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Impact of PAP titration on sleep architecture and respiratory parameters in obstructive sleep apnea

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ABSTRACT

Aim: To investigate the acute effects of PAP titration on respiratory disturbances, nocturnal oxygenation, and sleep stage organization by comparing diagnostic and titration studies.

Methods: Consecutive adults who completed overnight diagnostic PSG followed by attended PAP titration were eligible. Within-patient comparisons included respiratory indices, oxygenation, sleep stages, sleep continuity, arousal burden, snoring, and total sleep time. Additional paired subgroup analyses were performed according to baseline OSA severity and body mass index (BMI).

Results: A total of 266 paired sleep studies were analyzed. Mean age was 51.56 ± 14.04 years; 80.1% were men and 19.9% women. AHI decreased from 45.79 ± 23.14 to 4.03 ± 6.24 events/h, AI from 34.40 ± 22.54 to 1.90 ± 4.09 events/h, and ODI from 43.32 ± 23.33 to 3.39 ± 5.53 events/h (all $p < 0.001$). Minimum oxygen saturation increased from 74.06 ± 11.59% to 85.50 ± 7.42% ($p < 0.001$). REM sleep increased from 11.32 ± 8.90% to 18.13 ± 9.06% and N3 sleep from 6.23 ± 7.84% to 11.57 ± 10.73% (both $p < 0.001$), with reductions in snoring and arousal index. Total sleep time and PLM index did not change significantly. Respiratory indices improved across all OSA severity and BMI categories, while sleep-stage reorganization was most pronounced in severe OSA and obesity.

Conclusions: PAP titration during the first treatment night produced multidimensional physiological improvement beyond respiratory-event suppression. Early response patterns varied by OSA severity and BMI, suggesting that respiratory stabilization and sleep architecture recovery are related but not interchangeable outcomes and supporting assessment beyond residual AHI alone.

Keywords: Obstructive sleep apnea, polysomnography, positive airway pressure, PAP titration.

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1. Introduction

Obstructive sleep apnea (OSA) is a highly prevalent sleep-related breathing disorder in which recurrent upper airway obstruction causes intermittent hypoxemia, repeated arousals, and disruption of normal sleep organization. These disturbances are linked to daytime sleepiness, cognitive impairment, poor quality of life, and increased cardiovascular, metabolic, and mortality risks [1-4]. Therefore, effective recognition and treatment remain the central goals in contemporary sleep medicine.

Overnight polysomnography (PSG) provides a detailed assessment of sleep and breathing and

remains the reference standard for the diagnosis of OSA [2, 3, 5].

Although the apnea-hypopnea index (AHI) is widely used to grade disease severity and guide management, event frequency alone does not capture the full physiological burden of OSA or the breadth of treatment response [3,6]. Measures of oxygen desaturation, arousal frequency, sleep stage distribution, and sleep continuity can provide clinically relevant information beyond the AHI [5, 6]. PAP, most delivered as CPAP, is the standard treatment for OSA, particularly in moderate-to-severe disease [6].

Maintaining upper airway patency reduces obstructive events and hypoxemia and can lessen sleep fragmentation, allowing recovery of slow-wave (N3) and rapid eye movement (REM) sleep. These changes are thought to contribute to the long-term cardiovascular, metabolic, neurocognitive, and quality-of-life benefits associated with effective PAP treatment [7, 8].

Most evidence on PAP has focused on sustained treatment outcomes, whereas the physiological changes that occur during the first titration night have received less comprehensive evaluation. Few studies have simultaneously examined respiratory control, oxygenation, arousal burden, sleep continuity, snoring, and sleep architecture using paired measurements from the same individual [9-12]. A within-subject comparison is well-suited to this question because it limits the influence of between-person variability.

Accordingly, we compared diagnostic PSG with the first attended PAP titration study to characterize the immediate physiological responses to treatment. Respiratory indices were assessed together with nocturnal oxygenation, sleep continuity, sleep stage distribution, arousals, and snoring. We also explored whether these early changes differed across OSA severity and body mass index (BMI) categories, thereby extending the evaluation beyond residual AHI alone.

2. Materials and methods

2.1. Study Design and Participants: This retrospective observational study was conducted at the Sleep Disorders Center of Hisar Intercontinental Hospital in Istanbul, Türkiye. Adults aged ≥ 18 years who underwent overnight diagnostic PSG followed by manual PAP titration between January 2016 and June 2024 were screened.

Inclusion criteria required usable recordings from both nights. Studies were excluded if central sleep apnea was present, sleep recording time was insufficient, technical quality was inadequate, or polysomnographic data were incomplete.

