstructive sleep apnoea - New pathways for targeted therapy. Sleep Medicine Reviews. 2018; 37: 45–59. https://doi.org/10.1016/j.smrv.2016.12.003

[16] Gunta SP, Jakulla RS, Ubaid A, et al. Obstructive sleep apnea and cardiovascular diseases: Sad realities and untold truths regarding care of patients in 2022. Cardiovascular Therapeutics. 2022.

[17] Chang HP, Chen YF, Du JK. Obstructive sleep apnea treatment in adults. Kaohsiung Journal of Medical Sciences. 2020; 36: 7-12.

[18] Won CH, Qin L, Selim B, et al. Varying hypopnea definitions affect obstructive sleep apnea severity classification and association with cardiovascular disease. Journal of Clinical Sleep Medicine. 2018; 14(12). https://doi.org/10.5664/jcsm.7520

[19] American Academy of Sleep Medicine. The International Classification of Sleep Disorders. 3rd ed. Darien, IL: Author. 2014.

[20] Metin M, Avcu M. Is the examination of a single night polysomnography sufficient for a diagnosis of OSAS in adolescent patients? Erciyes Medical Journal. 2021; 43(4): 361–365.

[21] Han B, Chen WZ, Li YC, et al. Sleep and hypertension. Sleep and Breathing. 2020; 24(1): 351-356. https://doi.org/10.1007/s11325-019-01907-2

[22] Yuan FJ, Zhang SS, Liu X, et al. Correlation between obstructive sleep apnea hypopnea syndrome and hypertension: A systematic review and meta-analysis. Annals of Palliative Medicine. 2021; 10(12): 12251-12261. https://doi.org/10.21037/apm-21-3302

[23] Mokros L, Kuczynski W, Franczak L, et al. Morning diastolic blood pressure may be independently associated with severity of obstructive sleep apnea in non-hypertensive patients: A cross-sectional study. Journal of Clinical Sleep Medicine. 2017; 13(7): 905-910. https://doi.org/10.5664/jcsm.6664

[24] Brown J, Yazdi F, Jodari-Karimi M, et al. Obstructive sleep apnea and hypertension: Updates to a critical relationship. Current Hypertension Reports. 2022; 24: 173-184. https://doi.org/10.1007/s11906-022-01181-w

[25] Pallangyo P, Mgopa LR, Mkojera Z, et al. Obstructive sleep apnea and associated factors among hypertensive patients attending a tertiary cardiac center in Tanzania: A comparative cross-sectional study. Sleep Science Practice. 2021; 5(17). https://doi.org/10.1186/s41606-021-00069-z

[26] Peppard PE, Young T, Palta M, et al. Prospective study of the association between sleep-disordered breathing and hypertension. New England Journal of Medicine. 2000; 342(19): 1378-1384. https://doi.org/10.1056/NEJM200005113421901

[27] Patil SP, Ayappa IA, Caples SM, et al. Treatment of adult obstructive sleep apnea with positive airway pressure: An American Academy of Sleep Medicine clinical practice guideline. Journal of Clinical Sleep Medicine. 2019; 15(2): 335-343. https://doi.org/10.5664/jcsm.7640

[28] Baratta F, Pastori D, Fabiani M, et al. Severity of OSAS, CPAP and cardiovascular events: A follow-up study. European Journal of Clinical Investigation. 2018; 48: e12908. https://doi.org/10.1111/eci.12908

[29] Martinez-Garcia MA, Oscullo G, Ponce S, et al. Effect of continuous positive airway pressure in very elderly with moderate-to-severe obstructive sleep apnea pooled results from two multicenter randomized controlled trials. Sleep Medicine. 2022; 89: 71-77. https://doi.org/10.1016/j.sleep.2021.11.009

[30] Chowdhuri S, Quan SF, Almeida F, et al. An official American Thoracic Society research statement: Impact of mild obstructive sleep apnea in adults. American Journal of Respiratory and Critical Care

Medicine. 2016; 193(9): e37-54. https://doi.org/10.1164/rccm.201602-0361ST

[31] Ersoy E, Mercan Y. Risk factors for obstructive sleep apnea and the association of obstructive sleep apnea with daytime sleepiness, obesity and comorbidity. Journal of Turkish Sleep Medicine. 2021; 1: 20-27. https://doi.org/10.4274/jtsm.galenos.2020.02886

[32] Ullah M, Tamanna S. Racial disparity in cardiovascular morbidity and mortality associated with obstructive sleep apnea: The sleep heart health study. Sleep Medicine. 2023; 101: 528-534. https://doi.org/10.1016/j.sleep.2022.12.007

[33] Chaplin H, Ward K. How many hours per night is enough? A systematic review to identify optimal hours of CPAP therapy for sleep apnoea. Health Sciences Review. 2022; 5: 100061. https://doi.org/10.1016/j.hsr.2022.100061

[34] Cistulli PA, Armitstead J, Pepin JL, et al. Short-term CPAP adherence in obstructive sleep apnea: a big data analysis using real world data. Sleep Medicine. 2020; 59: 114-116. https://doi.org/10.1016/j.sleep.2019.01.004

[35] Weaver TE, Grunstein RR. Adherence to continuous positive airway pressure therapy: The challenge to effective treatment. Proceedings of the American Thoracic Society. 2008; 5(2): 173-178. https://doi.org/10.1513/pats.200708-119MG