

Polysomnographic analysis of sleep architecture and oxygen saturation parameters in primary snoring

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Abstract

Primary snoring is traditionally considered a benign condition. However, emerging evidence suggests that it may be associated with subtle disturbances in sleep architecture. Data on the effects of primary snoring on sleep architecture and nocturnal oxygenation, as assessed by full polysomnography, remain limited and inconsistent.

To evaluate sleep architecture and oxygen saturation parameters in individuals with primary snoring using full-night polysomnography, excluding patients with obstructive sleep apnea.

This retrospective study analyzed overnight polysomnographic recordings of individuals diagnosed with primary snoring. Participants with obstructive sleep apnea were excluded based on standard apnea–hypopnea index thresholds. Sleep architecture parameters, including sleep stage distribution and wake after sleep onset (WASO), indices of sleep fragmentation, respiratory event characteristics, and nocturnal oxygen saturation were assessed.

Primary snoring was associated with significant alterations in sleep architecture, characterized by reductions in slow-wave (N3) and rapid eye movement (REM) sleep, and an increase in wake after sleep onset (WASO). No significant increase in conventional sleep fragmentation indices was observed. Despite the absence of obstructive sleep apnea, primary snoring was associated with an increased number of obstructive respiratory events and a reduction in mean nocturnal oxygen saturation.

Primary snoring is associated with altered sleep architecture and nocturnal hypoxia, even in the absence of obstructive sleep apnea. Reductions in N3 and REM sleep may represent potential mechanisms linking snoring to sympathetic activation, elevated blood pressure, and increased cardiovascular risk, as well as impaired emotional regulation and memory processing. The observed intermittent hypoxia, although milder than that seen in obstructive sleep apnoea, may have pathophysiological significance. Further prospective studies are needed to evaluate the long-term clinical impact of alterations in sleep architecture and low-grade intermittent hypoxia in individuals with primary snoring.

1. Introduction

Snoring is the characteristic noise produced when airflow is partially obstructed during sleep, causing the soft tissues of the upper airway to vibrate [1]. It is highly prevalent in the general population, affecting an estimated 20–40% of adults [2]. Snoring occupies the mild end of the spectrum of sleep-disordered breathing and is often termed primary snoring when it occurs without accompanying obstructive sleep apnoea (OSA). In clinical and research practice, OSA is commonly defined by the apnoea–hypopnoea index (AHI), and primary snoring is typically considered in individuals below diagnostic thresholds for OSA [3]. Despite its ubiquity, clinical characterization of snoring remains challenging because definitions and measurement approaches vary widely, and subjective reports are prone to misclassification [4–6]. There is therefore increasing interest in the objective quantification of “snoring burden” using

polysomnography (PSG) or validated acoustic methods, including indices derived from overnight recordings of the frequency and intensity of snoring events [4, 7, 8].

Although primary snoring has long been regarded as a benign social nuisance, accumulating observational evidence suggests associations with adverse cardiometabolic profiles. Habitual snoring has been linked to elevated blood pressure and hypertension, even in analyses that accounted for coexisting OSA and other covariates [9, 10]. Snoring has also been associated with metabolic dysregulation, including metabolic syndrome, in population studies and meta-analyses, although much of this literature relies on self-reported exposure and remains vulnerable to residual confounding by adiposity and undiagnosed sleep-disordered breathing [11–13]. Beyond risk factors, several studies have examined subclinical vascular outcomes, reporting associations between snoring and carotid atherosclerotic markers in selected cohorts [5, 14, 15]. At the same time, findings across cohorts are not fully consistent. For example, long-term community follow-up did not identify snoring as an independent predictor of all-cause mortality or incident cardiovascular outcomes after accounting for relevant covariates, underscoring the need for more precise exposure assessment and better separation of snoring from coexisting sleep-disordered breathing [16]. Several mechanisms have been proposed to explain how snoring could be linked to adverse health outcomes, but these should be distinguished from established observations. From a sleep physiology perspective, snoring reflects partial upper-airway obstruction and inspiratory flow limitation, which may be accompanied by arousals and sleep fragmentation that could, over time, influence sleep continuity and architecture [2, 17]. Snoring also generates nocturnal noise that can reach levels associated with sleep disturbance and sympathetic activation, potentially affecting both the snorer and the bed partner, although the extent and clinical significance likely vary by context and measurement method [18]. Vibration-related vascular hypotheses are supported by experimental work showing that snoring-level tissue vibrations can induce endothelial dysfunction in carotid arteries, thereby providing a plausible pathway linking snoring-related mechanical stress to localized vascular changes [19, 20]. Finally, while primary snoring is defined by the absence of overt OSA by diagnostic criteria, subtle abnormalities in nocturnal oxygen saturation may still be relevant when studying early or subthreshold respiratory disturbance – hence the emerging emphasis on hypoxic “burden” and desaturation characteristics in sleep-disordered breathing research [21].

Polysomnographic evidence suggests that simple snoring may be associated with sleep bruxism, particularly through relationships between snore intensity, body position, and phasic bruxism activity in non-apneic individuals [22].

The available data indicate that age and sex may modify this relationship, with snoring and sleep bruxism appearing more intense in men, while older patients tended to snore more in N2 sleep and the supine position but showed fewer bruxism episodes [23].

In patients with myogenous temporomandibular disorders (TMD), recent PSG-based data did not show a direct positive association between overall snoring and masticatory muscle pain, but specific snoring patterns, especially during non-supine REM sleep, were inversely related to pain intensity, suggesting a

complex and potentially modulatory link between snoring and TMD-related muscle pain [24]. Another study showed that sleep-related respiratory complaints were common in this sample of persistent orofacial pain/temporomandibular disorders (~ 22% reported snoring and/or apnea symptoms on screening) and respiratory distress was consistently associated with greater psychosocial burden, with higher rates of clinically relevant scores across multiple domains, particularly anxiety, global mental health distress, perceived injustice, and pain-related disability[25].

Despite these concerns and the multifaceted nature of snoring, primary snoring remains relatively under investigated with full-night PSG, which provides objective, graded measures of snoring burden. Many PSG-based studies have stratified snorers into broad severity groups, which limits our ability to discern whether increasing snoring burden (within the non-OSA range) correlates with systematic differences in sleep stages, sleep continuity, or oxygen saturation [4, 17]. In parallel, methodological studies and recent digital health approaches highlight that objective snoring measurement is feasible and may improve comparability across studies, but there is still no widely adopted framework linking objective snoring burden to physiological PSG endpoints among adults without OSA [4, 7, 10]

The present study was designed to help address this gap. We performed a retrospective analysis of full-night PSG recordings from adults who met criteria for primary snoring, with OSA ruled out using standard diagnostic cut-offs [3]. We aimed to determine whether an increase in objectively measured snoring burden, quantified using PSG-derived metrics such as the snore index and snore train measures, is associated with changes in sleep architecture and oxygen saturation parameters in individuals without OSA. We hypothesized that greater snoring burden would be associated with altered sleep continuity and stage distribution, and with less favorable oxygen saturation profiles, even when respiratory event indices remain below diagnostic thresholds.

2. Materials and methods

2.1 Study Participants

This observational study included 149 adults diagnosed with primary snoring who underwent attended, full-night diagnostic polysomnography in the sleep laboratory of the Department of Diabetology, Hypertension, and Internal Diseases in Wroclaw Medical University between January 2017 and December 2024. The mean age of the participants was 33.67 years. The study was conducted in accordance with the Declaration of Helsinki. The study was approved by the local institutional ethics committee (No. 305/2025), and all participants provided written informed consent prior to any study procedures.

Eligibility criteria included age > 18 years, written informed consent, and an apnea–hypopnea index < 5 events per hour of sleep. Primary snoring was operationally defined as objectively confirmed snoring during polysomnography in the absence of obstructive sleep apnea, using AHI as the qualifying respiratory metric for inclusion [26].

Exclusion criteria included clinically relevant respiratory and/or heart insufficiency, or severe chronic respiratory disorders, active systemic disease, malignancy, clinically significant inflammation, severe neurological or psychiatric disorders, pregnancy, or breastfeeding. Prior treatment or ongoing use of agents with a known impact on sleep or ventilatory control, including sedatives, neuroleptics, antidepressants, analgesics, or muscle relaxants, led to exclusion. Recordings were excluded when polysomnography could not be completed, when the total recorded sleep time was insufficient for valid scoring, or when signal quality precluded reliable analysis of respiratory, oximetry, or audio channels.