The final cohort comprised 266 paired PSGs. Because diagnostic and titration measurements were obtained from the same individuals, each participant served as their own comparator, reducing the effect of interindividual variability on treatment-related changes (Figure 1). All studies were performed as attended overnight polysomnography in a sleep laboratory under AASM-based procedures. (Natus Embla SDx with RemLogic software version 4). Monitoring included electroencephalography, electrooculography, submental and bilateral tibial electromyography, electrocardiography, nasal airflow, thoracoabdominal effort, pulse oximetry, body position, and snoring assessment.

Experienced sleep technologists manually scored the sleep stages, respiratory events, desaturation, and arousals using the applicable American Academy of Sleep Medicine (AASM) criteria. AHI was calculated as the

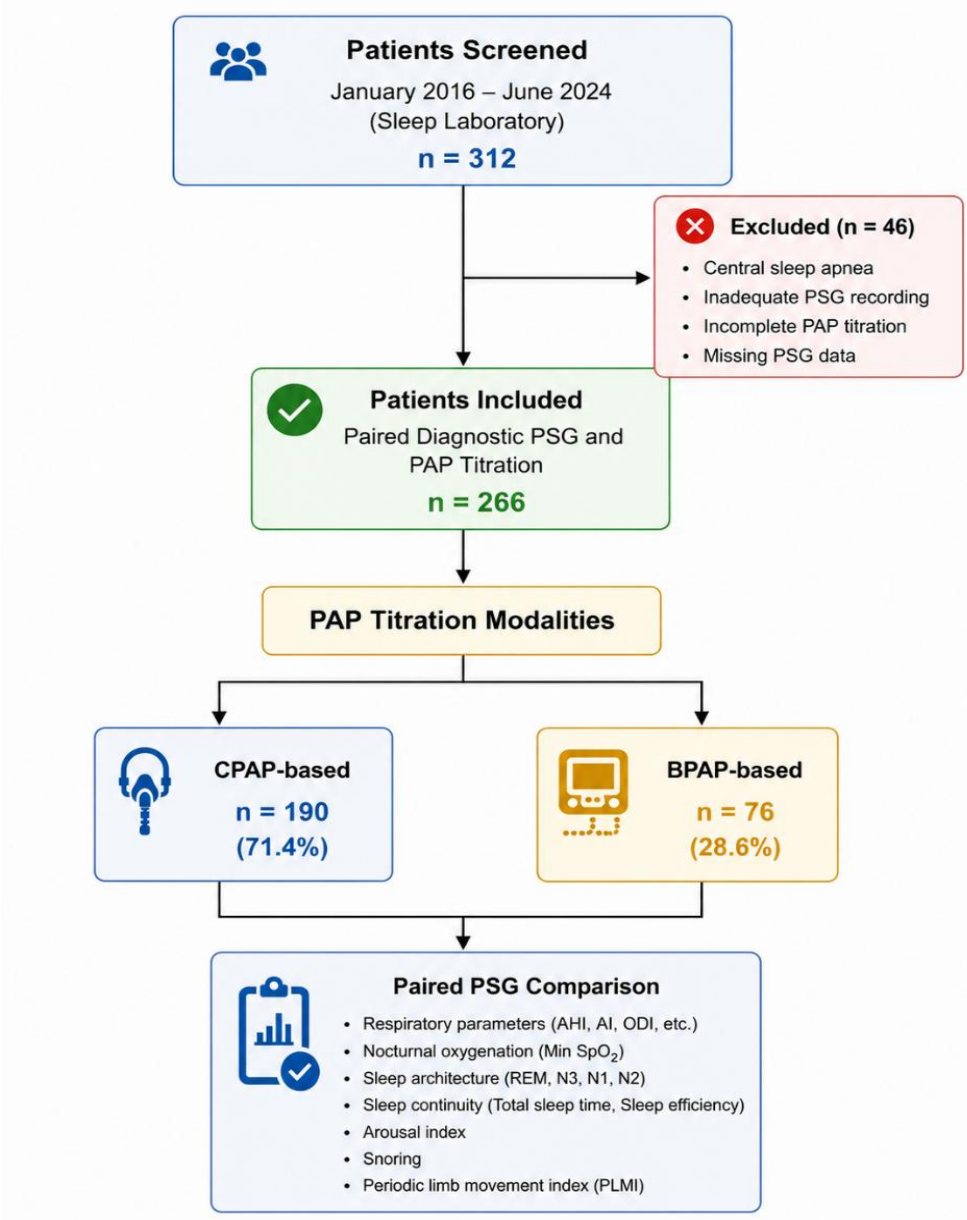


Figure 1. Study flow and participant selection. The diagram outlines the screening, exclusion, final cohort, and paired diagnostic-to-titration PSG analysis.

combined number of apneas and hypopneas per hour of sleep. For each study, we also recorded the apnea index (AI), oxygen desaturation index (ODI), minimum SpO_2, snoring burden, total sleep time (TST), total wake time (TWT), N1, N2, N3, and REM percentages; sleep and REM latencies, arousal index, periodic limb movement (PLM) index, and supine and non-supine AHI.

