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Hypoxic Burden Phenotyping in Level 3 Home Sleep Apnoea Testing

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Abstract*—Background:** *The apnoea-hypopnoea index remains the dominant metric for obstructive sleep apnoea severity classification despite well-documented limitations in predicting cardiovascular outcomes. Hypoxic burden, defined as the area under the oxygen saturation desaturation curve normalised to sleep time, has emerged as a more cardiovascularly relevant biomarker.* ***Objective*: To characterise hypoxic burden and its relationship with conventional obstructive sleep apnoea severity metrics in a consecutive clinical cohort tested under the FASTNAP protocol using Level 3 home sleep apnoea testing.* ***Methods****: Thirteen adults (8 male, 5 female; mean age 46.5 years) underwent Level 3 home sleep apnoea testing between February and October 2025. Extracted parameters included the apnoea-hypopnoea index, rapid eye movement apnoea-hypopnoea index, oxygen desaturation index, arousal index, minimum oxygen saturation, mean oxygen saturation, time below 90% oxygen saturation, and hypoxic burden. Obstructive sleep apnoea severity was classified by American Academy of Sleep Medicine 2023 criteria.* ***Results****: Obstructive sleep apnoea was absent in 2 patients (15.4%), mild in 4 (30.8%), moderate in 2 (15.4%), and severe in 5 (38.5%). Minimum oxygen saturation ranged from 61% to 94%; time below 90% oxygen saturation ranged from 0% to 87.7%. Marked dissociation was observed between the apnoea-hypopnoea index severity and hypoxaemic burden: patients with mild apnoea-hypopnoea index demonstrated time below 90% oxygen saturation of up to 17% and minimum oxygen saturation of 78%, while a severe case showed time below 90% oxygen saturation of only 0.9%. Four distinct dissociation phenotypes were identified.* ***Conclusions****: Apnoea-hypopnoea index classification systematically misrepresents hypoxic exposure. A multi-parameter oximetric profile incorporating hypoxic burden, oxygen desaturation index, time below 90% oxygen saturation, and minimum oxygen saturation should be mandated in Level 3 home sleep apnoea testing reporting.*

***Keywords*—** *Obstructive sleep apnoea; hypoxic burden; oxygen desaturation index; home sleep testing; oximetric phenotyping.*

I. Introduction

The apnoea-hypopnoea index (AHI) has anchored obstructive sleep apnoea (OSA) diagnosis and severity stratification for over four decades. Despite this, the index counts respiratory events without weighting for their haemodynamic consequences. Two patients with identical AHI values can experience vastly different degrees of nocturnal hypoxaemia depending on event duration, desaturation depth, and the proportion of sleep below physiologically critical oxygen thresholds [1,2].

Large prospective studies — including the Osteoporotic Fractures in Men (MrOS) Sleep Study, the Multi-Ethnic Study of Atherosclerosis (MESA) Sleep Study, and the Sleep Heart Health Study (SHHS) — have consistently demonstrated that hypoxic burden (HB), defined as the cumulative area under the oxygen saturation desaturation curve normalised to sleep time (expressed in minutes per hour), independently predicts all-cause and cardiovascular mortality beyond AHI [3,4,5]. The mechanistic basis for this observation lies in the dose-dependent relationship between hypoxic exposure duration and sympathetic activation, endothelial injury, and systemic inflammatory cytokine release [6].

Level 3 home sleep apnoea testing (HSAT) records airflow, respiratory effort, and peripheral oxygen saturation without electroencephalography-based sleep staging, providing an accessible platform for real-world oximetric phenotyping. The FASTNAP protocol, adopted at our centre, employs a standardised ambulatory HSAT workflow using the Dormir Bien platform enabling automated generation of multi-parameter oximetric reports including HB categorisation.

The objectives of this study were: (a) to characterise the distribution of AHI, oxygen desaturation index (ODI), time below 90% oxygen saturation (T90), and HB in a consecutive clinical referral population; (b) to examine concordance and discordance between AHI-based severity classification and oximetric burden; and (c) to identify oximetric phenotypes carrying disproportionate cardiovascular risk relative to their AHI classification.

