Efficacy of Continuous Positive Airway Pressure (CPAP) in Patients with Obstructive Sleep Apnea (OSA): A Real-World Study

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Abstract

Background: Sleep being an essential biological function, has a significant role. The basic structure of sleep consists of non-rapid eye movement (NREM) and rapid eye movement (REM). Obstructive sleep apnea-hypopnea syndrome (OSAHS) is the commonest of sleep-related breathing disorders, which is a chronic disorder characterized by the presence of partial or complete blockage of the upper airway causing reduced (hypopnea) or absent (apnea) airflow during sleep. Continuous positive airway pressure (CPAP) therapy reduces OSA severity, improves both daytime and nocturnal symptoms in particular excessive daytime sleepiness and fatigue.

Objective: Assessment of AHI, Arousal Index, and sleep pattern improvement (N1, N2, N3, and R duration), blood pressure pre and post CPAP therapy and compliance of patients to CPAP therapy over a period of ≥6 months

Methodology: A total 107 polysomnography (PSG) reports of patients who visited the sleep centre from March 2019 to March 2020 were included for the analysis.

Results: Our study reported a significant decrease (p<0.0001) in AHI, Arousal Index, and blood pressure post CPAP therapy in the overall population, both the genders and in all age groups. There was a significant increase in N3, and R (p<0.05) post CPAP therapy in most of the patients, whereas there was a marginal decrease in N1 and N2 post CPAP therapy.

Conclusion: Our results from the real-life clinical setting confirms that CPAP is effective at decreasing AHI, Arousal Index, blood pressure, and adherence to treatment with CPAP reported increased sleep efficiency and improvement in both nocturnal and diurnal symptoms in Indian patients with moderate to severe OSA.

Keywords: Continuous positive airway pressure, obstructive sleep apnea, CPAP, OSA, real-world

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Conflicting Interest: Nil.

Introduction

Significant role in learning, memory, emotional regulation, neural development, regulation of cardiovascular and metabolic processes, cellular toxin removal and survival. Good-quality sleep is a major requisite for the maintenance of healthy life.^[1]

The basic structure of sleep consists of non-rapid eye movement (NREM) and rapid eye movement (REM). NREM accounts for 75-80 percent of the total time duration of sleep and REM account for the rest. NREM has three stages: Stage 1, 2 and 3. REM is divided into phasic and tonic phases. This whole pattern is called sleep cycle.^[2, 3]

As per the International Classification of Sleep Disorders (ICSD), sleep disorders are divided into eight major categories. Sleep-related breathing disorders refers to a spectrum of breathing anomalies rang-

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ing from chronic or habitual snoring to Upper Airway Resistance Syndrome (UARS) to frank Obstructive Sleep Apnoea Hypopnoea Syndrome (OSAHS) or in some cases Obesity Hypoventilation Syndrome (OHS). ^[4] Due to late diagnosis and lack of knowledge about the prevalence of sleep-disordered breathing (SDB), there has been a mortality risk associated with SDB. Many studies have related SDB to hypertension, cardiovascular disease, depression, motor vehicle crashes, cognitive impairment, and diminished quality of life.^[5]

Obstructive sleep apnea-hypopnea syndrome (OSAHS) is the commonest of sleep-related breathing disorders which is a chronic disorder characterized by the presence of partial or complete blockage of the upper airway causing reduced (hypopnea) or absent (apnea) airflow during sleep. This is usually followed by arousal. OSAHS has nocturnal (snoring, apneas, choking at night, nocturia, insomnia) and diurnal (excessive sleepiness, memory loss, headaches, depression) symptoms to list a few of them. It is associated with decreased quality of life and significant medical comorbidities. Untreated OSAHS can lead to a host of cardiovascular diseases, including coronary artery disease (CAD), Hypertension, Stroke and Atrial Fibrillation.^[6]

OSAHS is prevalent in middle-aged persons all over the world, with a population prevalence between 2-5%. Indian prevalence rate being a little more in middle-aged Indian men (2.4-4.96 %) than in women which accounts for 1-2 %.^[6,7]

Few prominent risk factors for OSA include male gender, obesity, nasal congestion, hypothyroidism, menopause, and craniofacial and oropharyngeal features such as a large neck circumference.^[6, 7] Persons with a BMI greater than 30, neck size greater than 16 inches in women and 17 inches in men, enlarged tonsils, enlarged tongue and micrognathia may be at a higher risk. Eighty per cent of OSA sufferers report daytime sleepiness which leads to lack of concentration at work, road accidents.^[8, 9] Patients with these symptoms develop cognitive and neurobehavioral dysfunction, mood changes, which can affect the quality of life as well.^[10]