2.2 Polysomnographic methodology and snoring assessment

All participants underwent a single night, attended polysomnography conducted according to the American Academy of Sleep Medicine (AASM) recommendations [27]. A full-night polysomnographic assessment was performed using Nox-A1 (Nox Medical, Reykjavik, Iceland). The recording montage comprised electroencephalography with electrodes positioned according to the international 10–20 system, electrooculography, submental electromyography, and electrocardiography. Respiratory monitoring included airflow assessment with a nasal pressure transducer for hypopnea detection and an oronasal thermal sensor for apnea detection, as well as thoracoabdominal respiratory-effort belts, body-position sensors, and pulse oximetry for oxygen saturation. A synchronized acoustic signal was recorded for snoring assessment [28].

Polysomnograms were scored in 30-second epochs. Sleep stages were classified as wake, N1, N2, N3, and REM according to current AASM sleep staging rules, and automated outputs were manually reviewed to ensure scoring accuracy. Cortical arousals were scored based on an abrupt shift in EEG frequency lasting at least 3 seconds, preceded by at least 10 seconds of sleep, with the additional requirement of a concurrent increase in submental EMG tone during REM sleep [29].

Respiratory events were scored using AASM respiratory rules [27]. Apnea was defined by a marked reduction in the airflow signal consistent with AASM criteria, and hypopnea was scored when a reduction in airflow met duration criteria and was accompanied by either an oxygen desaturation of at least 3% from pre-event baseline, or an arousal, consistent with the recommended adult hypopnea rule [30].

Sleep architecture variables included total sleep time (TST), sleep efficiency (SE), sleep latency (SL), rapid eye movement sleep (REM) latency, wake after sleep onset (WASO), and the relative contribution of stage N1 sleep (N1), stage N2 sleep (N2), stage N3 sleep (N3), and REM to total sleep time. Oxygenation outcomes included mean oxygen saturation, nadir oxygen saturation, the proportion of sleep with oxygen saturation below 90%, and the oxygen desaturation index defined as the number of qualifying desaturation episodes per hour of sleep, using a predefined desaturation threshold [31].

Snoring was quantified from the acoustic channel recorded during polysomnography, using a protocol consistent with prior audio-based polysomnographic analyses. The full audio signal was obtained at a

high sampling frequency and further processed into an envelope signal, enabling automated event detection. Snore events were identified as breath-synchronous acoustic vibrations with a clear oscillatory pattern that exceeded background noise. Both inspiratory and expiratory snores were eligible, while non-snore sounds, including cough, groaning, bruxism-related sounds, bedding noise, movement artifacts, and prominent non-vibratory breathing sounds, were excluded. The snore index was defined as the number of individual snore events per hour of sleep. A snore train was defined as a period comprising multiple consecutive single snores, and snore train frequency was expressed per hour of sleep [32].

2.3 Statistical analysis.

Statistical analyses were performed using Dell Statistica software, version 13.1 (Dell Inc., Round Rock, TX, USA). The distributional properties of continuous variables were assessed prior to inferential testing, and homogeneity of variance was evaluated where required to support the choice of parametric procedures. Continuous variables were summarized using mean and standard deviation when distributional assumptions were met, whereas skewed variables were described using median and interquartile range. Categorical variables were reported as counts and proportions. Group comparisons and correlation analyses were conducted using parametric or non-parametric approaches as appropriate for the data structure and distribution, with a two-sided significance threshold of $p < 0.05$.

For snoring outcomes, the median snore index and the median snore train frequency were calculated. Based on the distribution of these median-based snoring measures in the study cohort, participants were stratified into four quartiles (Q1-Q4), reflecting increasing snoring burden. Comparative analyses of sleep architecture and oxygen saturation parameters were then performed across quartiles using methods selected according to the same distribution-guided principles described above. The number of patients participating in the study was estimated based on the sample size calculator assuming the following calculation conditions: population size 2 billion, fraction size 0.5, confidence level 95%, maximum error 10%. The number of patients recruited for the study exceeded the minimum value of 97 people.

3. Results

First, since definitive standards for snoring have not yet been established, we calculated the median of the snoring parameters and then compared these groups (Table 1). We then identified subgroups Q1, Q2, Q3, and Q4 for these parameters. We analyzed the snore index first. Based on the median value for this parameter, two subgroups were identified: the low snore index and the high snore index. Interestingly, no statistically significant differences were found in the proportions of individual sleep phases. Statistical analysis of the high–snore-index subgroup revealed significantly higher values of AHI (3.57 ± 2.49 vs. 1.72 ± 1.60), ODI (4.00 ± 2.89 vs. 2.06 ± 1.82), AI (0.89 ± 0.95 vs. 0.50 ± 0.58), OA (0.40 ± 0.60 vs. 0.15 ± 0.26), MA (0.03 ± 0.07 vs. 0.01 ± 0.03), and HI (2.80 ± 2.42 vs. 1.22 ± 1.41) compared with the low–snore-index subgroup. Conversely, mean oxygen saturation (94.54 ± 1.51 vs. 95.19 ± 1.13) and minimum

oxygen saturation (85.84 ± 8.29 vs. 88.80 ± 7.02) were significantly lower in the high–snore-index subgroup. Detailed data are presented in Table 1.

Table 1

Polysomnographic parameters of patients with primary snoring based on the snore index. Classification based on the median.

Parameter	ALL	Subgroup 1 (low snore index) $\leq 25,8$ (median)	Subgroup 2 (high snore index) > 25.8 (median)	p
	Average	Average	Average	
TST (min)	441,35 $\pm$ 51,22	440,57 $\pm$ 52,26	442,14 $\pm$ 50,49	ns
SE (%)	86,63 $\pm$ 11,2	86,63 $\pm$ 9,24	86,64 $\pm$ 12,95	ns
WASO (min)	39,4 $\pm$ 44,86	37,08 $\pm$ 33,61	41,76 $\pm$ 54,06	ns
SL (min)	19,81 $\pm$ 16,73	20,19 $\pm$ 16,04	19,42 $\pm$ 17,5	ns
N1 (% of TST)	4,53 $\pm$ 4,41	4,2 $\pm$ 5,01	4,86 $\pm$ 3,7	ns
N2 (% of TST)	49,06 $\pm$ 9,25	48,44 $\pm$ 9,72	49,69 $\pm$ 8,77	ns
N3 (% of TST)	24,07 $\pm$ 7,78	25,1 $\pm$ 8,62	23,01 $\pm$ 6,72	ns
REM (% of TST)	22,6 $\pm$ 6,31	22,75 $\pm$ 5,51	22,44 $\pm$ 7,06	ns
AHI (n/hour)	2,64 $\pm$ 2,28	1,72 $\pm$ 1,6	3,57 $\pm$ 2,49	p < 0.001
ODI (n/hour)	3,02 $\pm$ 2,59	2,06 $\pm$ 1,82	4 $\pm$ 2,89	p < 0.001
Snore (% of TST)	7,59 $\pm$ 12,9	0,5 $\pm$ 1,71	14,78 $\pm$ 15,19	p < 0.001
AI (n/hour)	0,69 $\pm$ 0,81	0,5 $\pm$ 0,58	0,89 $\pm$ 0,95	p < 0.001
OA (n/hour)	0,27 $\pm$ 0,48	0,15 $\pm$ 0,26	0,4 $\pm$ 0,6	p < 0.001
MA (n/hour)	0,02 $\pm$ 0,06	0,01 $\pm$ 0,03	0,03 $\pm$ 0,07	p < 0.001
CA (n/hour)	0,39 $\pm$ 0,55	0,33 $\pm$ 0,46	0,45 $\pm$ 0,62	ns
HI (n/hour)	2,01 $\pm$ 2,12	1,22 $\pm$ 1,41	2,8 $\pm$ 2,42	p < 0.001

TST, total sleep time; SE, sleep efficiency; WASO, wake after sleep onset; SL, sleep latency; N1, stage N1 sleep; N2, stage N2 sleep; N3, stage N3 sleep; REM, rapid eye movement sleep; AHI, apnea–hypopnea index; ODI, oxygen desaturation index; AI, apnea index; OA, obstructive apnea index; MA, mixed apnea index; CA, central apnea index; HI, hypopnea index; SpO₂, oxygen saturation; Mean SpO₂, mean oxygen saturation; Min. sat., minimum oxygen saturation; SpO₂ duration < 90%, duration of oxygen saturation below 90%; Aver. desat., average desaturation; Arousals, arousal index; Pulse aver., average pulse rate; Pulse max., maximum pulse rate; Pulse min., minimum pulse rate;

