



Severity classification of obstructive sleep apnea using AASM and SEPAR criteria: A cross-sectional reclassification analysis

Joaquín Nieto-Pino^{1,2} · Eva Retamal-Riquelme¹ · Mario Henriquez-Beltrán^{3,4,5} · Matías Otto-Yañez⁶ · Rodrigo Torres-Castro⁷ · Gonzalo Labarca^{8,9}

Received: 28 March 2025 / Accepted: 3 July 2025 / Published online: 2 August 2025
© The Author(s), under exclusive licence to Springer-Verlag GmbH Germany, part of Springer Nature 2025

Abstract

Purpose The severity of obstructive sleep apnea (OSA) is traditionally assessed using the Apnea–Hypopnea Index (AHI), as recommended by the American Academy of Sleep Medicine (AASM). However, the Spanish Society of Pulmonology and Thoracic Surgery (SEPAR) has proposed a multidimensional classification incorporating additional clinical factors. This study aimed to compare OSA severity distribution between the AASM and SEPAR classification systems in an adult population.

Methods A retrospective cross-sectional study was conducted. OSA severity was classified using AHI-based criteria (AASM) and SEPAR, which integrates total recording time with oxyhemoglobin saturation below 90% (T90%), body mass index (BMI), Epworth Sleepiness Scale (ESS), and cardiovascular history (CV). Transition analysis was performed to evaluate reclassification patterns, and logistic regression models were used to identify factors associated with higher severity categorization under SEPAR.

Results The SEPAR classification resulted in a higher proportion of cases categorized as severe compared to AASM (50.9% vs. 72.8%, $p < 0.001$). T90% and BMI were the primary contributors to this reclassification, with 24% of patients initially classified as moderate under AASM criteria reclassified as severe using SEPAR.

Conclusion AHI-based classification alone may underestimate OSA severity, particularly in individuals with significant nocturnal hypoxemia or obesity. Integrating multidimensional criteria identifies a more considerable proportion of severe cases, with potential implications for treatment prioritization and risk stratification. These findings support the need for broader evaluation strategies in clinical practice.

Keywords Obstructive sleep apnea · Agreement · Classification · Nocturnal hypoxemia

✉ Matías Otto-Yañez
matiasotto.kine@gmail.com

¹ Unidad de Sueño Adulto, Hospital Clínico La Florida, Santiago, Chile

² Health and Social Research Center, Universidad de Castilla-La Mancha, Cuenca, Spain

³ Núcleo de Investigación en Ciencias de la Salud, Universidad Adventista de Chile, Chillán, Chile

⁴ Translational Research in Respiratory Medicine, Hospital Universitari Arnau de Vilanova-Santa Maria, Biomedical Research Institute of Lleida (IRBLleida), Lleida, Spain

⁵ CIBER of Respiratory diseases (CIBERES), Institute of Health Carlos III, Madrid, Spain

⁶ Grupo de Investigación en Salud, Funcionalidad y Actividad Física (GISFAF), Kinesiología, Facultad de Ciencias de La Salud, Universidad Autónoma de Chile, Santiago, Chile

⁷ Department of Physical Therapy, University of Chile, Santiago, Chile

⁸ Departamento de Enfermedades Respiratorias, Facultad de Medicina, Pontificia Universidad Católica de Chile, Santiago, Chile

⁹ Division of Respiratory, Allergy and Sleep Medicine, Mayo Clinic, Jacksonville, FL, USA

Introduction

Obstructive sleep apnea (OSA) is a prevalent sleep disorder characterized by recurrent upper airway collapse during sleep [1], leading to intermittent hypoxia, arousal, and autonomic instability [2]. It is strongly associated with obesity, hypertension, cardiovascular diseases, and impaired quality of life [3, 4]. The estimated global prevalence of OSA exceeds one billion adults between 30 and 69 years [5].

OSA diagnosis relies on polysomnography (PSG), the gold standard, or home sleep apnea testing (HSAT), which offers high sensitivity and specificity [6, 7].

The severity of OSA is classified according to the AHI, which is the number of apneas or hypopneas recorded per hour of sleep during the study. OSA severity is classified as mild ($\text{AHI} \geq 5$ to < 15 per hour), moderate ($\text{AHI} \geq 15$ to < 30 per hour), and severe ($\text{AHI} \geq 30$ per hour) [1]. However, using AHI alone is a narrow perspective as it may not be informative due to the heterogeneity of OSA disease [8]. To account for the heterogeneity and multiple clinical presentations of OSA, investigators, physicians, and clinical staff must analyze the OSA population using the broad spectrum of available data. Current investigations have proposed additional parameters to evaluate the severity of OSA, such as phenotype characteristics, hypoxemia severity, and respiratory event duration [9, 10]. The new proposal from the Spanish Society of Pulmonology and Thoracic Surgery (SEPAR) includes additional parameters such as T90%, daytime somnolence, BMI, and CV comorbidities to evaluate the severity of OSA [11]. This study aimed to compare the distribution of OSA severity using American Academy of Sleep Medicine (AASM) and SEPAR classification systems