OSA patients experience frequent EEG arousals, defined as abrupt changes in EEG frequency to alpha or theta without spindle activity, which could be micro or actual awakening from sleep. These frequent arousals through sleep fragmentation potentially contribute to increased daytime fatigue which has been substantiated in models of upper airway resistance syndrome. ^[11] The number of arousals per hour is known as the arousal index—higher the arousal index, higher the fa-

tigue to feel.^[12]

OSA has been recognized as a risk factor for cardiovascular diseases, especially hypertension, stroke, arrhythmias and death. Hypoxia leads to hyperactivity of the sympathetic nervous system; increased oxidative stress and endothelial dysfunction; metabolic and hormonal changes, including activation of the renin-angiotensin-aldosterone system, which accompanies the elevation of BP in these patients.^[13,14] Several studies and international guidelines have emphasized OSA as a potentially curable secondary form of hypertension.

OSAHS can be predicted with initial screening by sleep questionnaires such as Stop Bang Questionnaire, Epworth Sleepiness Scale and Berlin Questionnaire, which are commonly applied in clinical practice.^[15]

Apnoea/hypopnoea index (AHI) is calculated by the number of obstructive events (apnoeas or hypopnoeas) per hour of sleep and obtained by nocturnal cardiorespiratory events monitoring. AHI is used to define the severity of OSA.^[6] Table 1 depicts the categorization of OSA severity using AHI.^[16]

Table 1: OSA severity based on AHI

	OSA Severity		
< 5	Normal or primary		
	snoring		
5 -15	Mild		
16-30	Moderate		
AHI >30	Severe		

Earlier OSA was treated by tracheostomies which had many associated complications. To overcome the obstacles and

for significant results, positive airway pressure (PAP) came into existence in the 1980s.^[4, 11] PAP device is set through a nasal or oronasal mask, overnight or during sleep hours at a set positive pressure. Patients with AHI greater than 15 are recommended for PAP therapy, while patients with AHI greater than 05 and below 15 are advised to use PAP only in the presence of symptoms.^[4]

Continuous Positive Airway Pressure (CPAP)

Continuous PAP (CPAP), a gold standard treatment for OSA was invented by Dr Colin Sullivan in 1981.^{[4, ^{17]} American Academy of Sleep Medicine (AASM) has provided guidelines to use CPAP, auto-adjusting PAP (APAP) for the treatment of OSA in adults.^[18] The minimum starting CPAP is 4 cm H₂O, and the maximum is 15 cm H₂O for children and 20 cm H₂O for patients greater than or equal to 12 years.^[19] An evidence-based Indian initiative on Obstructive sleep apnoea (INOSA) and consensus guidelines recommend split-night study PSG with an initial Diagnostic study followed by 03 hours of CPAP titration if AHI >40 in patients.^[20]} CPAP therapy reduces OSA severity, improves both daytime and nocturnal symptoms in particular excessive daytime sleepiness and fatigue. CPAP has the potential to reduce cardiovascular and mortality events.^[18]

A meta-analysis by the American Academy of Sleep Medicine reported substantial improvement in day time sleepiness, quality of life, blood pressure, cardiovascular events in patients with CPAP therapy.^[21]

Furthermore, a clinical study by Gulshan Battan et al., to assess the effect of CPAP therapy on the Epworth sleepiness scale (ESS) in Indian patients with moderate and severe OSA enrolled 47 patients. Out of 47 patients, 24 had moderate, and 23 had severe OSA. ESS in both groups improved significantly after one and three months of CPAP therapy.^[22]

Further clinical trials by Guilleminault C, Miyauchi Y, Gupta A *et al.* reported a statistically significant reduction in AHI in patients with CPAP therapy.^[23-25] Several studies conducted around the world suggests the effectiveness of CPAP in treating OSA.

Since there are a limited number of real-world studies being performed in India therefore, this observational study was carried out to understand the efficacy of CPAP therapy in a real-world population of various ages, genders and comorbidities.

Objectives

The present study was done with the following objectives:

- To determine AHI, Arousal Index, and sleep pattern improvement (N1, N2, N3, and R duration) pre and post CPAP therapy
- To assess the blood pressure improvement pre and post CPAP therapy
- Compliance of patients to CPAP therapy over a period of ≥6 months

Methodology

Study Type: This is an observational, retrospective study based on the polysomnography (PSG) reports from inpatient clinical setting in New Delhi, India. Anonymized and de-identified data were used for analysis. Data de-identified by removing personal identifiers. PSG reports of patients visited the sleep centre from March 2019 to March 2020 were included in the study.