Parameter	ALL	Subgroup 1 (low snore index) $\leq 25,8$ (median)	Subgroup 2 (high snore index) > 25.8 (median)	<i>p</i>
	Average	Average	Average	
Mean SpO ₂ (%)	94,87 $\pm$ 1,37	95,19 $\pm$ 1,13	94,54 $\pm$ 1,51	<i>p</i> < 0.001
min. sat.	87,33 $\pm$ 7,79	88,8 $\pm$ 7,02	85,84 $\pm$ 8,29	<i>p</i> < 0.001
SpO ₂ Duration < 90%	1,53 $\pm$ 4,01	1,28 $\pm$ 3,15	1,78 $\pm$ 4,74	ns
Aver. desat.	3,16 $\pm$ 0,69	2,98 $\pm$ 0,78	3,35 $\pm$ 0,54	<i>p</i> < 0.001
Arousals (n/hour)	4,63 $\pm$ 5,37	4,57 $\pm$ 7,08	4,7 $\pm$ 2,74	ns
Pulse aver. n/hour)	63,19 $\pm$ 9,55	62,38 $\pm$ 8,94	64,01 $\pm$ 10,11	ns
Pulse max. n/hour)	100,81 $\pm$ 19,7	98,07 $\pm$ 13,51	103,58 $\pm$ 24,21	ns
Pulse min. n/hour)	49,63 $\pm$ 9,14	49,3 $\pm$ 8,67	49,96 $\pm$ 9,63	ns
TST, total sleep time; SE, sleep efficiency; WASO, wake after sleep onset; SL, sleep latency; N1, stage N1 sleep; N2, stage N2 sleep; N3, stage N3 sleep; REM, rapid eye movement sleep; AHI, apnea–hypopnea index; ODI, oxygen desaturation index; AI, apnea index; OA, obstructive apnea index; MA, mixed apnea index; CA, central apnea index; HI, hypopnea index; SpO ₂ , oxygen saturation; Mean SpO ₂ , mean oxygen saturation; Min. sat., minimum oxygen saturation; SpO ₂ duration < 90%, duration of oxygen saturation below 90%; Aver. desat., average desaturation; Arousals, arousal index; Pulse aver., average pulse rate; Pulse max., maximum pulse rate; Pulse min., minimum pulse rate;				

Subsequently, the study participants were divided into four subgroups based on their snore index scores. A statistically significant difference in REM sleep duration was observed between subgroups 3 and 4 (24.05 $\pm$ 7.00 vs. 20.83 $\pm$ 6.83). Detailed results for the remaining comparisons are presented in Table 2.

Table 2
Polysomnographic parameters of patients with primary snoring based on the snoring index.
Classification based on quartiles

Parameter	ALL	Subgroup 1 (irrelevant snoring): ≤7,4 (Q1)	Subgroup 2 (mild snoring): 7,5–25,9 (Q2)	Subgroup 3 (moderate snoring): 26– 121 (Q3)	Subgroup 4 (severe snoring): >121 (Q4)	p
	Average	Average	Average	Average	Average	
TST (min)	441,35 ± 51,22	427,32 ± 58,66	454,18 ± 41,24	447,06 ± 47,79	437,22 ± 53,26	ns
SE (%)	86,63 ± 11,2	85,7 ± 11,35	87,59 ± 6,41	88,43 ± 10,59	84,84 ± 14,88	ns
WASO (min)	39,4 ± 44,86	42,48 ± 40,6	31,54 ± 23,79	28,69 ± 19,54	54,82 ± 72,07	ns
SL (min)	19,81 ± 16,73	19,37 ± 12,67	21,04 ± 19,03	20,33 ± 16,76	18,51 ± 18,39	ns
N1 (% of TST)	4,53 ± 4,41	3,66 ± 2,31	4,75 ± 6,74	4,59 ± 3,55	5,14 ± 3,87	ns
N2 (% of TST)	49,06 ± 9,25	48,27 ± 10,82	48,62 ± 8,58	47,55 ± 10,23	51,83 ± 6,48	ns
N3 (% of TST)	24,07 ± 7,78	24,74 ± 8,69	25,48 ± 8,65	23,81 ± 7,05	22,22 ± 6,36	ns
REM (% of TST)	22,6 ± 6,31	23,33 ± 5,76	22,16 ± 5,25	24,05 ± 7.00	20,83 ± 6,83	A3-A4: 0.04
AHI (n/hour)	2,64 ± 2,28	1,23 ± 1,35	2,22 ± 1,7	3,13 ± 2,29	4,02 ± 2,62	A1-A2: 0.02 A1-A3: < 0.001 A1-A4:< 0.001 A2-A4: < 0.001
ODI (n/hour)	3,02 ± 2,59	1,66 ± 1,52	2,46 ± 2,03	3,56 ± 2,86	4,44 ± 2,89	A1-A3: 0.004 A1-A4: <0.001 A2-A4: 0.002

Parameter	ALL	Subgroup 1 (irrelevant snoring): ≤7,4 (Q1)	Subgroup 2 (mild snoring): 7,5–25,9 (Q2)	Subgroup 3 (moderate snoring): 26– 121 (Q3)	Subgroup 4 (severe snoring): >121 (Q4)	p
	Average	Average	Average	Average	Average	
Snore (% of TST)	7,59 ± 12,9	0,36 ± 1,63	0,65 ± 1,79	5,24 ± 3,97	24,32 ± 16,27	A1-A2: <0.001 A1-A3: <0.001 A1-A4: <0.001 A2-A3: <0.001 A2-A4: <0.001 A3-A4: <0.001
AI (n/hour)	0,69 ± 0,81	0,38 ± 0,52	0,62 ± 0,62	0,77 ± 0,72	1 ± 1,13	A1-A4: 0.004
OA (n/hour)	0,27 ± 0,48	0,1 ± 0,21	0,2 ± 0,29	0,32 ± 0,43	0,48 ± 0,74	A1-A3: 0.005 A1-A4: < 0.001 A2-A4: 0.005
MA (n/hour)	0,02 ± 0,06	0,01 ± 0,04	0 ± 0,02	0,02 ± 0,07	0,04 ± 0,07	A1-A4: 0.04 A2-A4: 0.005
CA (n/hour)	0,39 ± 0,55	0,27 ± 0,39	0,41 ± 0,51	0,43 ± 0,57	0,46 ± 0,68	ns
HI (n/hour)	2,01 ± 2,12	0,85 ± 1,02	1,6 ± 1,65	2,35 ± 2,09	3,25 ± 2,66	A1-A3: 0.005 A1-A4: < 0.001 A2-A4: 0.001

Parameter	ALL	Subgroup 1 (irrelevant snoring): ≤7,4 (Q1)	Subgroup 2 (mild snoring): 7,5–25,9 (Q2)	Subgroup 3 (moderate snoring): 26– 121 (Q3)	Subgroup 4 (severe snoring): >121 (Q4)	p
	Average	Average	Average	Average	Average	
Mean SpO ₂ (%)	94,87 ± 1,37	95,22 ± 1,15	95,16 ± 1,13	94,91 ± 1,15	94,16 ± 1,74	A1-A4: 0.004 A2-A4: 0.007
min. sat.	87,33 ± 7,79	88,63 ± 7,39	88,97 ± 6,72	85,78 ± 8,89	85,89 ± 7,76	A1-A4: 0.02 A2-A4: 0.02
SpO ₂ Duration < 90%	1,53 ± 4,01	1,72 ± 3,72	0,83 ± 2,39	2,08 ± 5,02	1,48 ± 4,5	A1-A4: 0.003 A2-A4: 0.006
Aver. desat.	3,16 ± 0,69	2,85 ± 1,04	3,11 ± 0,34	3,36 ± 0,67	3,34 ± 0,37	A1-A2: 0.002 A1-A3: < 0.001 A1-A4: 0.03
Arousals (n/hour)	4,63 ± 5,37	3,79 ± 2,4	5,36 ± 9,79	4,99 ± 3,11	4,42 ± 2,31	ns
Pulse aver. n/hour)	63,19 ± 9,55	63,22 ± 9,81	61,52 ± 8	61,8 ± 8,66	66,22 ± 11,06	ns
Pulse max. n/hour)	100,81 ± 19,7	100,32 ± 15,66	95,76 ± 10,59	100,84 ± 21,56	106,32 ± 26,62	ns
Pulse min. n/hour)	49,63 ± 9,14	48,7 ± 10,53	49,92 ± 6,32	49,95 ± 7,4	49,97 ± 11,55	ns

TST, total sleep time; SE, sleep efficiency; WASO, wake after sleep onset; SL, sleep latency; N1, stage N1 sleep; N2, stage N2 sleep; N3, stage N3 sleep; REM, rapid eye movement sleep; AHI, apnea–hypopnea index; ODI, oxygen desaturation index; AI, apnea index; OA, obstructive apnea index; MA, mixed apnea index; CA, central apnea index; HI, hypopnea index; SpO₂, oxygen saturation; Mean SpO₂, mean oxygen saturation; Min. sat., minimum oxygen saturation; SpO₂ duration < 90%, duration of oxygen saturation

below 90%; Aver. desat., average desaturation; Arousals, arousal index; Pulse aver., average pulse rate; Pulse max., maximum pulse rate; Pulse min., minimum pulse rate; TST, total sleep time.