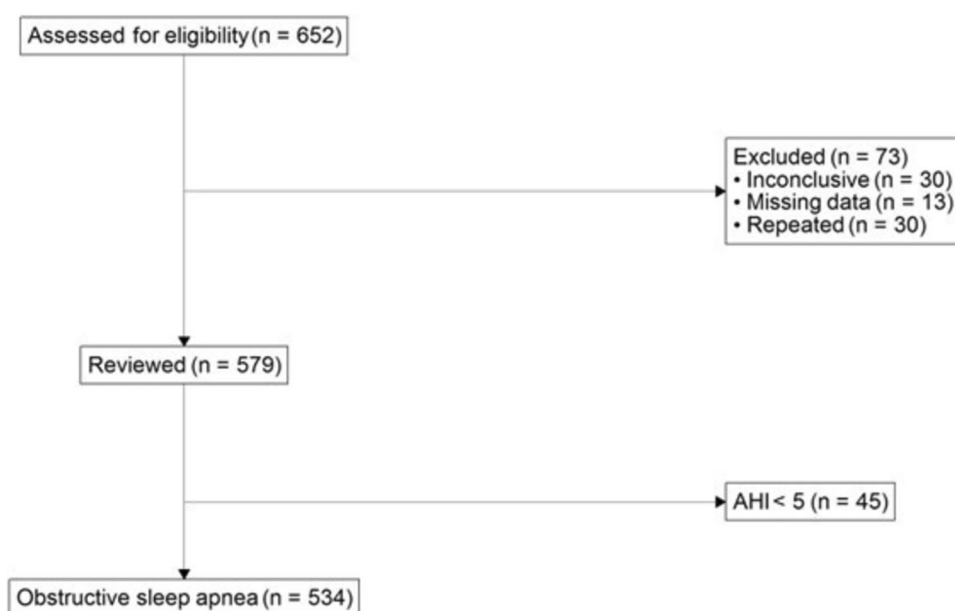
and to assess how a multidimensional approach modifies patient classification.

Methods

The study was conducted retrospectively from February 2018 to June 2023 at a public secondary care hospital. The Ethics-Scientific Committee reviewed and approved the research protocol (12–09-2023). The study included subjects older than 18 years with a confirmed diagnosis of OSA ($\text{AHI} \geq 5$ events/h) by HSAT. Subjects with $\text{AHI} < 5$ events/h were excluded from the study (Fig. 1). The AASM criteria classify the severity of OSA into three categories: mild, moderate, and severe. For this study, the cut-off scores proposed by the international document SEPAR [11] were used as follows:

- i) T90%; OSA was considered mild if T90% was $< 1\%$, moderate if $\text{T90\%} \geq 1\%$ and $< 15\%$, and severe if it was $\geq 15\%$.
- ii) ESS: OSA was considered mild if the score was < 10 , moderate ≥ 10 and < 15 , and severe if it was ≥ 15 .
- iii) BMI: if it was < 25 , it was considered mild OSA; between ≥ 25 and < 30 , moderate OSA; if it was $\text{BMI} \geq 30$, severe OSA.
- iv) Cardiovascular history (CV): OSA was considered mild if one does not have cardiovascular comorbidities, moderate if one has one or more cardiovascular risk factors (hypertension, diabetes mellitus, dyslipidemia), and severe if one has a stroke and/or myocardial infarction.

Fig. 1 Flowchart of patient inclusion and exclusion for the final OSA cohort



- v) Worst-case scenario (WCS): This involves considering the most severe category among all the above criteria for each case.

Study measures

All patients underwent an initial clinical evaluation, which included the extraction of data about demographics (age, educational level, sex), anthropometry measurements (weight, height, BMI (kg/m^2), neck and waist circumferences), and alcohol and tobacco consumption. Data on comorbidities and medical treatment were obtained from self-reports, with confirmation in clinical and medical records.

Concerning the sleep evaluation component of our study, the excessive daytime somnolence was evaluated using the validated Spanish version of the Epworth Sleepiness Scale (ESS) [12]. The OSA diagnosis was performed using the ApneaLink Air® device (Resmed, San Diego, CA, USA), a 5-channel HSAT that records nasal flow, thoracoabdominal effort, oximetry, snoring, and heart rate [7]. Registers were manually reviewed using the ApneaLink® version 10.20 software (Resmed, San Diego, CA, USA) by a sleep medicine specialist, according to the current standards of the AASM [1].