Study Settings: Inpatient (IPD) setting of sleep centre in New Delhi, India

Source of Data: PSG reports of patients with complaints or diagnosis of OSA

Inclusion Criteria:

1. Diagnosis of OSA as per the physician's discretion.

- Adult patients were included >18 years of age
 Exclusion Criteria:
- 1. Patients with age <18 years have been excluded from the study.

Data Collection Process:

- 1. PSG reports based retrospective data was retrieved and included for analysis after assessing for inclusion/exclusion criteria.
- Information on the patient profiles (age, gender, BMI), AHI values, Arousal Index, Sleep pattern (N1, N2, N3 and R duration) pre and post CPAP, comorbidities, blood pressure were collected and analyzed.

Statistical Analysis

Data analysis was done using Microsoft Excel (2016), and R Studio-3.6.2. Descriptive statistics were presented in the form of categorical and continuous variables. Categorical variables (like age, gender) were expressed as percentages and compared by using exact tests (Chi-square/Fisher exact test). Continuous variables (like age, AHI, Arousal Index) were expressed as means and compared by using T statistics Statistical significance was considered at p<0.05.

Ethical Consideration: Confidentiality of patients has been maintained by de-identifying personal information, and only anonymized data from electronic medical records were used for the analysis.

Results

This study assessed the efficacy of Continuous positive pressure therapy (CPAP) in 107 Obstructive sleep apnea (OSA) patients.

The mean age of the sample population was 54.50 years, and 81.30% were male. The majority (50.46%) of the patients were in the Older adults age group (above 55 years). Sixty-four patients (59.81%) were in the obese category (BMI \geq 30). The baseline BMI, AHI, Arousal Index, and Sleep pattern for patients in the study are presented in Table 2.

Assessment of AHI, Arousal Index, and Sleep Pattern Improvement (N1, N2, N3, and R duration) Pre- and Post-CPAP Therapy:

Out of 107 patients, 62 patients had pre and post CPAP AHI values. There was a significant decrease in AHI (p<0.0001) post CPAP therapy in the overall population, both the genders and in all age groups (Table 3-4, Figure 1).

Likewise, out of 107 patients, 36 patients had pre and post CPAP Arousal Index values. There was a significant decrease in Arousal Index (p<0.0001) post

Total number of patients	107
Age (Mean ± SD) (years)	54.50 (13.94)
Age-wise distribution	
Young Adults (18-35 years)	13 (12.14%)
Middle-Aged Adults (36–55 years)	40 (37.38%)
Older Adults (Above 55 years)	54 (50.46%)
Gender	
Male	87 (81.30%)
Female	20 (18.69%)
BMI wise distribution	
Underweight (<18.5)	0 (0%)
Normal (18.5–24.9)	7 (6.54%)
Overweight (25–29.9)	28 (26.16%)
Obese (≥30)	64 (59.81%)
Data not available	8 (7.47%)
Baseline Parameters	Mean (SD)
Weight (kg)	93.77 (20.22)
Height (cm)	169.13 (8.72)
BMI (kg/m ²)	32.60 (6.21)
AHI (events/ hour)	47.98 (32.34)
Arousal Index (events/ hour) (n=36)	52.71 (23.16)
N1 (% TST) (n=78)	7.82 (13.29)
N2 (% TST) (n=78)	60.21 (15.47)
N3 (% TST) (n=78)	16.10 (6.12)
R Duration (% TST) (n=78)	14.63 (9.98)
Systolic Blood Pressure (mm Hg) (n=94)	130.32 (8.97)
Diastolic Blood Pressure (mm Hg) (n=94)	85.53 (7.53)
Mean Duration of CPAP therapy (hours)	6.11 (2.02)
(Mean±SD) (N=65)	

Table 2: Demographic details of the patients

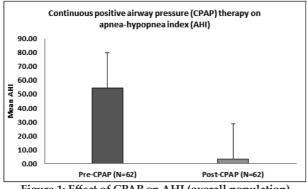
*TST: Total Sleep Time

CPAP therapy in the overall population, both the genders and in all age groups (Table 5-6, Figure 2).