Subgroup analysis by snore train percentage showed no significant differences in most sleep architecture parameters, including TST, SE, WASO, SL, and the proportions of N1, N2, and REM sleep. However, the proportion of N3 sleep was significantly lower in the high snore train percentage subgroup than in the low snore train percentage subgroup (23.16 ± 6.72 vs. 24.89 ± 8.59 ; $p < 0.05$).

Respiratory parameters were significantly higher in the high snore-train percentage subgroup, including AHI (3.46 ± 2.44 vs. 1.89 ± 1.85), ODI (4.01 ± 2.94 vs. 2.13 ± 1.85), OA (0.40 ± 0.62 vs. 0.16 ± 0.26), MA (0.03 ± 0.07 vs. 0.01 ± 0.03), and HI (2.70 ± 2.34 vs. 1.37 ± 1.68), as well as the proportion of sleep time spent snoring (15.38 ± 15.22 vs. 0.51 ± 1.67) (all $p < 0.001$).

In turn, mean oxygen saturation (94.54 ± 1.54 vs. 95.16 ± 1.12) and minimum oxygen saturation (85.76 ± 8.45 vs. 88.76 ± 6.89) were significantly lower in the high snore train percentage subgroup (both $p < 0.001$). Additionally, the duration of $\text{SpO}_2 < 90\%$ (1.85 ± 4.83 vs. 1.23 ± 3.10 ; $p < 0.05$) and average desaturation (3.38 ± 0.52 vs. 2.96 ± 0.77 ; $p < 0.001$) were significantly higher in this subgroup.

No significant differences were found in arousal index or pulse parameters. Detailed data are presented in Table 3.

Table 3

Polysomnographic parameters of patients with primary snoring based on the snore train percentage.
Classification based on the median.

Parameter	ALL	Subgroup 1 (low snore train percentage) $\leq 1,3$ (median)	(high snore train percentage) $> 1,3$ (median) Kolumna3	Kolumna4p
	Average	Average	Average	
TST (min)	441,35 $\pm$ 51,22	440,16 $\pm$ 53,37	442,66 $\pm$ 49,1	ns
SE (%)	86,63 $\pm$ 11,2	86,89 $\pm$ 9,17	86,35 $\pm$ 13,13	ns
WASO (min)	39,4 $\pm$ 44,86	35,83 $\pm$ 33,48	43,33 $\pm$ 54,7	ns
SL (min)	19,81 $\pm$ 16,73	20,24 $\pm$ 15,79	19,33 $\pm$ 17,8	ns
N1 (% of TST)	4,53 $\pm$ 4,41	4,41 $\pm$ 5,15	4,65 $\pm$ 3,45	ns
N2 (% of TST)	49,06 $\pm$ 9,25	48,23 $\pm$ 9,63	49,98 $\pm$ 8,78	ns
N3 (% of TST)	24,07 $\pm$ 7,78	24,89 $\pm$ 8,59	23,16 $\pm$ 6,72	p < 0.05
REM (% of TST)	22,6 $\pm$ 6,31	22,94 $\pm$ 5,5	22,22 $\pm$ 7,11	ns
AHI (n/hour)	2,64 $\pm$ 2,28	1,89 $\pm$ 1,85	3,46 $\pm$ 2,44	p < 0.001
ODI (n/hour)	3,02 $\pm$ 2,59	2,13 $\pm$ 1,85	4,01 $\pm$ 2,94	p < 0.001
Snore (% of TST)	7,59 $\pm$ 12,9	0,51 $\pm$ 1,67	15,38 $\pm$ 15,22	p < 0.001
AI (n/hour)	0,69 $\pm$ 0,81	0,52 $\pm$ 0,58	0,88 $\pm$ 0,97	ns
OA (n/hour)	0,27 $\pm$ 0,48	0,16 $\pm$ 0,26	0,4 $\pm$ 0,62	p < 0.001
MA (n/hour)	0,02 $\pm$ 0,06	0,01 $\pm$ 0,03	0,03 $\pm$ 0,07	p < 0.001
CA (n/hour)	0,39 $\pm$ 0,55	0,35 $\pm$ 0,46	0,44 $\pm$ 0,63	ns

Parameter	ALL	Subgroup 1 (low snore train percentage) $\leq 1,3$ (median)	(high snore train percentage) $> 1,3$ (median) Kolumna3	Kolumna4p
	Average	Average	Average	
HI (n/hour)	2,01 $\pm$ 2,12	1,37 $\pm$ 1,68	2,7 $\pm$ 2,34	p < 0.001
Mean SpO2 (%)	94,87 $\pm$ 1,37	95,16 $\pm$ 1,12	94,54 $\pm$ 1,54	p < 0.001
min. sat.	87,33 $\pm$ 7,79	88,76 $\pm$ 6,89	85,76 $\pm$ 8,45	p < 0.001
SpO2 Duration < 90%	1,53 $\pm$ 4,01	1,23 $\pm$ 3,1	1,85 $\pm$ 4,83	p < 0.05
Aver. desat.	3,16 $\pm$ 0,69	2,96 $\pm$ 0,77	3,38 $\pm$ 0,52	p < 0.001
Arousals (n/hour)	4,63 $\pm$ 5,37	4,88 $\pm$ 7,06	4,36 $\pm$ 2,43	ns
Pulse aver. n/hour)	63,19 $\pm$ 9,55	62,34 $\pm$ 8,71	64,12 $\pm$ 10,37	ns
Pulse max. n/hour)	100,81 $\pm$ 19,7	98,1 $\pm$ 13,26	103,77 $\pm$ 24,7	ns
Pulse min. n/hour)	49,63 $\pm$ 9,14	49,34 $\pm$ 8,49	49,94 $\pm$ 9,85	ns

TST, total sleep time; SE, sleep efficiency; WASO, wake after sleep onset; SL, sleep latency; N1, stage N1 sleep; N2, stage N2 sleep; N3, stage N3 sleep; REM, rapid eye movement sleep; AHI, apnea–hypopnea index; ODI, oxygen desaturation index; AI, apnea index; OA, obstructive apnea index; MA, mixed apnea index; CA, central apnea index; HI, hypopnea index; SpO₂, oxygen saturation; Mean SpO₂, mean oxygen saturation; Min. sat., minimum oxygen saturation; SpO₂ duration < 90%, duration of oxygen saturation below 90%; Aver. desat., average desaturation; Arousals, arousal index; Pulse aver., average pulse rate; Pulse max., maximum pulse rate; Pulse min., minimum pulse rate; TST, total sleep time.

When participants were divided into four subgroups based on snore train percentage, a progressive increase in respiratory disturbance parameters was observed. The highest values of AHI (3.96 ± 2.71), ODI (4.40 ± 2.93), HI (3.31 ± 2.76), and snoring percentage (24.62 ± 15.95) were observed in the subgroup with the highest snore train percentage. Significant Differences were primarily observed between the lowest- and highest-percentage subgroups.

Similarly, mean oxygen saturation (94.15 ± 1.73) and minimum oxygen saturation (85.32 ± 8.79) were lowest in the subgroup with the highest snore train percentage, and significant differences were observed mainly between the extreme subgroups.

No statistically significant differences were found in sleep architecture parameters across the four subgroups. Detailed results for the remaining comparisons are presented in Table 4.