II. Methods

Study Design and Setting

This retrospective cross-sectional analysis included consecutive patients who underwent Level 3 HSAT at a tertiary-care sleep laboratory (Department of Physiology, Sri Aurobindo Medical College and PGI, Indore) between February 2025 and October 2025 under the FASTNAP protocol. The study was conducted in accordance with the Declaration of Helsinki; all patient data were de-identified prior to analysis.

Patient Selection

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Inclusion criteria were: age 18 years or above, completed ambulatory Level 3 HSAT under the FASTNAP protocol, and technically adequate recording (total recording time 4 hours or more). Patients with prior diagnosed sleep-disordered breathing on treatment and those with known central apnoea syndromes were excluded. Thirteen patients met the criteria and constitute the study cohort.

Device and Signal Acquisition

Studies were conducted using the Dormir Bien Level 3 HSAT system, which records nasal airflow (thermistor and pressure transducer), pulse oximetry, heart rate, body position, and snoring. Automated scoring was reviewed manually by a registered polysomnographic technologist and the reporting sleep physician.

Outcome Measures

The following parameters were extracted from validated reports: AHI (events per hour), rapid eye movement AHI (REM-AHI, events per hour), ODI (3% oxygen saturation desaturations per hour), arousal index (arousals per hour), minimum oxygen saturation, mean oxygen saturation, T90 (percentage of sleep time with oxygen saturation below 90%), and HB (min/hr, where directly reported). OSA severity was classified per American Academy of Sleep Medicine (AASM) 2023 criteria (normal: AHI below 5; mild: 5 to 14.9; moderate: 15 to 29.9; severe: 30 or above) [7]. HB was categorised as low (below 30 min/hr), moderate (30 to 70 min/hr), or high (above 70 min/hr) based on published cardiovascular mortality thresholds from the MrOS Sleep Study [3].

Statistical Analysis

Descriptive statistics are reported as mean plus or minus standard deviation, median with interquartile range, and range. Given the small sample size (n = 13), no formal inferential statistics were computed. This report is intended as hypothesis-generating and descriptive in nature. Analysis was performed using Microsoft Excel and R version 4.3.

III. Results

Cohort Characteristics

The cohort comprised 13 adults (8 male [61.5%], 5 female [38.5%]) with a mean age of 46.5 plus or minus 14.4 years (range 23 to 69 years). Five patients had an abnormal Epworth Sleepiness Scale score (11 or above) and four were hypertensive. Demographic and polysomnographic data are presented in Table 1.

TABLE 1. Patient demographics, polysomnographic parameters, and obstructive sleep apnoea severity classification.

ID	Sex	Age (yr)	Apnoea-Hypopnoea Index	Rapid Eye Movement Apnoea-Hypopnoea Index	Oxygen Desaturation Index	Arousal Index	Severity
Pt-01	M	43	14.2	9.1	18.4	23.4	Mild
Pt-02	M	53	53.9	48.5	35.2	59.0	Severe
Pt-03	M	53	13.8	27.2	21.5	29.9	Mild
Pt-04	M	42	9.6	12.4	12.7	22.1	Mild
Pt-05	M	23	4.5	1.1	4.9	22.3	Normal
Pt-06	F	58	6.7	14.9	10.4	27.2	Mild
Pt-07	F	38	28.9	56.0	27.0	65.9	Moderate
Pt-08	M	42	109	81.2	49.4	30.5	Severe
Pt-09	F	69	41.5	41.5	29.0	43.8	Severe
Pt-10	F	63	29.8	10.7	14.0	47.4	Moderate
Pt-11	F	57	60.4	62.2	50.8	49.2	Severe
Pt-12	M	23	1.9	2.8	3.4	43.3	Normal
Pt-13	M	50	16.0	21.1	26.8	14.3	Moderate

AHI: apnoea-hypopnoea index; ODI: oxygen desaturation index; N/R: not reported.