Among the sleep characteristics, sleep efficien-

Table 3: Change in AHI (events/ hour) pre- and post-CPAP (overall population)

AHI (events/ hour)	Mean (SD)	P-value
Pre-CPAP (N=62)	54.31 (31.69)	P < 0.0001
Post-CPAP (N=62)	3.31 (2.88)	P < 0.0001



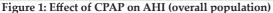


Table 4: Change in AHI (events/ hour) pre- and post-CPAP (gender & age wise)

AHI (events/ hour)	Pre-CPA	P (N=62)	Post-CPAP (N=62)		
inin (events, nour)	Mean	SD	Mean	SD	
Male (n=51)	52.61	30.78	3.02	1.99	
Female (n=11)	62.21	36.12	4.66	5.33	
18-35 (n=6)	49.65	41.36	4.56	3.42	
36–55 (n=26)	52.60	33.09	3.26	3.56	
Above 55 (n=30)	56.73	28.18	3.09	1.89	
P-value	P < 0.0001				

cy improved markedly post CPAP therapy in all the patients. Out of 107 patients, 38 patients were eligible for analysis of pre and post-sleep pattern (N1, N2, N3, AND R) duration values. There was a significant increase in N3, and R (p<0.05) post CPAP therapy in most of the patients, whereas there was a marginal decrease in N1 and N2 post CPAP therapy. (Table 7-9, Figure 3).

Evaluation of the Blood Pressure Improvement Preand Post CPAP therapy:

Out of 107 patients, 94 patients had pre and post CPAP Blood Pressure values. There was a significant decrease in blood pressure (p<0.0001) post CPAP therapy in the overall population, both the genders and in all age groups (Table 10-11).

Compliance of Patients to CPAP Therapy Over ≥Six Months:

Out of 107 patients, 50 patients (46.72%) had data available for follow-up visit over \geq six months. Among

Table 5: Change in Arousal Index (events/ hour) pre- and

post-CPAP (overall population)

Arousal Index (events/ hour)	Mean (SD)	P-value
Pre-CPAP (N=36)	52.71 (23.16)	P < 0.0001
Post-CPAP (N=36)	8.61 (4.57)	

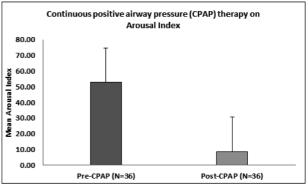


Figure 2: Effect of CPAP on Arousal Index (overall population)

-	0				
Arousal Index	Pre-CPAP (N=36)				Groups
(events/ hour)	Mean	SD	Mean	SD	
Male (n=30)	51.41	21.34	8.27	4.41	Male (n=31)
Female (n=6)	59.16	28.31	10.30	4.54	
18-35 (n=2)	20.85	4.25	9.25	4.55	
36–55 (n=16)	57.21	22.29	7.41	3.37	Female
Above 55 (n=18)	52.24	21.64	9.59	5.08	(n=7)
P-value		P < 0.0001			

Table 6: Change in Arousal Index (events/ hour) pre- and post-CPAP (gender & age wise)

Table 7: Change in sleep pattern pre- and post-CPAP (overall population)

population)							
	Pre-CPAP		Post-0	P-value			
Sleep Pattern	Mean	SD	Mean	SD	, vuide		
Sleep efficiency (%)	79.33	12.79	82.56	12.16	0.158		
N1 duration (% TST)	3.59	3.30	3.32	4.36	0.496		
N2 duration (% TST)	65.04	12.64	57.97	11.42	0.0004		
N3 duration (% TST)	17.23	6.74	20.00	6.18	0.043		
R Duration (% TST)	13.49	7.68	18.57	7.63	0.0004		

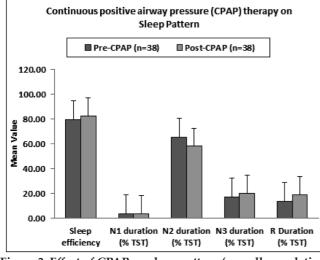


Figure 3: Effect of CPAP on sleep pattern (overall population)

these 50 patients, 31 patients (62.0%) were compliant with CPAP therapy and reported improvement in symptoms.

Age-Specific Prevalence of OSA at Different Scores of AHI Based on PSG Reports:

Amongst 107 patients majority patients (54, 50.46%)

Table 8: Change in sleep pattern pr	re- and post-CPAP (gender-wise)
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_	Sleep Pattern	Pre-CPAP (n=38)		Post-CP.	P-value		
•	Sleep Fattern	Mean	SD	Mean	SD	r-value	
	Sleep efficiency (%)	81.46	10.77	83.25	11.37	0.411	
	N1 duration (% TST)	3.48	2.80	2.85	2.77	0.095	
	N2 duration (% TST)	66.75	10.60	59.49	7.52	0.004	
	N3 duration (% TST)	17.16	5.31	19.79	5.73	0.104	
	R Duration (% TST)	12.21	6.04	17.72	4.56	0.001	
	Sleep efficiency	69.86	16.02	79.46	14.69	0.268	
	N1 duration (% TST)	4.04	4.85	5.40	7.88	0.319	
2	N2 duration (% TST)	57.44	17.09	51.26	19.79	0.155	
	N3 duration (% TST)	17.53	10.91	20.94	7.71	0.289	
	R Duration (% TST)	19.19	10.75	22.34	14.18	0.531	