Table 4

Polysomnographic parameters of patients with primary snoring based on the snore train percentage.
Classification based on quartiles

Parameter	ALL	Subgroup 1 (irrelevant snore train %): $\leq 0,1$ (Q1)	Subgroup 2 (mild snore train %): 0,2–1,3 (Q2)	Subgroup 3 (moderate snore train %): 1,4–9,7 (Q3)	Subgroup 4 (severe snore train %): $>9,7$ (G4)	p
	Average	Average	Average	Average	Average	
TST (min)	441,35 $\pm 51,22$	435,43 $\pm$ 55,84	447,73 $\pm$ 49,11	454,31 $\pm$ 41,59	431,95 $\pm$ 53,44	ns
SE (%)	86,63 $\pm$ 11,2	85,99 $\pm$ 10,43	88,32 $\pm$ 6,59	88,01 $\pm$ 10,75	84,83 $\pm$ 14,98	ns
WASO (min)	39,4 $\pm$ 44,86	38,37 $\pm$ 37,6	31,76 $\pm$ 25,61	31,09 $\pm$ 20,62	54,58 $\pm$ 71,8	ns
SL (min)	19,81 $\pm$ 16,73	20,64 $\pm$ 15,06	19,62 $\pm$ 17,14	18,68 $\pm$ 15,88	19,93 $\pm$ 19,6	ns
N1 (% of TST)	4,53 $\pm$ 4,41	3,61 $\pm$ 2,29	5,7 $\pm$ 7,68	4,69 $\pm$ 3,73	4,61 $\pm$ 3,23	ns
N2 (% of TST)	49,06 $\pm$ 9,25	48,04 $\pm$ 10,89	48,52 $\pm$ 7,35	47,87 $\pm$ 10,45	51,91 $\pm$ 6,46	ns
N3 (% of TST)	24,07 $\pm$ 7,78	25,04 $\pm$ 9,71	24,65 $\pm$ 6,57	23,97 $\pm$ 7,09	22,42 $\pm$ 6,37	ns
REM (% of TST)	22,6 $\pm$ 6,31	23,31 $\pm$ 5,34	22,36 $\pm$ 5,8	23,46 $\pm$ 7,67	21,08 $\pm$ 6,45	ns
AHI (n/hour)	2,64 $\pm$ 2,28	1,43 $\pm$ 1,41	2,64 $\pm$ 2,21	2,9 $\pm$ 2	3,96 $\pm$ 2,71	A1-A2: <0.05 A1-A3: <0.001 A1:A4: <0.001
ODI (n/hour)	3,02 $\pm$ 2,59	1,78 $\pm$ 1,57	2,69 $\pm$ 2,12	3,58 $\pm$ 2,92	4,4 $\pm$ 2,93	A1-A3: <0.001 A1:A4: <0.001
Snore (% of TST)	7,59 $\pm$ 12,9	0,52 $\pm$ 2,12	0,48 $\pm$ 0,36	5,32 $\pm$ 3,69	24,62 $\pm$ 15,95	A1-A2: <0.001 A1-A3: <0.001 A1:A4: <0.001

Parameter	ALL	Subgroup 1 (irrelevant snore train %): ≤0,1 (Q1)	Subgroup 2 (mild snore train %): 0,2–1,3 (Q2)	Subgroup 3 (moderate snore train %): 1,4–9,7 (Q3)	Subgroup 4 (severe snore train %): >9,7 (G4)	p
	Average	Average	Average	Average	Average	
						A2-A3: <0.001 A2-A4: <0.001 A3-A4: <0.001
AI (n/hour)	0,69 ± 0,81	0,44 ± 0,53	0,65 ± 0,64	0,86 ± 0,91	0,89 ± 1,03	ns
OA (n/hour)	0,27 ± 0,48	0,13 ± 0,27	0,21 ± 0,24	0,31 ± 0,47	0,47 ± 0,72	A1:A4: <0.001
MA (n/hour)	0,02 ± 0,06	0,01 ± 0,04	0 ± 0	0,03 ± 0,08	0,04 ± 0,07	ns
CA (n/hour)	0,39 ± 0,55	0,29 ± 0,4	0,43 ± 0,53	0,51 ± 0,69	0,37 ± 0,57	ns
HI (n/hour)	2,01 ± 2,12	0,99 ± 1,14	1,98 ± 2,18	2,04 ± 1,57	3,31 ± 2,76	A1-A3: <0.001 A1:A4: <0.001
Mean SpO2 (%)	94,87 ± 1,37	95,11 ± 1,12	95,25 ± 1,14	94,96 ± 1,2	94,15 ± 1,73	A1:A4: <0.05 A2-A4: <0.05
min. sat.	87,33 ± 7,79	88,94 ± 6,84	88,47 ± 7,08	86,24 ± 8,17	85,32 ± 8,79	A1:A4: <0.01
SpO2 Duration < 90%	1,53 ± 4,01	1,62 ± 3,66	0,61 ± 1,77	2,51 ± 5,2	1,24 ± 4,45	ns
Aver. desat.	3,16 ± 0,69	2,89 ± 0,94	3,09 ± 0,35	3,44 ± 0,64	3,32 ± 0,39	A1-A3: <0.001 A2-A3: <0.05
Arousals (n/hour)	4,63 ± 5,37	5,22 ± 8,7	4,35 ± 3	4,5 ± 2,75	4,23 ± 2,13	ns

Parameter	ALL	Subgroup 1 (irrelevant snore train %): $\leq 0,1$ (Q1)	Subgroup 2 (mild snore train %): 0,2–1,3 (Q2)	Subgroup 3 (moderate snore train %): 1,4–9,7 (Q3)	Subgroup 4 (severe snore train %): $>9,7$ (G4)	<i>p</i>
	Average	Average	Average	Average	Average	
Pulse aver. n/hour)	63,19 $\pm$ 9,55	63,12 $\pm$ 9,82	61,09 $\pm$ 6,52	63,24 $\pm$ 11,21	64,92 $\pm$ 9,62	ns
Pulse max. n/hour)	100,81 $\pm$ 19,7	100,21 $\pm$ 14,57	94,73 $\pm$ 10,17	101,21 $\pm$ 22,78	106,14 $\pm$ 26,43	ns
Pulse min. n/hour)	49,63 $\pm$ 9,14	49,03 $\pm$ 10,02	49,83 $\pm$ 5,29	48,79 $\pm$ 11,18	51 $\pm$ 8,47	ns

TST, total sleep time; SE, sleep efficiency; WASO, wake after sleep onset; SL, sleep latency; N1, stage N1 sleep; N2, stage N2 sleep; N3, stage N3 sleep; REM, rapid eye movement sleep; AHI, apnea–hypopnea index; ODI, oxygen desaturation index; AI, apnea index; OA, obstructive apnea index; MA, mixed apnea index; CA, central apnea index; HI, hypopnea index; SpO₂, oxygen saturation; Mean SpO₂, mean oxygen saturation; Min. sat., minimum oxygen saturation; SpO₂ duration < 90%, duration of oxygen saturation below 90%; Aver. desat., average desaturation; Arousals, arousal index; Pulse aver., average pulse rate; Pulse max., maximum pulse rate; Pulse min., minimum pulse rate; TST, total sleep time.

Analysis based on the number of snore trains per hour showed that the high-frequency subgroup had significantly higher values of AHI (3.68 ± 2.48 vs. 1.58 ± 1.41), ODI (4.08 ± 2.93 vs. 1.94 ± 1.63), OA (0.39 ± 0.61 vs. 0.16 ± 0.26), MA (0.03 ± 0.07 vs. 0.01 ± 0.03), and HI (2.89 ± 2.44 vs. 1.08 ± 1.13) (all $p < 0.001$). The proportion of N3 sleep was also significantly lower in this group (23.03 ± 6.63 vs. 25.08 ± 8.73 ; $p < 0.05$).

Mean oxygen saturation (94.51 ± 1.53 vs. 95.20 ± 1.11) and minimum oxygen saturation (86.10 ± 7.83 vs. 88.55 ± 7.67) were significantly lower in the high-frequency subgroup (both $p < 0.001$). Additionally, average desaturation was significantly higher (3.29 ± 0.37 vs. 3.04 ± 0.89 ; $p < 0.05$). Detailed results for the remaining comparisons are shown in Table 5.

Table 5

Polysomnographic parameters of patients with primary snoring based on the number of snore trains per hour. Classification based on the median.

Parameter	ALL	Subgroup 1 (low number of snore trains per hour) $\leq 1,2$ (mediana)	Subgroup 2 (high number of snore trains per hour) $> 1,2$ (mediana)	p
	Average	Average	Average	
TST (min)	441,35 $\pm$ 51,22	440,31 $\pm$ 53,46	442,82 $\pm$ 49,4	ns
SE (%)	86,63 $\pm$ 11,2	86,79 $\pm$ 9,43	86,5 $\pm$ 12,89	ns
WASO (min)	39,4 $\pm$ 44,86	36,07 $\pm$ 34,03	43,08 $\pm$ 54	ns
SL (min)	19,81 $\pm$ 16,73	20,38 $\pm$ 15,66	18,74 $\pm$ 17,44	ns
N1 (% of TST)	4,53 $\pm$ 4,41	4,38 $\pm$ 5	4,73 $\pm$ 3,73	ns
N2 (% of TST)	49,06 $\pm$ 9,25	47,97 $\pm$ 10,33	50,17 $\pm$ 7,97	ns
N3 (% of TST)	24,07 $\pm$ 7,78	25,08 $\pm$ 8,73	23,03 $\pm$ 6,63	p < 0.05
REM (% of TST)	22,6 $\pm$ 6,31	23,06 $\pm$ 6,04	22,08 $\pm$ 6,61	ns
AHI (n/hour)	2,64 $\pm$ 2,28	1,58 $\pm$ 1,41	3,68 $\pm$ 2,48	p < 0.001
ODI (n/hour)	3,02 $\pm$ 2,59	1,94 $\pm$ 1,63	4,08 $\pm$ 2,93	p < 0.001
Snore (% of TST)	7,59 $\pm$ 12,9	0,65 $\pm$ 1,88	14,83 $\pm$ 15,31	p < 0.001
AI (n/hour)	0,69 $\pm$ 0,81	0,49 $\pm$ 0,59	0,91 $\pm$ 0,94	
OA (n/hour)	0,27 $\pm$ 0,48	0,16 $\pm$ 0,26	0,39 $\pm$ 0,61	p < 0.001
MA (n/hour)	0,02 $\pm$ 0,06	0,01 $\pm$ 0,03	0,03 $\pm$ 0,07	p < 0.001
CA (n/hour)	0,39 $\pm$ 0,55	0,32 $\pm$ 0,46	0,47 $\pm$ 0,62	
HI (n/hour)	2,01 $\pm$ 2,12	1,08 $\pm$ 1,13	2,89 $\pm$ 2,44	p < 0.001