After the diagnostic study, the patients underwent overnight PAP titration with manual pressure adjustment. CPAP was used initially, and the pressure increased stepwise to control obstructive events, snoring, respiratory effort-related arousals, and oxygen desaturation while preserving comfort. The therapeutic pressure was selected according to the standard AASM titration guidance.

2.2. Outcome Measures: The prespecified primary outcome was the within-patient change in AHI from diagnostic PSG to PAP titration night. Secondary outcomes included changes in AI, ODI, minimum SpO_2, sleep and REM latencies, N1-N3 and REM sleep proportions, TST, TWT, arousal index, snoring burden, PLM index, and positional AHI values.

2.3. Subgroup analyses: To further investigate the immediate physiological response to PAP therapy, predefined subgroup analyses were performed according to both OSA severity and body mass index (BMI). Based on the diagnostic polysomnography, OSA severity was classified as mild (AHI 5.0–14.9 events/h), moderate (15.0–29.9 events/h), or severe (≥30.0 events/h).

Patients were also categorized according to BMI as normal weight (18.5–24.9 kg/m²), overweight (25.0–29.9 kg/m²), or obese (≥30.0 kg/m²). Patients were stratified into mild (n = 13), moderate (n = 61), and severe (n = 192) OSA groups, and into normal-weight (n = 18), overweight (n = 64), and obese (n = 184) BMI groups.

Within each subgroup, paired comparisons between the diagnostic and PAP titration nights were performed using the paired Student's T-test to evaluate changes in respiratory parameters, nocturnal oxygenation, sleep architecture, and sleep continuity. These subgroup analyses were conducted to determine whether the immediate physiological response to PAP differed according to baseline disease severity or BMI.

2.4. Statistical Analysis: Analyses were conducted using IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, NY, USA). The distributional characteristics of continuous variables were examined using the Kolmogorov-Smirnov test. Continuous data are reported as mean ± standard deviation (SD). Diagnostic and titration-night values were compared within participants using a paired Student's t-test. Statistical tests were two-sided, with $p<0.05$ defining significance.

2.5. Ethical Approval: The Institutional Ethics Committee of Hisar Intercontinental Hospital approved the study protocol (approval no. 26-74; 29 June 2026).

3. Results

A total of 266 patients were included in the study, comprising 213 men (80.1%) and 53 women (19.9%), with a mean age of 51.56 ± 14.04 years and a median BMI of 32.53 kg/m² (IQR, 28.93–36.65).

During titration, 190 patients (71.4%) were managed with CPAP-based modalities and 76 (28.6%) with BPAP-based ones. A comparison of the two nights showed broad changes in respiratory physiology (Table 1).

The mean AHI fell from 45.79 ± 23.14 to 4.03 ± 6.24 events/h (mean difference, 41.76 events/h; $p < 0.001$). AI declined from 34.40 ± 22.54 to 1.90 ± 4.09 events/h and ODI from 43.32 ± 23.33 to 3.39 ± 5.53 events/h (both $p < 0.001$). Minimum SpO_2 rose from 74.06 ± 11.59% to 85.50 ± 7.42% ($p < 0.001$).

The sleep stage distribution also shifted during titration. N3 sleep increased from 6.23 ± 7.84% to 11.57 ± 10.73%, and REM sleep from 11.32 ± 8.90% to 18.13 ± 9.06% (both $p < 0.001$), whereas N1 and N2 proportions decreased. REM latency shortened significantly; by contrast, sleep latency increased modestly but significantly (p=0.0018).

The arousal index, snoring burden, supine AHI, and non-supine AHI were all lower during PAP titration (all $p < 0.001$). The TST, TWT, and PLM index showed no statistically significant between-night differences (all $p > 0.05$)

Table 1. Within-patient comparison of polysomnographic variables during diagnostic PSG and PAP titration.