Table 9: Change in sleep pattern pre- and post-CPAP (age-wise)

Table 9: Change in sleep pattern pre- and post-CFAF (age-wise)							
Groups	Sleep Pattern	Pre-CPAI	? (n=38)	Post-CP.	P-value		
Groups	Sheep Function	Mean	SD	Mean	SD		
	Sleep efficiency	54.40	0.00	86.70	0.00		
	N1 duration (% TST)	5.40	0.00	2.30	0.00		
18-35 (n=1)	N2 duration (% TST)	70.70	0.00	45.10	0.00		
	N3 duration (% TST)	9.00	0.00	25.50	0.00		
	R Duration (% TST)	15.00	0.00	27.10	0.00		
	Sleep efficiency	81.95	15.43	85.55	9.87	0.363	
	N1 duration (% TST)	4.05	4.32	4.48	6.24	0.554	
36–55 (n=17)	N2 duration (% TST)	62.82	18.02	57.53	14.58	0.146	
	N3 duration (% TST)	16.12	7.69	19.58	5.63	0.115	
	R Duration (% TST)	15.46	10.83	18.16	10.46	0.360	
	Sleep efficiency	78.35	8.84	79.80	13.75	0.571	
	N1 duration (% TST)	3.11	2.20	2.39	1.42	0.096	
Above 55 (n=20)	N2 duration (% TST)	66.64	5.36	59.00	8.06	0.003	
	N3 duration (% TST)	18.59	5.67	20.09	6.77	0.418	
	R Duration (% TST)	11.75	3.04	18.50	4.16	P<0.0001	

were above 55 years old. Older adults (above 55 years) have a higher prevalence (46.73% with AHI≥5) than Young adults age group (18-35 years with AHI≥5).

Gender-Specific Prevalence of OSA at Different Scores of AHI Based on PSG Reports:

Amongst 107 patients majority patients (87, 81.30%)

Systolic BP (mm Hg)	Mean (SD)	P-value	
Pre-CPAP (N=94)	130.32 (8.97)	P < 0.0001	
Post-CPAP (N=94)	126.13 (6.05)	r < 0.0001	
Diastolic BP (mm Hg)	Mean (SD)	P-value	
Diastolic BP (mm Hg) Pre-CPAP (N=94)	Mean (SD) 85.53 (7.53)	P-value	

Table 10: Change in blood pressure (systolic and diastolic) pre- and post-CPAP (overall population)

were males. Males have a higher prevalence (75.70% with AHI≥5) of OSA in comparison to females (18.69% with AHI≥5).

Existence of Comorbidities with Obstructive Sleep Apnea:

Amongst 107 patients, the most prev-

alent comorbidity associated with OSA was hypertension (81.82%), followed by obesity (38.18%), and diabetes mellitus (30.91%) (Figure 4).

Discussion

Assessment of AHI, Arousal Index, and Sleep Pattern Improvement (N1, N2, N3, and R duration) Pre- and Post-CPAP Therapy:

Our study reported a significant decrease in AHI (p<0.0001) for 62 patients who had pre and post CPAP AHI values.

A retrospective study by Aaron B Holley et al. en-

Table 11: Change in blood pressure (systolic and diastolic) pre- and post-CPAP (gender & age wise)

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		PAP BP	Post-CPAP BP (N=94)		
Systolic BP (mm Hg)	(IN=	(N=94)		=94)	
	Mean SD		Mean	SD	
Male (n=78)	130.00	9.26	125.72	6.12	
Female (n=16)	131.88	7.50	128.13	5.44	
18-35 (n=13)	126.15	126.15 7.38		4.99	
36–55 (n=33)	131.21	10.66	125.15	7.02	
Above 55 (n=48)	130.83	7.59	127.21	5.29	
P-value	P < 0.0001				

Diastolic BP (mm Hg)	PRE CPAP BP (N=94)		Post-CPAP BP (N=94)	
_	Mean	SD	Mean	SD
Male (n=78)	85.83	6.71	83.10	5.87
Female (n=16)	84.06	10.83	81.25	8.06
18-35 (n=13)	82.31	4.21	82.31	4.21
36–55 (n=33)	86.36	8.81	82.73	6.64
Above 55 (n=48)	85.83	6.95	82.96	6.43
P-value	P < 0.0001			

Table 12: Age-specific prevalence rates of OSA (95% confidence interval) at different scores of AHI based on polysomnography