Obstructive Sleep Apnoea Severity Distribution

OSA severity was classified as normal in 2 patients (15.4%), mild in 4 (30.8%), moderate in 2 (15.4%), and severe in 5 (38.5%), giving a moderate-to-severe prevalence of 53.8%. The AHI ranged from 1.9 to 109 events per hour (median 16.0). The patient with the highest AHI of 109 (Pt-08) had 607 scored respiratory events, consistent with near-continuous obstructive activity throughout the recording period.

Oximetric Profile and Hypoxic Burden

Minimum oxygen saturation ranged from 61% to 94% (mean 81.2 plus or minus 9.7%). T90 ranged from 0% to 87.7%. Hypoxic burden was directly reported in 4 of 13 patients, ranging from 12.38 to 49.09 min/hr (all within the low-to-moderate category). Detailed oximetric data are presented in Table 2 and summary statistics in Table 3.

Scatter Plot Analysis

Figure 1 illustrates the dissociation between AHI and oximetric burden across the cohort. In the left panel (AHI versus T90), patients above the 5% threshold carry clinically significant nocturnal hypoxaemia regardless of AHI category. In the right panel (AHI versus minimum oxygen saturation), patients with mild OSA achieving oxygen saturation nadirs below 80% are identifiable — a group whose cardiovascular risk is structurally concealed by AHI-only reporting.

Apnoea-Hypopnoea Index and Hypoxaemia Dissociation Phenotypes

Phenotype A — Silent hypoxaemia (low apnoea-hypopnoea index, high time below 90% oxygen saturation): Pt-03 (AHI 13.8, mild OSA) had minimum oxygen saturation of 78% and T90 of 17%, driven by prolonged individual event durations and significant REM-predominant OSA (REM-AHI 27.2 versus overall AHI 13.8). Under standard AHI-only

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classification, this patient would be offered conservative management, yet the T90 of 17% exceeds the threshold associated with pulmonary hypertension risk and the minimum oxygen saturation falls in the range associated with impaired neurocognitive function and arrhythmia susceptibility.

TABLE 2. Oximetric profile and hypoxic burden data for each patient.

ID	Minimum SpO2 (%)	Mean SpO2 (%)	Time below 90% SpO2 (%)	Oxygen Desaturation Index	Hypoxic Burden (min/hr)	Hypoxic Burden Category
Pt-01	80	94	3.9	18.4	43.65	Moderate
Pt-02	80	96	0.9	35.2	N/R	—
Pt-03	78	91	17.0	21.5	N/R	—
Pt-04	91	96	0.0	12.7	N/R	—
Pt-05	94	96	0.0	4.9	N/R	—
Pt-06	84	94	0.3	10.4	12.38	Low
Pt-07	68	96	0.9	27.0	49.09	Moderate
Pt-08	61	78	87.7	49.4	N/R	—
Pt-09	71	93	11.2	29.0	N/R	—
Pt-10	69	92	19.8	14.0	39.83	Moderate
Pt-11	85	95	1.0	50.8	N/R	—
Pt-12	89	96	0.0	3.4	N/R	—
Pt-13	87	94	0.6	26.8	N/R	—

T90: time below 90% oxygen saturation; HB: hypoxic burden; N/R: not reported.

TABLE 3. Summary statistics for key parameters across the cohort (n = 13).

Parameter	Minimum	Maximum	Mean ± SD	Median (IQR)
Age (years)	23	69	46.5 ± 14.4	50 (38–57)
Apnoea-Hypopnoea Index (events/hr)	1.9	109	26.2 ± 31.2	16.0 (9.6–41.5)
Rapid Eye Movement AHI (events/hr)	1.1	81.2	31.0 ± 24.0	21.1 (9.1–48.5)
Oxygen Desaturation Index (events/hr)	3.4	50.8	22.7 ± 15.4	21.5 (12.7–35.2)
Minimum SpO2 (%)	61	94	81.2 ± 9.7	84 (78–90)
Mean SpO2 (%)	78	96	93.2 ± 4.4	94 (92–96)
Time below 90% SpO2 (%)	0	87.7	10.4 ± 24.5	0.9 (0–11.2)
Hypoxic Burden (min/hr) [n=4]	12.38	49.09	36.2 ± 15.5	41.7

*Hypoxic burden statistics based on 4 patients in whom the parameter was directly reported by the platform.