Variable	Diagnostic PSG Mean ± SD	PAP Titration Mean ± SD	Mean Difference	95% CI	*p* value
Sleep latency (min)	13.59 ± 13.48	17.70 ± 20.01	−4.11	−6.67 to −1.54	0.0018
REM latency (min)	141.52 ± 89.22	110.45 ± 70.77	31.06	17.82 to 44.31	<0.001
Total sleep time (min)	352.28 ± 79.57	338.11 ± 81.76	14.17	−2.74 to 31.08	0.099
Total wake time (min)	54.89 ± 47.16	54.64 ± 51.62	0.25	−8.61 to 9.11	0.955
N1 sleep (%)	17.95 ± 9.01	11.97 ± 7.46	5.97	3.85 to 8.10	<0.001
N2 sleep (%)	63.64 ± 13.17	58.11 ± 13.53	5.53	2.09 to 8.97	0.002
N3 sleep (%)	6.23 ± 7.84	11.57 ± 10.73	−5.34	−7.74 to −2.95	<0.001
REM sleep (%)	11.32 ± 8.90	18.13 ± 9.06	−6.81	−9.22 to −4.40	<0.001
AHI (events/h)	45.79 ± 23.14	4.03 ± 6.24	41.76	39.01 to 44.51	<0.001
AI (events/h)	34.40 ± 22.54	1.90 ± 4.09	32.50	29.77 to 35.24	<0.001
ODI (events/h)	43.32 ± 23.33	3.39 ± 5.53	39.93	37.02 to 42.84	<0.001
Minimum SpO_2 (%)	74.06 ± 11.59	85.50 ± 7.42	−11.44	−12.73 to −10.16	<0.001
Arousal index (events/h)	15.10 ± 17.67	5.46 ± 5.86	9.64	5.97 to 13.30	<0.001
Snoring %	83.86 ± 116.39	23.19 ± 37.22	60.68	31.04 to 90.32	<0.001
PLM index (events/h)	7.22 ± 15.32	8.18 ± 14.33	−0.96	−3.49 to 1.57	0.455
Supine AHI (events/h)	54.86 ± 26.74	6.06 ± 10.54	48.80	42.63 to 54.97	<0.001
Non-supine AHI (events/h)	33.15 ± 30.14	3.40 ± 13.76	29.75	22.90 to 36.59	<0.001

Values are presented as mean ± standard deviation (SD). The mean differences were calculated as diagnostic PSG − PAP titration PSG. P values were obtained using paired Student's t-test. PSG, polysomnography; PAP, positive airway pressure; AHI, apnea–hypopnea index; AI, apnea index; ODI, oxygen desaturation index; REM, rapid eye movement; N1, stage N1 sleep; N2, stage N2 sleep; N3, slow-wave sleep; SpO_2, peripheral oxygen saturation; PLM, periodic limb movement; CI, confidence interval.

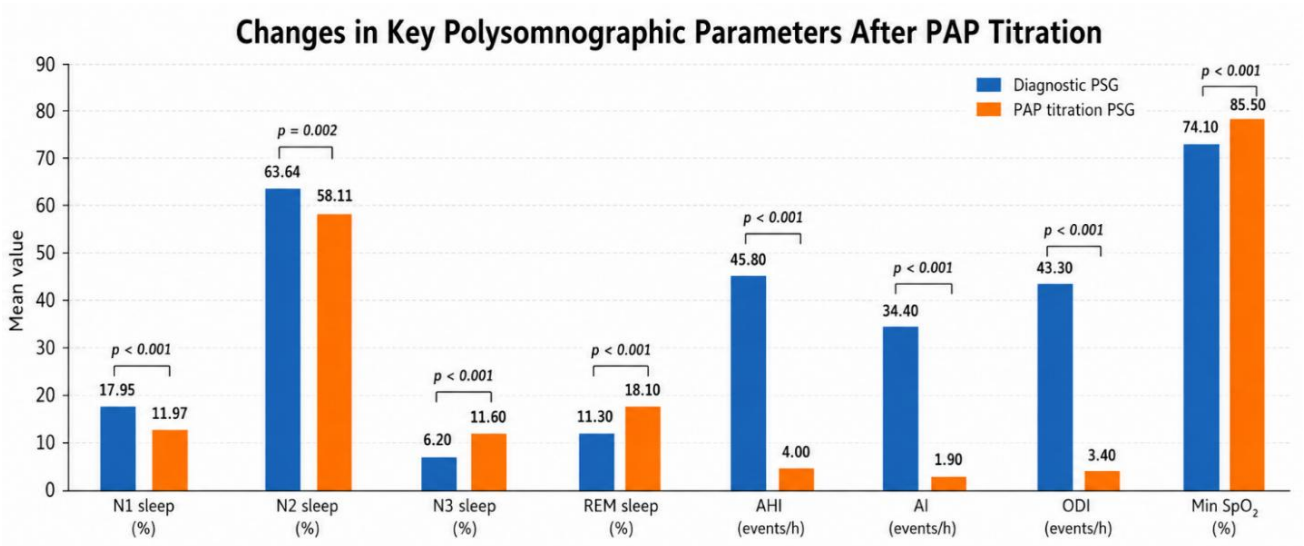


Figure 2. Changes in selected polysomnographic measures after PAP titration.