Age, years	Prevalence Rate, % (95% CI)				
	AHI≥5	AHI ≥ 10	AHI ≥ 15		
18-35	11.01 (6.41-18.26)	8.41 (4.49-15.22)	6.54 (3.20-12.89)		
36–55	36.45 (27.95-45.89)	35.51 (27.09-44.94)	30.84 (22.88-40.13)		
Above 55	46.73 (37.55-56.13)	45.79 (36.66-55.22)	42.99 ((34.01-52.45)		
Total	94.39 (88.30-97.40)	89.72 (82.52-94.16)	80.37 (71.85-86.79)		

 Table 13: Gender-specific prevalence rates of OSA (95% confidence interval) at different scores of AHI based on polysomnography

Condor	Patients	Prevalence Rate, % (95% CI)			
Gender	(n = 107) n (%)	AHI ≥ 5	AHI ≥ 10	AHI ≥ 15	
Male	87 (81.30)	75.70 (66.78-82.84)	71.96 (62.80 - 79.60)	64.49 (55.06 - 72.91)	
Female	20 (18.69)	18.69 (12.44 - 27.11)	17.76 (11.67-26.08)	15.89(10.16-23.98)	

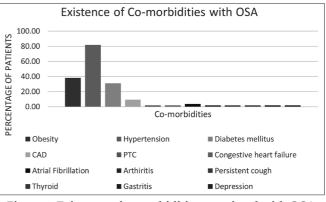
rolled 497 patients and concluded 70.1% of all patients achieved an AHI < 5 using CPAP.[26] Similarly, a systematic review and meta-analysis concluded significant improvement in AHI post CPAP therapy.^[27]

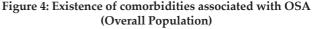
In our study, 36 patients who had pre and post CPAP Arousal Index values reported a significant decrease in Arousal Index (p<0.0001) post CPAP therapy.

A systematic review and meta-analysis to compare CPAP with other oral appliances in patients with OSA concluded significant improvement (p = 0.001) in arousal index post CPAP therapy.^[28]

A network meta-analysis also concluded CPAP as a most efficacious treatment for OSA patients and significantly reduced arousal index.^[29] A study by Ryo Tachikawa *et al.* 29 patients reported mean baseline arousal index (events/h) 34.0 and a decrease (19.4) in arousal index was observed.^[30]

Our study reported a significant increase in sleep efficiency, N3, and R duration (p<0.05) post CPAP therapy for 38 patients who had pre and post-sleep pat-





tern (N1, N2, N3, AND R) duration values available for analysis.

A retrospective study of 45 patients with OSA, the mean age of patients was 52.7 ± 5.6 years. The study reported a significant decrease (P < 0.05) in non-rapid eye movement (NREM) sleep after CPAP therapy.^[31]

The results of AHI, arousal index and sleep pattern reported in our study was in similar trends to clinical trials and retrospective studies discussed above. Factors like early detection of symptoms of OSA and initiation of CPAP therapy could be the contributory reasons to decrease in AHI, arousal index and sleep pattern values.

Evaluation of the Blood Pressure Improvement Preand Post CPAP Therapy:

In our study, 94 patients had pre and post CPAP Blood Pressure values. There was a significant decrease in blood pressure (p<0.0001) post CPAP therapy.

Few systematic review and meta-analysis reported CPAP significantly reduce blood pressure in patients with OSA.^[32-33]

Our results were in concordance with the findings of other clinical studies.

Age and Gender-Specific Prevalence of OSA at Different Scores of AHI Based on PSG Reports:

In our study, older adults (above 55 years) and males have a higher prevalence (46.73% and 75.70% with AHI \geq 5) than young adults age group and females.

A population-based study to assess the prevalence and clinical features of OSA enrolled 305 patients to undergo polysomnography. The study reported 52.1% of males of age group 60-70 years with $AHI \ge 5$.^[34]

Another prevalence study in middle-aged urban Indian men, concluded 19.4% of males of age group 55–65 years with AHI≥5.^[35]

Existence of Comorbidities with Obstructive Sleep Apnea:

In our study, the most prevalent comorbidity associated with OSA was hypertension (81.82%), followed by obesity (38.18%), and diabetes mellitus (30.91%).

Similar findings were reported in the PREDICT trial, enrolled 278 patients, randomized in 1:1 ratio to receive CPAP (n=140) and best supportive care (BSC). The study reported 70% of patients in the CPAP group had hypertension, followed by diabetes.^[36]

Study Strengths and Limitations

Though various clinical studies highlighted the efficacy of CPAP, still there was a dire need for realworld studies to assess the efficacy of CPAP in patients with OSA. This is one of the few real-world studies in Indian OSA patients. The data presented in our study provides useful real-world evidence on the efficacy of CPAP. There are few inherent limitations in the study because of its retrospective observational nature.