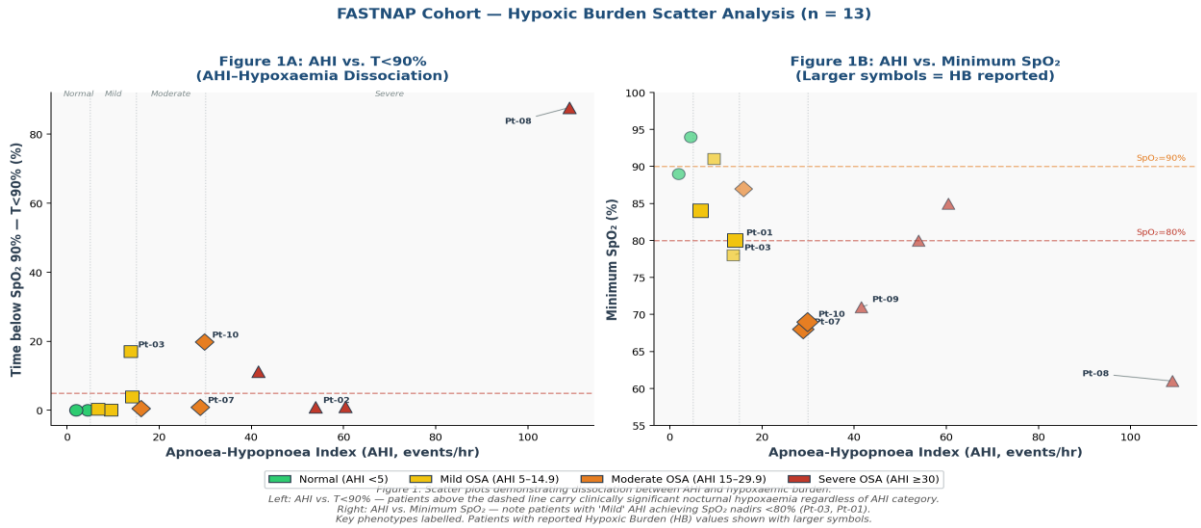

Figure 1. Scatter plots demonstrating dissociation between the apnoea-hypopnoea index and oximetric burden in the FASTNAP cohort (n = 13). Left panel: apnoea-hypopnoea index versus time below 90% oxygen saturation. Right panel: apnoea-hypopnoea index versus minimum oxygen saturation. Colour denotes obstructive sleep apnoea severity grade. Larger symbols (right panel) indicate patients with directly reported hypoxic burden values. Key phenotype patients are labelled.

Phenotype B — Event-dominant without depth (high apnoea-hypopnoea index, preserved mean oxygen saturation): Pt-02 (AHI 53.9, severe OSA) had mean oxygen saturation of 96% and T90 of only 0.9% despite 242 scored events, reflecting rapid arousal response after each event that truncates individual desaturation durations. Hypoxic burden is paradoxically modest despite a high AHI.

Phenotype C — Extreme physiological compromise (very high apnoea-hypopnoea index, catastrophic hypoxaemia): Pt-08 (AHI 109) had mean oxygen saturation of 78%, minimum oxygen saturation of 61%, and T90 of 87.7%, spending only 10.4% of sleep above 90% oxygen saturation. This pattern is consistent with obesity hypoventilation syndrome overlying

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obstructive apnoeas, and immediate bilevel ventilation was clinically indicated.