Paired testing showed lower AHI, AI, ODI, and N1 sleep and higher N3 sleep, REM sleep, and minimum SpO_2 levels during titration. Brackets denote paired comparisons and their corresponding p-values. Abbreviations: PAP, positive airway pressure; PSG, polysomnography; AHI, apnea–hypopnea index; AI, apnea index; ODI, oxygen desaturation index; REM, rapid eye movement; N1, stage N1 sleep; N3, slow-wave sleep; SpO_2, peripheral oxygen saturation.

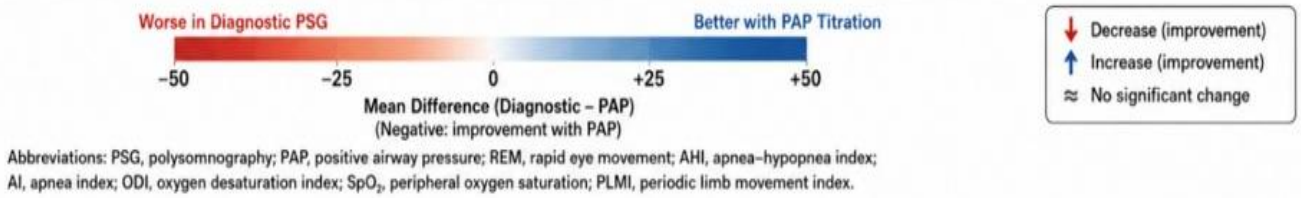
Figure 2 shows paired changes following PAP titration, and Table 2 summarizes the direction and magnitude of the changes across the full set of polysomnographic measures.

3.1. OSA subgroup analysis: Baseline OSA severity was used to define mild (n=13), moderate (n=61), and severe (n=192) groups (Table 3).

AHI and ODI decreased, and minimum SpO_2 increased significantly in each severity category. Absolute changes were largest in patients with severe OSA, consistent with the greater pretreatment respiratory burden in that group. The clearest stage redistribution occurred in patients with severe OSA, where N1 and N2 decreased, whereas N3 and REM sleep increased significantly. The mild and moderate groups generally changed in the same direction, but most stage-specific comparisons were not statistically significant. Sleep latency was prolonged in the moderate and severe groups but not in the mild OSA group. A significant reduction in REM latency was confined to severe OSA, while TST and TWT remained statistically unchanged across the severity strata.

Table 2. Direction and magnitude of polysomnographic changes from diagnostic PSG to PAP titration (n=266).

Parameter	Unit	Diagnostic PSG Mean ± SD	PAP Titration PSG Mean ± SD	Mean Difference (95% CI) (Diagnostic – PAP)	p value	Direction of Change
Sleep latency	min	20.2 ± 17.0	13.1 ± 12.3	7.1 (4.9 to 9.3)	<0.001	↓
REM latency	min	103.1 ± 62.1	145.3 ± 85.5	−42.2 (−55.0 to −29.4)	<0.001	↑
Total sleep time	min	362.0 ± 79.3	392.6 ± 66.1	−30.6 (−41.7 to −19.5)	<0.001	↑
Total wake time	min	61.8 ± 44.6	34.0 ± 29.8	27.8 (21.6 to 34.1)	<0.001	↓
Sleep efficiency	%	83.2 ± 9.8	89.7 ± 7.6	−6.5 (−7.9 to −5.1)	<0.001	↑
N1 sleep	%	6.2 ± 5.4	4.3 ± 4.1	1.9 (1.2 to 2.6)	<0.001	↓
N2 sleep	%	51.6 ± 9.9	50.3 ± 8.7	1.3 (−0.2 to 2.8)	0.092	≈
N3 sleep	%	16.0 ± 6.8	20.5 ± 6.7	−4.5 (−5.7 to −3.3)	<0.001	↑
REM sleep	%	18.5 ± 6.5	22.7 ± 6.4	−4.2 (−5.3 to −3.1)	<0.001	↑
AHI	events/h	45.8 ± 23.6	4.0 ± 3.8	41.8 (39.5 to 44.1)	<0.001	↓
AI	events/h	22.1 ± 15.1	1.6 ± 1.7	20.5 (19.1 to 21.9)	<0.001	↓
ODI	events/h	39.6 ± 22.0	3.7 ± 4.1	35.9 (33.9 to 37.9)	<0.001	↓
Minimum SpO_2	%	78.2 ± 7.8	91.6 ± 3.6	−13.4 (−14.3 to −12.5)	<0.001	↑
Arousal index	events/h	32.6 ± 16.4	15.3 ± 10.2	17.3 (15.2 to 19.4)	<0.001	↓
Snoring	%	56.8 ± 17.8	7.3 ± 10.5	49.5 (46.5 to 52.5)	<0.001	↓
PLMI	events/h	12.6 ± 14.6	8.1 ± 9.5	4.5 (2.8 to 6.2)	<0.001	↓
Supine AHI	events/h	49.3 ± 24.2	4.6 ± 5.6	44.7 (42.0 to 47.4)	<0.001	↓
Non-supine AHI	events/h	36.1 ± 20.5	3.3 ± 4.5	32.8 (30.6 to 35.0)	<0.001	↓