Parameter	ALL	Subgroup 1 (low number of snore trains per hour) $\leq 1,2$ (mediana)	Subgroup 2 (high number of snore trains per hour) $> 1,2$ (mediana)	p
	Average	Average	Average	
Mean SpO ₂ (%)	94,87 $\pm$ 1,37	95,2 $\pm$ 1,11	94,51 $\pm$ 1,53	p < 0.001
min. sat.	87,33 $\pm$ 7,79	88,55 $\pm$ 7,67	86,1 $\pm$ 7,83	p < 0.001
SpO ₂ Duration < 90%	1,53 $\pm$ 4,01	1,53 $\pm$ 3,8	1,55 $\pm$ 4,27	p < 0.05
Aver. desat.	3,16 $\pm$ 0,69	3,04 $\pm$ 0,89	3,29 $\pm$ 0,37	p < 0.05
Arousals (n/hour)	4,63 $\pm$ 5,37	4,7 $\pm$ 7,11	4,58 $\pm$ 2,69	ns
Pulse aver. n/hour)	63,19 $\pm$ 9,55	62,13 $\pm$ 8,91	64,45 $\pm$ 10,04	ns
Pulse max. n/hour)	100,81 $\pm$ 19,7	97,87 $\pm$ 13,62	104,04 $\pm$ 24,17	ns
Pulse min. n/hour)	49,63 $\pm$ 9,14	49,18 $\pm$ 8,59	50,2 $\pm$ 9,7	ns

TST, total sleep time; SE, sleep efficiency; WASO, wake after sleep onset; SL, sleep latency; N1, stage N1 sleep; N2, stage N2 sleep; N3, stage N3 sleep; REM, rapid eye movement sleep; AHI, apnea–hypopnea index; ODI, oxygen desaturation index; AI, apnea index; OA, obstructive apnea index; MA, mixed apnea index; CA, central apnea index; HI, hypopnea index; SpO₂, oxygen saturation; Mean SpO₂, mean oxygen saturation; Min. sat., minimum oxygen saturation; SpO₂ duration < 90%, duration of oxygen saturation below 90%; Aver. desat., average desaturation; Arousals, arousal index; Pulse aver., average pulse rate; Pulse max., maximum pulse rate; Pulse min., minimum pulse rate; TST, total sleep time.

The study participants were subsequently divided into four subgroups based on the number of snoring episodes per hour. No significant differences were observed in most sleep architecture parameters. However, significant differences in WASO were found between subgroups 1 and 2 (44.77 $\pm$ 40.39 vs. 27.61 $\pm$ 24.06; p < 0.05) and between subgroups 2 and 4 (27.61 $\pm$ 24.06 vs. 53.34 $\pm$ 71.58; p < 0.001).

Respiratory parameters increased progressively with snore train frequency. Significantly higher AHI, AI, OA, and HI values were observed in subgroups 3 and 4 compared with subgroup 1, with the highest values in subgroup 4. Similarly, MA values were significantly higher in subgroup 4 compared to subgroups 1 and 2.

Regarding oxygenation, the minimum oxygen saturation was significantly lower, whereas the average desaturation was significantly higher in subgroup 4 than in subgroup 1. Detailed results are presented in Table 6.

Table 6

Polysomnographic parameters of patients with primary snoring based on the number of snore trains per hour. Classification based on quartiles

Parameter	ALL	Subgroup 1 (irrelevant snore trains/hour): ≤0,275 (Q1)	Subgroup 2 (mild snore trains/hour): 0,2–1,3 (Q2)	Subgroup 3 (moderate snore trains/hour): 1,21–6,6 (Q3)	Subgroup 4 (severe snore trains/hour): >6,6 (G4)	p
	Average	Average	Average	Average	Average	
TST (min)	441,35 ± 51,22	430,65 ± 58,07	449,72 ± 47,45	447,17 ± 44,25	438,34 ± 54,45	ns
SE (%)	86,63 ± 11,2	85,56 ± 10,94	87,98 ± 7,63	88,84 ± 8,02	84,11 ± 16,25	ns
WASO (min)	39,4 ± 44,86	44,77 ± 40,39	27,61 ± 24,06	33,11 ± 25,35	53,34 ± 71,58	A1-A2: <0.05 A2-A4: <0.001
SL (min)	19,81 ± 16,73	20,9 ± 15,71	19,87 ± 15,8	19,41 ± 18,86	18,06 ± 16,09	ns
N1 (% of TST)	4,53 ± 4,41	3,58 ± 2,43	5,15 ± 6,57	4,51 ± 3,82	4,96 ± 3,68	ns
N2 (% of TST)	49,06 ± 9,25	49,45 ± 10,45	46,53 ± 10,14	48,59 ± 8,84	51,79 ± 6,72	ns
N3 (% of TST)	24,07 ± 7,78	24,1 ± 9,01	26,04 ± 8,45	23,51 ± 7,29	22,54 ± 5,95	ns
REM (% of TST)	22,6 ± 6,31	22,86 ± 4,9	23,26 ± 7,04	23,41 ± 6,29	20,72 ± 6,73	ns
AHI (n/hour)	2,64 ± 2,28	1,22 ± 1,23	1,92 ± 1,5	3,24 ± 2,08	4,13 ± 2,8	A1-A3: <0.001 A1:A4: <0.001 A2-A3: <0.05 A2-A4: <0.01
ODI (n/hour)	3,02 ± 2,59	1,65 ± 1,4	2,23 ± 1,81	3,7 ± 2,85	4,47 ± 2,99	ns
Snore (% of TST)	7,59 ± 12,9	0,3 ± 1,63	0,99 ± 2,07	5,53 ± 4,77	24,39 ± 16,54	A1-A2: <0.001

Parameter	ALL	Subroup 1 (irrelevant snore trains/hour): ≤0,275 (Q1)	Subgrip2 (mild snore trains/hour): 0,2–1,3 (Q2)	Subgrup 3 (moderate snore trains/hour): 1,21–6,6 (Q3)	Subgrup 4 (severe snore trains/hour): >6,6 (G4)	<i>p</i>
	Average	Average	Average	Average	Average	
						A1-A3: <0.001 A1:A4: <0.001 A2-A3: <0.001 A2-A4: <0.001 A3-A4: <0.001
AI (n/hour)	0,69 ± 0,81	0,36 ± 0,44	0,62 ± 0,69	0,75 ± 0,67	1,06 ± 1,15	A1-A3: <0.05 A1:A4: <0.001
OA (n/hour)	0,27 ± 0,48	0,09 ± 0,2	0,22 ± 0,3	0,29 ± 0,42	0,51 ± 0,74	A1-A3: <0.05 A1:A4: <0.001
MA (n/hour)	0,02 ± 0,06	0,01 ± 0,04	0,01 ± 0,02	0,03 ± 0,08	0,03 ± 0,07	A1:A4: <0.05 A2-A4: <0.05
CA (n/hour)	0,39 ± 0,55	0,26 ± 0,36	0,38 ± 0,53	0,43 ± 0,53	0,5 ± 0,71	ns
HI (n/hour)	2,01 ± 2,12	0,87 ± 0,99	1,29 ± 1,24	2,48 ± 2,03	3,31 ± 2,77	A1-A3: <0.001 A1:A4: <0.001 A2-A3: <0.01 A2-A4: <0.01
Mean SpO2 (%)	94,87 ± 1,37	95,17 ± 1,17	95,23 ± 1,06	94,75 ± 1,26	94,27 ± 1,76	ns