Phenotype D — Rapid eye movement-dependent hypoxaemia: Pt-07 (overall AHI 28.9, REM-AHI 56.0) had minimum oxygen saturation of 68% and HB of 49.09 min/hr, representing moderate HB despite a moderate overall AHI — reflecting the clinically severe hypoxic exposure concentrated within REM sleep periods.

IV. Discussion

The present data demonstrate clear and clinically consequential dissociation between AHI-based OSA severity classification and actual hypoxic exposure in a real-world clinical HSAT cohort. These findings mirror the broader literature challenging the prognostic adequacy of AHI as a sole severity descriptor [1-5].

The seminal work of Azarbarzin et al. demonstrated in the MrOS Sleep Study that HB independently predicted cardiovascular mortality (hazard ratio 1.42 per standard deviation increase; 95% confidence interval 1.12 to 1.80) after full AHI adjustment [3]. Our identification of Phenotype A — patients with mild AHI but T90 exceeding 17% — directly instantiates this clinical paradox. A T90 of 17% has been associated with right heart strain and pulmonary hypertension risk in population studies, and a minimum oxygen saturation of 78% falls within the range associated with impaired nocturnal arterial oxygen delivery [9].

The ODI-AHI dissociation observed in several patients (notably Pt-13, ODI 26.8 versus AHI 16.0) reflects a recognised limitation of Level 3 HSAT: respiratory disturbances generating desaturations below the thermistor-based apnoea or hypopnoea threshold contribute to ODI without AHI credit. This shadow burden argues for ODI as a mandatory co-variable in all HSAT interpretation [10].

The high prevalence of REM-predominant OSA in this cohort (53.8% with REM-AHI at least 1.5-fold greater than overall AHI) is consistent with published estimates and carries significant implications given the association between REM-OSA and nocturnal hypertension [8]. All four hypertensive patients in the cohort had elevated REM-AHI — a hypothesis-generating observation warranting prospective evaluation.

The arousal index modulates the AHI-hypoxaemia relationship bidirectionally. Rapid arousals truncate individual desaturation durations, limiting HB contribution. Pt-08, with AHI of 109 but arousal index of only 30.5, exemplifies a blunted arousal phenotype permitting catastrophic sustained hypoxaemia (T90 87.7%) — a distinction that is entirely invisible to AHI-based severity classification.

Current AASM Level 3 HSAT reporting guidelines mandate AHI and ODI but do not uniformly require HB reporting [7,10]. Our data support adoption of a multi-parameter oximetric reporting standard that mandates HB alongside AHI. The Dormir Bien platform used in the FASTNAP protocol generates HB categorisation for a subset of studies, and we advocate for universal HB computation as a platform-level requirement.

Several limitations warrant acknowledgement. The sample size of 13 limits statistical power and generalisability. Level 3 HSAT inherently underestimates AHI relative to polysomnography due to the absence of sleep staging (denominator is recording time, not actual sleep time). HB was directly reported in only 4 of 13 patients, limiting cohort-wide HB analysis. Structured comorbidity data including body mass index, blood pressure values, and medication history were not uniformly available. A prospective FASTNAP registry with mandatory comorbidity capture is planned to address these gaps.

In conclusion, AHI-centric OSA severity classification systematically misrepresents hypoxic exposure in a clinically heterogeneous population. Four distinct AHI-hypoxaemia dissociation phenotypes are identifiable through multi-parameter oximetric profiling, each carrying different clinical management implications. Hypoxic burden, ODI, T90, and minimum oxygen saturation together constitute an oximetric severity profile that should be reported as a mandatory adjunct to AHI in all Level 3 HSAT. As the FASTNAP cohort expands prospectively, these data will form the basis for Indian-specific HB reference ranges and risk-stratification algorithms that move beyond AHI-centric obstructive sleep apnoea classification.

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Conflict of Interests:

The corresponding author (Razzakur Rahman) serves as Senior Clinical Advisor to Deckmount Electronics and Clinical Advisor to SlumberSense Global. These affiliations did not influence study design, data analysis, or manuscript preparation. The remaining authors declare no conflicts of interest.

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