Abbreviations: PSG, polysomnography; PAP, positive airway pressure; REM, rapid eye movement; AHI, apnea–hypopnea index; AI, apnea index; ODI, oxygen desaturation index; SpO_2, peripheral oxygen saturation; PLMI, periodic limb movement index.

The heatmap presents mean values for both nights, paired mean differences (diagnostic PSG - PAP titration PSG), 95% confidence intervals, and p-values. Color intensity reflects the magnitude and direction of change, and arrows indicate whether values increased or decreased relative to the diagnostic PSG.

Abbreviations: PSG, polysomnography; PAP, positive airway pressure; AHI, apnea–hypopnea index; AI, apnea index; ODI, oxygen desaturation index; REM, rapid eye movement; N1, stage N1 sleep; N2, stage N2 sleep; N3, slow-wave sleep; SpO_2, peripheral oxygen saturation; PLMI, periodic limb movement index; CI, confidence interval.

Table 3. Within-patient comparison of polysomnographic variables by OSA severity.

Parameter	Mild OSA n=13			Moderate OSA n=61			Severe OSA n=192		
	1st PSG	2nd PSG	p	1st PSG	2nd PSG	p	1st PSG	2nd PSG	p
Sleep latency	16.05 ± 21.42	16.30 ± 24.66	0.952	12.31 ± 9.42	19.53 ± 15.16	<0.001	13.88 ± 14.11	17.20 ± 21.18	0.046
REM latency	148.10 ± 111.77	138.50 ± 80.92	0.739	135.38 ± 101.92	119.41 ± 70.60	0.291	143.13 ± 84.20	105.93 ± 70.21	<0.001
Total sleep time	398.00 ± 67.88	350.50 ± 20.51	0.391	351.76 ± 118.33	349.12 ± 88.51	0.890	350.80 ± 65.44	334.32 ± 81.54	0.098
Total wake time	59.70 ± 27.15	65.20 ± 40.73	0.669	45.38 ± 57.18	58.24 ± 51.70	0.219	57.61 ± 44.63	53.17 ± 52.61	0.382
N1 sleep (%)	18.70 ± 11.60	9.25 ± 1.91	0.399	16.18 ± 9.63	13.13 ± 5.91	0.275	18.46 ± 8.87	11.72 ± 7.99	<0.001
N2 sleep (%)	46.15 ± 5.73	54.15 ± 9.69	0.214	61.42 ± 16.25	59.19 ± 13.26	0.614	64.94 ± 11.89	57.93 ± 13.88	<0.001
N3 sleep (%)	15.90 ± 0.14	10.15 ± 3.46	0.247	6.39 ± 9.03	8.86 ± 10.15	0.270	5.83 ± 7.44	12.44 ± 11.03	<0.001
REM sleep (%)	19.20 ± 5.94	26.45 ± 4.31	0.500	16.01 ± 12.57	18.82 ± 7.98	0.375	9.62 ± 6.93	17.62 ± 9.41	<0.001
AHI	10.25 ± 2.62	2.67 ± 3.50	<0.001	23.42 ± 4.00	1.65 ± 1.36	<0.001	55.20 ± 20.09	4.88 ± 7.07	<0.001
ODI	8.92 ± 4.22	2.13 ± 3.99	0.003	22.20 ± 5.17	1.42 ± 1.16	<0.001	52.29 ± 21.14	4.11 ± 6.28	<0.001
Min SpO_2 (%)	82.18 ± 7.22	87.09 ± 6.28	0.017	80.41 ± 7.33	87.67 ± 5.88	<0.001	71.43 ± 11.90	84.68 ± 7.81	<0.001
Arousal index	4.90 ± 3.38	4.25 ± 4.75	0.819	8.77 ± 7.43	4.21 ± 2.50	0.016	17.66 ± 19.20	5.93 ± 6.50	<0.001

Values are presented as mean ± standard deviation (SD). P values were obtained using paired Student's t-test. 1st PSG, diagnostic polysomnography; 2nd PSG, PAP titration polysomnography. Sleep latency, REM latency, total sleep time, and total wake time were expressed in minutes (min). AHI, apnea–hypopnea index (events/h); ODI, oxygen desaturation index (events/h); SpO_2, peripheral oxygen saturation (%); arousal index, arousals (events/h).