Conclusion

Our results from the real-life clinical setting support findings from previously conducted clinical trials and other observational studies and confirms that CPAP is effective at decreasing AHI, Arousal Index, blood pressure and adherence to treatment with CPAP reported increased sleep efficiency and improvement in both nocturnal and diurnal symptoms in Indian patients with moderate to severe OSA.

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References:

- 1. Mukherjee S, Patel SR, Kales SN, Ayas NT, Strohl KP, Gozal D, *et al.* An official American Thoracic Society statement: the importance of healthy sleep. Recommendations and future priorities. *Am J Respir Crit Care Med.* 2015 Jun 15;191(12):1450-8.
- Colten HR, Altevogt BM. Sleep physiology. In: Sleep disorders and sleep deprivation: An unmet public health problem 2006. *Washington DC*; 2006. p. 34-35.
- 3. Carley DW, Farabi SS. Physiology of sleep. *Diabetes Spectr.* 2016 Feb 1;29(1):5-9.
- 4. Sateia MJ. International classification of sleep disorders. *Chest.* 2014 Nov 1;146(5):1387-94.
- 5. Young T, Finn L, Peppard PE, Szklo-Coxe M, Austin D, Nieto FJ, *et al.* Sleep disordered breathing and mortality: eighteenyear follow-up of the Wisconsin sleep cohort. *Sleep. 2008* Aug 1;31(8):1071-8.
- Spicuzza L, Caruso D, Di Maria G. Obstructive sleep apnoea syndrome and its management. *Ther Adv Chronic Dis.* 2015 Sep;6(5):273-85.
- 7. Joshi JM. Obstructive sleep apnea and its prevalence in India. *RespiMirror*. 2015 Apr 1;5(1):1-2.
- 8. Jordan AS, McSharry DG, Malhotra A. Adult obstructive sleep apnoea. *Lancet*. 2014 Feb 22;383(9918):736-47.
- Guilleminault C, Quo SD. Sleep-disordered breathing. A view at the beginning of the new Millennium. *Dent Clin North Am.* 2001 Oct;45(4):643-56.
- 10. Yue HJ, Bardwell W, Ancoli-Israel S, Loredo JS, Dimsdale JE. Arousal frequency is associated with increased fatigue in obstructive sleep apnea. *Sleep Breath*. 2009 Nov 1;13(4):331-9.
- 11. Vaessen TJ, Overeem S, Sitskoorn MM. Cognitive complaints in obstructive sleep apnea. *Sleep Med Rev.* 2015 Feb 1;19:51-8.

- 12. Parati G, Lombardi C, Hedner J, Bonsignore MR, Grote L, Tkacova R, et al. Position paper on the management of patients with obstructive sleep apnea and hypertension: joint recommendations by the European Society of Hypertension, by the European Respiratory Society and by the members of European COST (COoperation in Scientific and Technological research) ACTION B26 on obstructive sleep apnea. J hypertens. 2012 Apr 1;30(4):633-46.
- Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo Jr JL, *et al.* The seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure: the JNC 7 report. *JAMA*. 2003 May 21;289(19):2560-71.
- 14. Mancia G, Fagard R, Narkiewicz K, Redón J, Zanchetti A, Böhm M, et al. Task Force Members. 2013 ESH/ESC Guidelines for the management of arterial hypertension: the Task Force for the management of arterial hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). J hypertens. 2013 Jul;31(7):1281-357.
- 15. Modi M. The diagnosis of OSA. *RespiMirror*. 2015 Apr 1;5,(1):6-7.
- 16. Berry RB, Budhiraja R, Gottlieb DJ, Gozal D, Iber C, Kapur VK, *et al.* Rules for scoring respiratory events in sleep: update of the 2007 AASM manual for the scoring of sleep and associated events: deliberations of the sleep apnea definitions task force of the American Academy of Sleep Medicine. *J Clin Sleep Med.* 2012 Oct 15;8(5):597-619.
- Shevade M. Positive airway pressure in obstructive sleep apnea. *RespiMirror*. 2015 Apr 1;5,(1):11-12.
- Patil SP, Ayappa IA, Caples SM, Kimoff RJ, Patel SR, Harrod CG. Treatment of adult obstructive sleep apnea with positive airway pressure: an American Academy of Sleep Medicine clinical practice guideline. *J Clin Sleep Med.* 2019 Feb 15;15(2):335.
- 19. Weiss P, Kryger M. Positive airway pressure therapy for obstructive sleep apnea. *Otolaryngol Clin North Am.* 2016 Dec 1;49(6):1331-41.
- 20. Sharma SK, Katoch VM, Mohan A, Kadhiravan T, Elavarasi A, Ragesh R, *et al.* Consensus and evidence-based Indian initiative on obstructive sleep apnea guidelines 2014. *Lung India.* 2015 Jul;32(4):422.
- 21. Patil SP, Ayappa IA, Caples SM, Kimoff RJ, Patel SR, Harrod CG. Treatment of adult obstructive sleep apnea with positive airway pressure: an American Academy of Sleep Medicine systematic review, meta-analysis, and GRADE assessment. *J Clin Sleep Med.* 2019 Feb 15;15(2):301.
- 22. Battan G, Kumar S, Panwar A, Atam V, Kumar P, Gangwar A, et al. Effect of CPAP therapy in improving daytime sleepiness in Indian patients with moderate and severe OSA. *J Clin Diagn Res. 2016* Nov;10(11):OC14.
- 23. Guilleminault C, Lin CM, Goncalves MA, Ramos E. A prospective study of nocturia and the quality of life of elderly patients with obstructive sleep apnea or sleep onset insomnia. J Psychosom Res. 2004 May 1;56(5):511-5.
- 24. Miyauchi Y, Okazoe H, Okujyo M, Inada F, Kakehi T, Kikuchi H, et al. Effect of the continuous positive airway