Parameter	ALL	Subgroup 1 (irrelevant snore trains/hour): ≤0,275 (Q1)	Subgroup 2 (mild snore trains/hour): 0,2–1,3 (Q2)	Subgroup 3 (moderate snore trains/hour): 1,21–6,6 (Q3)	Subgroup 4 (severe snore trains/hour): >6,6 (G4)	p
	Average	Average	Average	Average	Average	
min. sat.	87,33 ± 7,79	89,08 ± 7,01	88,03 ± 8,32	86,73 ± 6,79	85,44 ± 8,83	A1:A4: <0.05
SpO2 Duration < 90%	1,53 ± 4,01	1,4 ± 3,23	1,65 ± 4,33	1,72 ± 4,06	1,36 ± 4,53	ns
Aver. desat.	3,16 ± 0,69	2,78 ± 1,01	3,28 ± 0,68	3,27 ± 0,32	3,31 ± 0,43	A1-A2: <0.05 A1-A3: <0.05 A1:A4: <0.05
Arousals (n/hour)	4,63 ± 5,37	3,76 ± 2,45	5,61 ± 9,67	4,5 ± 3,1	4,67 ± 2,23	ns
Pulse aver. n/hour)	63,19 ± 9,55	63,22 ± 9,82	61,07 ± 7,91	63,72 ± 11,32	65,19 ± 8,63	ns
Pulse max. n/hour)	100,81 ± 19,7	101,11 ± 14,65	94,71 ± 11,89	102,97 ± 24,4	105,14 ± 24,22	ns
Pulse min. n/hour)	49,63 ± 9,14	48,53 ± 10,59	49,82 ± 6,13	49,21 ± 10,87	51,22 ± 8,38	ns

TST, total sleep time; SE, sleep efficiency; WASO, wake after sleep onset; SL, sleep latency; N1, stage N1 sleep; N2, stage N2 sleep; N3, stage N3 sleep; REM, rapid eye movement sleep; AHI, apnea–hypopnea index; ODI, oxygen desaturation index; AI, apnea index; OA, obstructive apnea index; MA, mixed apnea index; CA, central apnea index; HI, hypopnea index; SpO₂, oxygen saturation; Mean SpO₂, mean oxygen saturation; Min. sat., minimum oxygen saturation; SpO₂ duration < 90%, duration of oxygen saturation below 90%; Aver. desat., average desaturation; Arousals, arousal index; Pulse aver., average pulse rate; Pulse max., maximum pulse rate; Pulse min., minimum pulse rate; TST, total sleep time.

Next, correlation analyses were conducted. All snoring indices showed positive correlations with sleep-disordered breathing severity (AHI, ODI, HI), indicating that increased snoring is associated with more frequent apnea and hypopnea events. Negative correlations with mean oxygen saturation (Mean SpO₂) indicate poorer oxygenation with a higher snoring burden. Higher snoring indices were also associated

with a greater proportion of N2 sleep and a reduced proportion of deep sleep (N3). Detailed results are presented in Table 7.

Table 7

Correlation results between parameters defining the severity of primary snoring and polysomnographic parameters.

Parameter	Snore index	Snore Train Percentage	Number of snore trains/hour
TST (min)	ns	ns	ns
SE (%)	ns	ns	ns
WASO (min)	ns	ns	ns
SL (min)	ns	ns	ns
N1 (% of TST)	ns	ns	ns
N2 (% TST)	0.20 (0.014)	0.20 (0.015)	0.16 (0.046)
N3 (% TST)	-0.16 (0.046)	-0.17 (0.040)	NS
REM (% of TST)	ns	ns	ns
AHI (n/h)	0.45 (< 0.001)	0.44 (< 0.001)	0.48 (< 0.001)
ODI (n/h)	0.41 (< 0.001)	0.40 (< 0.001)	0.43 (< 0.001)
Snore (% TST)	0.94 (< 0.001)	0.95 (< 0.001)	0.82 (< 0.001)
AI (n/h)	ns	ns	0.25 (0.003)
OA (n/h)	0.19 (0.024)	0.19 (0.023)	0.25 (0.002)
HI (n/h)	0.45 (< 0.001)	0.45 (< 0.001)	0.44 (< 0.001)
Mean SpO ₂ (%)	-0.28 (< 0.001)	-0.28 (0.001)	-0.25 (0.003)
Average desaturation	0.20 (0.017)	0.18 (0.030)	ns
Average pulse (n/h)	ns	ns	0.21 (0.011)
TST, total sleep time; SE, sleep efficiency; WASO, wake after sleep onset; SL, sleep latency; N1, stage N1 sleep; N2, stage N2 sleep; N3, stage N3 sleep; REM, rapid eye movement sleep; AHI, apnea–hypopnea index; ODI, oxygen desaturation index; AI, apnea index; OA, obstructive apnea index; MA, mixed apnea index; SpO ₂ , oxygen saturation; Mean SpO ₂ , mean oxygen saturation;			

4. Discussion

Primary snoring is best conceptualized as part of a continuum of sleep-disordered breathing rather than a benign or purely social phenomenon. Contemporary viewpoints emphasize that “primary” snoring, defined by the absence of diagnostic obstructive sleep apnea based on conventional apnea-hypopnea

index thresholds, remains an area with unresolved biological and clinical implications rather than a settled null category [33]. Objective, multi-night community data further support the premise that snoring exposure can be associated with adverse cardiovascular phenotypes independently of sleep apnea burden[34]. Complementary vascular imaging data also suggest that primary snoring may relate to structural vascular remodeling in settings without diagnosed obstructive sleep apnea, raising the possibility that the pathobiology of snoring may involve vibration-related or hemodynamic mechanisms[35].

Against this background, an altered distribution of sleep stages with reduced slow-wave sleep N3, reduced rapid eye movement sleep, and increased wake after sleep onset is clinically meaningful because it implies a shift away from consolidated and potentially restorative sleep. This pattern is consistent with a model in which repetitive subclinical respiratory loading and upper airway narrowing destabilize sleep-state regulation, even when the respiratory event rate remains below the diagnostic thresholds for obstructive sleep apnea. Evidence on sleep architecture, specifically in primary snoring, is limited and inconsistent across published cohorts, underscoring the importance of careful interpretation given confounding and definitional heterogeneity. For example, polysomnography-based clinical datasets comparing controls, primary snoring, and obstructive sleep apnea have reported that major architecture shifts may be attenuated after adjustment for age and body mass index, suggesting that cohort composition and residual confounding can materially influence conclusions[36].

Large polysomnography-derived analyses that explicitly included a primary snoring group have shown that arousal burden relates to hypertension risk in obstructive sleep apnea, but not in primary snorers, supporting the concept that arousal frequency may not be the dominant pathway linking nonapneic snoring to downstream risk[37]. In this context, observing architecture shifts without parallel increases in arousal indices may indicate that the regulatory cost of snoring-related respiratory loading can manifest as stage redistribution and wakefulness after sleep onset rather than as overt arousal escalation.

Mechanistically, the most plausible link between primary snoring and altered sleep architecture involves events that fall below traditional scoring thresholds, including subtle obstructive respiratory events, inspiratory flow limitation, and changes in respiratory effort that can perturb sleep physiology without meeting criteria for apnea, hypopnea, desaturation, or cortical arousal. Physiologic studies demonstrate that nonapneic respiratory events without desaturation or cortical arousal can still produce measurable increases in airway resistance and consistent autonomic consequences, as reflected in heart rate dynamics, suggesting that “silent” respiratory loading is not physiologically inert [38]. This supports an interpretation in which sleep-stage redistribution may be driven by repetitive autonomic perturbations, altered ventilatory mechanics, and increased work of breathing, which do not necessarily translate into higher-scoring arousal indices. In parallel, reviews describing prolonged partial upper airway obstruction highlight that sustained flow limitation with crescendo snoring can occur with relatively fewer arousals than classic cyclic obstructive sleep apnea patterns, and that such phenotypes may be underrecognized when analysis is anchored mainly to event counts rather than to the time burden and physiology of

partial obstruction[39]. These frameworks provide a coherent explanation for why altered architecture and wakefulness after sleep onset can coexist with stable arousal indices in primary snoring.

In the present study, we demonstrate for the first time that primary snoring, in the absence of polysomnographic criteria for obstructive sleep apnea, is associated not only with sleep fragmentation but also with a significant reduction in the proportion of REM sleep and N3 sleep. Previous polysomnographic studies in primary snorers have mainly reported increased arousal indices, frequent microarousals, and reduced sleep efficiency, whereas changes in sleep-stage distribution were inconsistent or not detected at all, as reported in early observational and laboratory-based studies focusing on sleep continuity rather than sleep macrostructure [5].