AHI and ODI decreased, and minimum SpO_2 increased significantly in each severity category. Absolute changes were largest in patients with severe OSA, consistent with the greater pretreatment respiratory burden in that group. The clearest stage redistribution occurred in patients with severe OSA, where N1 and N2 decreased, whereas N3 and REM sleep increased significantly. The mild and moderate groups generally changed in the same direction, but most stage-specific comparisons were not statistically significant. Sleep latency was prolonged in the moderate and severe groups but not in the mild OSA group. A significant reduction in REM latency was confined to severe OSA, while TST and TWT remained statistically unchanged across the severity strata.

3.2. BMI Subgroup Analysis

BMI categories were normal weight (n=18), overweight (n=64), and obese (n=184) (Table 4).

Table 4. Within-patient comparison of polysomnographic variables according to BMI category.

Parameter	Normal weight BMI n=18			Overweight BMI n=64			Obese BMI n=184		
	1st PSG	2nd PSG	p	1st PSG	2nd PSG	p	1st PSG	2nd PSG	p
Sleep latency	11.86 ± 10.17	20.29 ± 17.66	0.049	14.63 ± 13.83	20.01 ± 19.10	0.041	13.36 ± 13.62	16.68 ± 20.51	0.040
REM latency	122.45 ± 52.49	111.96 ± 62.96	0.607	141.23 ± 98.55	115.42 ± 68.04	0.096	143.13 ± 88.29	108.59 ± 72.55	<0.001
Total sleep time	384.67 ± 52.34	339.92 ± 41.25	0.034	375.27 ± 82.63	329.33 ± 114.11	0.004	337.38 ± 78.44	341.99 ± 67.61	0.673
Total wake time	31.17 ± 18.88	46.85 ± 35.53	0.339	57.19 ± 52.57	68.10 ± 60.45	0.172	56.85 ± 46.88	49.34 ± 48.56	0.190
N1 sleep (%)	12.37 ± 8.87	8.98 ± 3.82	0.443	18.25 ± 8.95	14.02 ± 7.64	0.040	18.52 ± 9.01	11.40 ± 7.59	<0.001
N2 sleep (%)	63.68 ± 9.38	61.27 ± 12.10	0.703	59.78 ± 14.49	58.00 ± 15.85	0.542	65.44 ± 12.77	57.76 ± 12.75	0.001
N3 sleep (%)	9.73 ± 9.93	12.28 ± 14.93	0.711	8.31 ± 10.35	10.48 ± 11.98	0.262	4.80 ± 5.78	11.99 ± 9.74	<0.001
REM sleep (%)	12.20 ± 7.63	17.45 ± 8.62	0.356	13.65 ± 10.86	17.50 ± 10.18	0.059	10.12 ± 7.94	18.51 ± 8.73	<0.001
AHI	29.97 ± 19.81	3.77 ± 8.29	<0.001	38.54 ± 18.74	4.80 ± 8.17	<0.001	49.90 ± 23.63	3.78 ± 5.15	<0.001
ODI	25.76 ± 13.05	1.31 ± 1.50	<0.001	36.74 ± 18.53	3.58 ± 6.78	<0.001	47.43 ± 24.39	3.52 ± 5.24	<0.001
Min SpO_2 (%)	81.76 ± 4.55	89.82 ± 4.39	<0.001	76.09 ± 10.64	86.34 ± 7.00	<0.001	72.60 ± 12.01	84.79 ± 7.65	<0.001
Arousal index	8.82 ± 12.09	4.33 ± 5.18	0.201	13.05 ± 15.33	5.70 ± 6.33	0.005	16.89 ± 19.23	5.49 ± 5.82	<0.001

Values are presented as mean ± standard deviation (SD). P values were obtained using paired Student's t-test. 1st PSG, diagnostic polysomnography; 2nd PSG, PAP titration polysomnography. Sleep latency, REM latency, total sleep time, and total wake time were expressed in minutes (min). BMI, body mass index; AHI, apnea–hypopnea index (events/h); ODI, oxygen desaturation index (events/h); SpO_2, peripheral oxygen saturation (%); arousal index, arousals (events/h).

Significant improvements in AHI, ODI, and minimum SpO_2 were observed in all BMI categories. Patients with obesity showed the largest absolute respiratory changes and a higher baseline burden of respiratory events. Stage redistribution was strongest in the obese group, with significant decreases in N1 and N2 and increases in N3 and REM. Among overweight patients, only the reduction in N1 reached significance, whereas most stage changes in the normal-weight group remained nonsignificant despite a similar overall direction. Sleep latency increased in each BMI category. REM latency was significantly shortened only in patients with obesity. TWT did not change significantly in any BMI group; TST decreased in normal-weight and overweight patients but remained unchanged in the obese group.