pressure on the nocturnal urine volume or night-time frequency in patients with obstructive sleep apnea syndrome. Urology. 2015 Feb 1;85(2):333-6.

- 25. Gupta A, Shukla G, Afsar M, Poornima S, Pandey RM, Goyal V, *et al*. Role of positive airway pressure therapy for obstructive sleep apnea in patients with stroke: a randomized controlled trial. *J Clin Sleep Med*. 2018 Apr 15;14(4):511-21.
- 26. Holley AB, Lettieri CJ, Shah AA. Efficacy of an adjustable oral appliance and comparison with continuous positive airway pressure for the treatment of obstructive sleep apnea syndrome. *Chest.* 2011 Dec 1;140(6):1511-6.
- 27. Schwartz M, Acosta L, Hung YL, Padilla M, Enciso R. Effects of CPAP and mandibular advancement device treatment in obstructive sleep apnea patients: a systematic review and meta-analysis. *Sleep Breath.* 2018 Sep 1;22(3):555-68.
- 28. Li W, Xiao L, Hu J. The comparison of CPAP and oral appliances in treatment of patients with OSA: a systematic review and meta-analysis. *Respir Care.* 2013 Jul 1;58(7):1184-95.
- 29. Liu T, Li W, Zhou H, Wang Z. Verifying the relative efficacy between continuous positive airway pressure therapy and its alternatives for obstructive sleep apnea: a network metaanalysis. *Front Neurol.* 2017 Jun 28;8:289.
- Tachikawa R, Minami T, Matsumoto T, Murase K, Tanizawa K, Inouchi M, *et al.* Changes in habitual sleep duration after continuous positive airway pressure for obstructive sleep apnea. *Ann Am Thorac Soc.* 2017 Jun;14(6):986-93.
- Zhang C, Lv J, Zhou J, Su L, Feng L, Ma J, et al. The effect of CPAP treatment on EEG of OSAS patients. *Sleep Breath*. 2015 Dec 1;19(4):1121-4.
- 32. Lei Q, Lv Y, Li K, Ma L, Du G, Xiang Y, et al. Effects of continuous positive airway pressure on blood pressure in patients with resistant hypertension and obstructive sleep apnea: a systematic review and meta-analysis of six randomized controlled trials. J Bras Pneumol. 2017 Sep;43(5):373-9.
- 33. Fava C, Dorigoni S, Dalle Vedove F, Danese E, Montagnana M, Guidi GC, *et al*. Effect of CPAP on blood pressure in patients with OSA/hypopnea: a systematic review and metaanalysis. *Chest. 2014* Apr 1;145(4):762-71.
- 34. Durán J, Esnaola S, Rubio R, Iztueta Á. Obstructive sleep apnea–hypopnea and related clinical features in a populationbased sample of subjects aged 30 to 70 yr. *Am J Respir Crit Care Med.* 2001 Mar 1;163(3):685-9.
- 35. Udwadia ZF, Doshi AV, Lonkar SG, Singh CI. Prevalence of sleep-disordered breathing and sleep apnea in middleaged urban Indian men. *Am J Respir Crit Care Med.* 2004 Jan 15;169(2):168-73.
- 36. McMillan A, Bratton DJ, Faria R, Laskawiec-Szkonter M, Griffin S, Davies RJ, *et al*, PREDICT Investigators. Continuous positive airway pressure in older people with obstructive sleep apnoea syndrome (PREDICT): a 12-month, multicentre, randomised trial. *Lancet Respir Med. 2014* Oct 1;2(10):804-12.

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