In cases where the presence of a comorbid sleep disorder had a substantial impact on sleep efficiency (e.g., insomnia, restless legs syndrome, circadian rhythm disorders, among others), treatment for the identified disorder was initiated, and HSAT was delayed until amelioration of those conditions, thereby ensuring that the duration of the study closely approximated total sleep time. Data were extracted about the following variables: AHI (apneas or hypopneas associated with at least 3% oxygen desaturation per hour), minimum oxygen saturation (nadir SpO_2), T90%, obstructive apnea index (OAI), and central apnea index (CAI).

Statistical analysis

The variables are presented as percentages for categorical variables. For numerical variables, age is expressed using the range, median, and interquartile range (IQR), while other numerical variables are represented as mean $\pm$ standard deviation. Pearson's Chi-squared test was employed to compare categorical variables, and the Welch Two Sample t-test or Wilcoxon rank-sum test was used for numerical variables. A p-value of less than 0.05 was considered statistically significant. Severity distribution was compared between the AASM and SEPAR classification systems. A transition analysis was conducted to evaluate the migration of patients across severity categories. Logistic regression models were used to explore factors associated with

reclassification across severity categories under the SEPAR criteria.

The agreement between the AASM classification of OSA and those proposed in the SEPAR international document was analyzed [11]. The kappa coefficient was used to measure the concordance of both criteria. This coefficient represents the agreement between observers [13]. Weighted kappa was used since the variable to be evaluated is ordinal.

A kappa coefficient below 0.0 denoted poor agreement. Kappa values falling within the range of 0.0 to 0.20 were interpreted as suggesting slight agreement, whereas values between 0.21 and 0.40 were indicative of fair agreement. Moderate agreement fell within the range of 0.41 to 0.60. Substantial agreement was observed between 0.61 and 0.80. Kappa values exceeding 0.80 were considered to reflect nearly perfect agreement [14].

Inclusion criteria comprised cases with an AHI of ≥ 5 events per hour (e/h). The severity of OSA was stratified based on AHI values into mild (AHI between 5 and 14.9 e/h), moderate (AHI between 15 and 29.9 e/h), and severe (AHI ≥ 30). All statistical analyses were performed using statistical language R (R-project) in the integrated development environment RStudio version 2022.07.2+576 (Posit, PBC; Boston, MA, USA). *DescTools* package was used to calculate the kappa statistics, *ggsankey* for flowcharts, and *gtsummary* for tables.

Results

The study included 534 OSA patients, with a predominance of males (51.7%) and a median age of 57 years. The mean BMI was 35.8 kg/m^2 , with 72.8% classified as obese. Hypertension was the most common comorbidity (63.3%). Sleep-related metrics, including AHI, OAI, CAI, T90%, and ESS, are presented in Table 1.

OSA severity classification varied significantly depending on the applied criteria (Table 2). Using AHI-based classification (AASM), 21.2% of cases were classified as mild, 27.9% as moderate, and 50.9% as severe. When alternative metrics were applied, a notable increase in severe cases was observed, particularly with T90% (62.7%) and BMI (72.8%). Conversely, severity classification based on ESS resulted in fewer cases classified as severe (33.1%). The cardiovascular (CV) criterion yielded a higher proportion of moderate cases (52.1%), while the worst-case scenario (WCS) assigned 92.1% of cases to the severe category.

Figures 2 and 3 illustrate the reclassification patterns. The Sankey diagram (Fig. 3) highlights the redistribution of cases from moderate to severe when T90% and BMI were incorporated. 45% of cases classified as moderate under