4. Discussion

This paired analysis showed that PAP titration produces an immediate multidimensional physiological response in patients with OSA. During the first treatment night, respiratory event frequency, oxygenation, arousal burden, and sleep stage organization all changed substantially, whereas total sleep duration and PLM frequency were largely stable. This pattern indicates that effective PAP begins to alter sleep physiology as soon as obstructive breathing is controlled, rather than acting solely through a reduction in AHI [11-14, 16].

Respiratory control represented a prominent component of the acute response. The pronounced reductions in AHI, AI, and ODI, accompanied by higher minimum oxygen saturation, are consistent with the rapid stabilization of the upper airway and attenuation of intermittent hypoxemia. These findings are consistent with previous reports showing that PAP improves nocturnal ventilation and limits the physiological consequences of recurrent obstruction [10, 12, 14, 17, 18]. Because intermittent hypoxia contributes to cardiovascular and metabolic risks in OSA, early improvement in oxygenation may also have clinical relevance [3,6].

Sleep architecture changed in parallel, but not simply as a mirror of respiratory control. REM and N3 sleep increased, N1 and N2 sleep decreased, and arousal burden decreased during titration. Recurrent respiratory events and cortical arousals are known to disrupt normal sleep organization and suppress restorative stages [2, 3, 11]. Our findings indicate that once these disturbances are controlled, partial restoration of physiological sleep staging can occur on the first treatment night [11].

Total sleep time and periodic limb movement index did not differ significantly between the diagnostic PSG and PAP titration nights. This observation suggests that the primary benefit of PAP lies in improving sleep quality rather than increasing sleep duration [12, 14]. Likewise, periodic limb movements appear to be relatively independent of respiratory stabilization, indicating that they may require separate clinical evaluation when persistent [12].

Subgroup findings further showed that this early response was not identical across patients. Respiratory indices improved across all OSA severity and BMI categories, but the most pronounced reorganization of sleep stages occurred in patients with severe OSA and obesity. Greater absolute respiratory changes are expected in these groups, given their larger baseline burden; however, the unequal recovery of sleep architecture suggests that the normalization of breathing and sleep structure are related but not interchangeable outcomes [11, 16]. This supports the evaluation of early PAP effectiveness with more than residual AHI alone [6, 13, 16, 19].

Sleep latency provides an additional example of this dissociation. Despite broad physiological improvements, the time to sleep onset increased modestly during titration. Initial exposure to PAP involves a new mask, airflow sensation, and unfamiliar equipment, all of which may delay sleep onset and contribute to the first-night adaptation effect. Because these changes occurred alongside clear improvements in respiratory control, oxygenation, arousals, and restorative sleep, a small increase in sleep latency during the first titration night should not be viewed as evidence of treatment failure.

Neither the TST nor the PLM index changed significantly between nights. Thus, the earliest

benefit of PAP may be expressed more through sleep organization and physiological stability than through a longer sleep duration [11, 14, 20]. The lack of a significant change in PLM index also suggests that limb-movement activity is not necessarily corrected by respiratory stabilization and may require independent clinical consideration when it remains prominent [12].

The principal contribution of this study is the simultaneous characterization of several physiological domains during the first PAP titration night using paired data from the same patient. Most established evidence concerns longer-term PAP effects, whereas this design captures the immediate changes in breathing, oxygenation, arousals, sleep continuity, snoring, and sleep stages after pressure adjustment. Using each participant as their own comparator reduces between-subject variability, and stratification by OSA severity and BMI demonstrates that the size and pattern of the early response partly depend on baseline characteristics. In this sense, titration night provides information about acute physiological recovery in addition to its conventional role in selecting therapeutic pressure [11, 13, 14, 21].
The findings should be interpreted considering several limitations of this study. This study was retrospective and conducted at a single center, and only short-term physiological responses during titration were available. We did not evaluate long-term adherence, subsequent clinical outcomes, cardiovascular biomarkers, daytime sleepiness, or QoL. Therefore, prospective multicenter studies are needed to determine whether specific first-night response patterns are associated with sustained PAP use or later clinical benefits.

4.1. Conclusion: In conclusion, effective PAP titration was associated with rapid improvements in respiratory disturbances, nocturnal oxygenation, arousal burden, and sleep architecture in patients with OSA. The first-night response was not confined to AHI reduction and varied in magnitude across the severity and BMI strata. Therefore, the assessment of PAP effectiveness may be strengthened by considering oxygenation, sleep continuity, and sleep stage recovery, alongside the frequency of residual respiratory events.

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