The reduction in N3 sleep observed in our cohort may have important physiological consequences. Slow-wave sleep is a key component of restorative sleep and is involved in energy conservation, synaptic downscaling, and neuroendocrine regulation, including growth hormone secretion and autonomic balance[41]. Experimental data indicate that selective suppression of slow-wave sleep impairs metabolic and autonomic regulation. Tasali et al. demonstrated that reduction of N3 sleep is associated with decreased insulin sensitivity and adverse metabolic effects, underscoring the systemic relevance of this sleep stage[42]. In the context of primary snoring, our findings suggest that repetitive vibratory stimuli and increased upper airway resistance may interfere with the generation or maintenance of deep sleep, even in the absence of apneas or significant oxygen desaturation.

While several studies have reported an association between primary snoring and the proportion of N3 sleep, to the best of our knowledge, this is the first study to identify a relationship between primary snoring and the proportion of REM sleep. Concomitantly, the reduction in REM sleep observed in this study points toward potential neurocognitive and emotional consequences. REM sleep plays a central role in memory consolidation, especially emotional and procedural memory, and in emotional regulation [43], [44]. Limitation of REM sleep has also been linked to altered central thermoregulation due to the physiological suspension of normal thermoregulatory responses during this sleep stage [41]. Chronic REM sleep reduction may therefore contribute to impaired cognitive performance, emotional dysregulation, and increased stress reactivity. This may help explain why some individuals with primary snoring report daytime cognitive complaints and non-restorative sleep despite normal apnea–hypopnea indices.

The clinical relevance of intermittent hypoxia lies in its established links to vascular and cardiometabolic risk pathways in obstructive sleep apnea, but extrapolation to primary snoring requires restraint. In obstructive sleep apnea, intermittent hypoxia is implicated in oxidative stress biology, endothelial dysfunction, and sympathetic activation, and these pathways are central in mechanistic accounts of sleep-disordered breathing-related cardiovascular risk[45]. Experimental and translational evidence also suggests that intermittent hypoxia can activate endothelial inflammatory pathways through sympathetic activation and catecholamine signaling, providing biologically plausible links between nocturnal respiratory stressors and vascular dysfunction[46]. However, the magnitude, duration, and patterning of

hypoxemia typical of primary snoring likely differ from those of moderate-to-severe obstructive sleep apnea, and there is a paucity of longitudinal evidence directly testing whether low-amplitude intermittent hypoxia, as may occur in primary snoring, produces similar long-term vascular consequences. This evidentiary gap limits the strength of inference, and it supports a future research agenda focused on hypoxemic burden metrics, vascular biomarkers, and longitudinal outcomes in carefully phenotyped primary snoring cohorts rather than direct transfer of obstructive sleep apnea risk models.

Methodological strengths and limitations should be carefully weighed because they influence the robustness of the identified architecture and oxygenation signals. The lack of an adaptation night raises the possibility of a first-night effect, which is known to increase wake after sleep onset and reduce deep NREM sleep in laboratory-recorded sleep, and can therefore bias absolute sleep architecture estimates[47]. Even if this effect is non-differential across groups, it may still widen variance and obscure or inflate between-group differences depending on susceptibility. Counterbalancing this limitation, the use of full attended polysomnography is a major strength because it provides comprehensive staging, arousal scoring, respiratory event characterization, and oxygenation assessment in a single synchronized framework, which is essential when the hypothesized phenotype involves subtle interactions among sleep stages, breathing mechanics, and oxygenation rather than a single screening metric[36].

5. Conclusions

Primary snoring is associated with altered sleep architecture and nocturnal hypoxia, which may represent potential mechanisms linking snoring to sympathetic activation, elevated blood pressure, and increased cardiovascular risk. Further prospective studies are needed to evaluate the long-term clinical impact of alterations in sleep architecture and low-grade intermittent hypoxia in individuals with primary snoring.

Declarations

Author Contributions:

Validation, P.G.; formal analysis, P.G.; investigation, H.M., P.M., P.J., M.F., A.J. ; resources, P.M.; data curation, P.M.; methodology, H.M.,M.W.; writing—original draft preparation, H.M., P.M.; writing - review and editing, N.D., B.C., M.W., W.T.,; visualization, P.M.; supervision, H.M.. All authors have read and agreed to the published version of the manuscript

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Institutional Review Board Statement: The study was approved by the local Ethics Committee (no. KB-305/2025) and was conducted by adopting the principles of the Declaration of Helsinki

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Data availability: The datasets generated and/or analyzed during the current study are not publicly available because they contain sensitive clinical and polysomnographic data that could potentially compromise participant privacy. However, the datasets are available from the corresponding author upon reasonable request, subject to applicable ethical and institutional requirements.

Conflicts of Interest: The authors declare no conflict of interest

Informed Consent Statement: It was a retrospective study.

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Figures

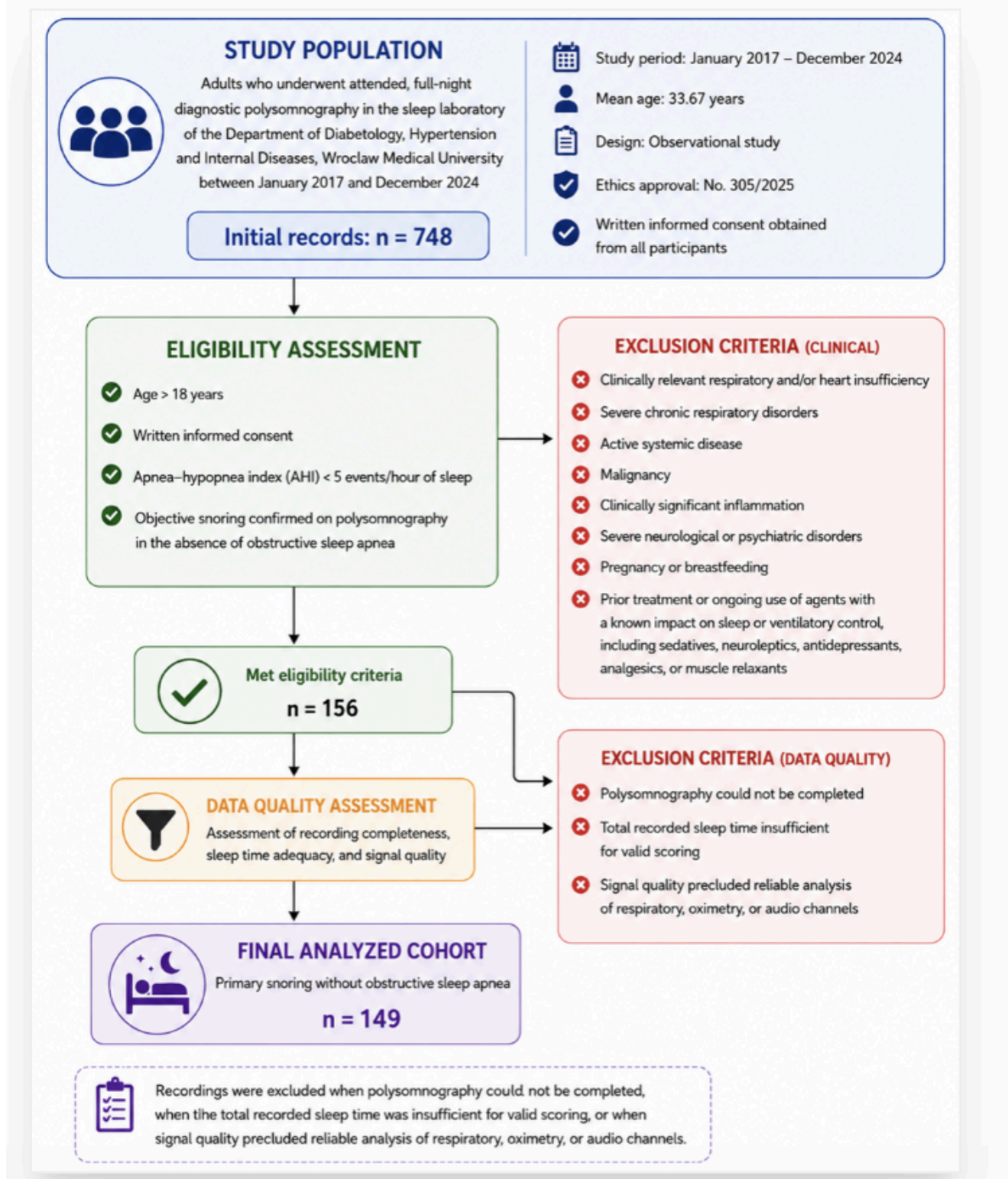


Figure 1

Unnumbered image in the Materials & Methods section.