Table 1 Cohort characteristics stratified by sex

Cohort Characteristics	All cases, N=534*	Female, N=258*	Male, N=276*	p-value**
Sociodemographic data				
Age (years)	57 (12)	57 (12)	58 (13)	0.4
Educational level				
≥ 12 years	358 (67%)	162 (63%)	196 (71%)	0.037
< 12 years	175 (33%)	96 (37%)	79 (29%)	
Alcohol consumption	39 (7.3%)	9 (3.5%)	30 (11%)	0.001
Active Smoker	126 (24%)	72 (28%)	54 (20%)	0.023
Anthropometric data				
BMI (kg/m ²)	35.8 (8.3)	36.9 (8.7)	34.7 (7.6)	0.002
Waist Circumference (cm)	114 (16)	111 (16)	116 (16)	0.002
Neck Circumference (cm)	43.0 (4.9)	40.5 (4.4)	45.2 (4.2)	<0.001
Comorbidities				
Hypertension	338 (63%)	154 (60%)	184 (67%)	0.095
Diabetes Mellitus	202 (38%)	104 (40%)	98 (36%)	0.3
Dyslipidemia	214 (40%)	108 (42%)	106 (38%)	0.4
Myocardial infarction	72 (13%)	29 (11%)	43 (16%)	0.14
Stroke	72 (13%)	24 (9.3%)	48 (17%)	0.006
Obesity	389 (73%)	198 (77%)	191 (69%)	0.05
Sleep evaluation				
ESS (points)	11.7 (5.7)	11.7 (5.8)	11.7 (5.5)	>0.9
AHI (events/hour)	37.7 (27.0)	35.1 (28.2)	40.2 (25.6)	0.028
OAI (events/hour)	13.7 (20.1)	9.2 (16.8)	17.9 (21.9)	<0.001
CAI (events/hour)	0.2 (0.7)	0.1 (0.5)	0.3 (0.9)	0.015
T90 (%)	36.4 (32.1)	37.2 (33.8)	35.6 (30.4)	0.6
ODI				0.092
Sleep Study Duration (hours)	38.6 (27.3)	36.6 (29.1)	40.6 (25.4)	0.051
	7.2 (1.3)	7.0 (1.2)	7.3 (1.4)	

Abbreviations: *BMI* Body Mass Index; *ESS* Epworth Sleepiness Scale; *AHI* Apnea–Hypopnea Index; *OAI* Obstructive Apnea Index; *CAI* Central Apnea Index; *T90%* Percentage of time with SpO₂ below 90%; *ODI* Oxygen Desaturation Index. *Mean (SD); *n* (%); ** Welch Two Sample *t*-test; Pearson's Chi-squared test

AHI were reclassified as severe using BMI, and 35% using T90%.

Agreement between AHI and alternative criteria The level of agreement between AHI and alternative criteria varied from slight to moderate (Table 2, Fig. 2). The highest agreement

Table 2 Severity distribution of clinical and sleep-related variables by sex

Characteristic	All cases, N=534*	Female, N=258*	Male, N=276*	p-value**
AHI				0.026
Mild	113 (21.2%)	63 (24.4%)	50 (18.1%)	
Moderate	149 (27.9%)	79 (30.6%)	70 (25.4%)	
Severe	272 (50.9%)	116 (45.0%)	156 (56.5%)	
T90%				0.1
Mild	21 (3.9%)	8 (3.1%)	13 (4.7%)	
Moderate	178 (33.3%)	97 (37.6%)	81 (29.3%)	
Severe	335 (62.7%)	153 (59.3%)	182 (65.9%)	
ESS				0.6
Mild	199 (37.3%)	100 (38.8%)	99 (35.9%)	
Moderate	158 (29.6%)	71 (27.5%)	87 (31.5%)	
Severe	177 (33.1%)	87 (33.7%)	90 (32.6%)	
BMI				0.15
Mild	21 (3.9%)	9 (3.5%)	12 (4.3%)	
Moderate	124 (23.2%)	51 (19.8%)	73 (26.4%)	
Severe	389 (72.8%)	198 (76.7%)	191 (69.2%)	
CV				0.043
Mild	127 (23.8%)	64 (24.8%)	63 (22.8%)	
Moderate	278 (52.1%)	144 (55.8%)	134 (48.6%)	
Severe	129 (24.2%)	50 (19.4%)	79 (28.6%)	
WCS				0.3
Mild	2 (0.4%)	1 (0.4%)	1 (0.4%)	
Moderate	40 (7.5%)	15 (5.8%)	25 (9.1%)	
Severe	492 (92.1%)	242 (93.8%)	250 (90.6%)	

Abbreviations: *AHI* Apnea–Hypopnea Index; *T90%* Percentage of time with SpO₂ below 90%; *ESS* Epworth Sleepiness Scale; *BMI* Body Mass Index; *CV* Cardiovascular history; *WCS* Worst Case Scenario; **n* (%); ** Pearson's Chi-squared test

was observed between AHI and T90% (kappa=0.39, 95% CI: 0.33–0.45), whereas AHI and ESS showed only slight agreement (kappa=0.08, 95% CI: 0.02–0.14). Similarly, agreement between AHI and BMI was fair (kappa=0.21, 95% CI: 0.14–0.26). For cardiovascular disease-based classification, the agreement was slight (kappa=0.12, 95% CI: 0.06–0.18).

When considering WCS, most cases were classified as severe (92.1%). The agreement between AHI and WCS remained low (kappa=0.12, 95% CI: 0.09–0.16), indicating that AHI alone may not fully capture disease burden when multidimensional classification is applied. Figure 3 visually depicts these classification changes across all severity criteria.

Sex-based differences were identified in the classification by cardiovascular disease (*p*=0.043) but not in other

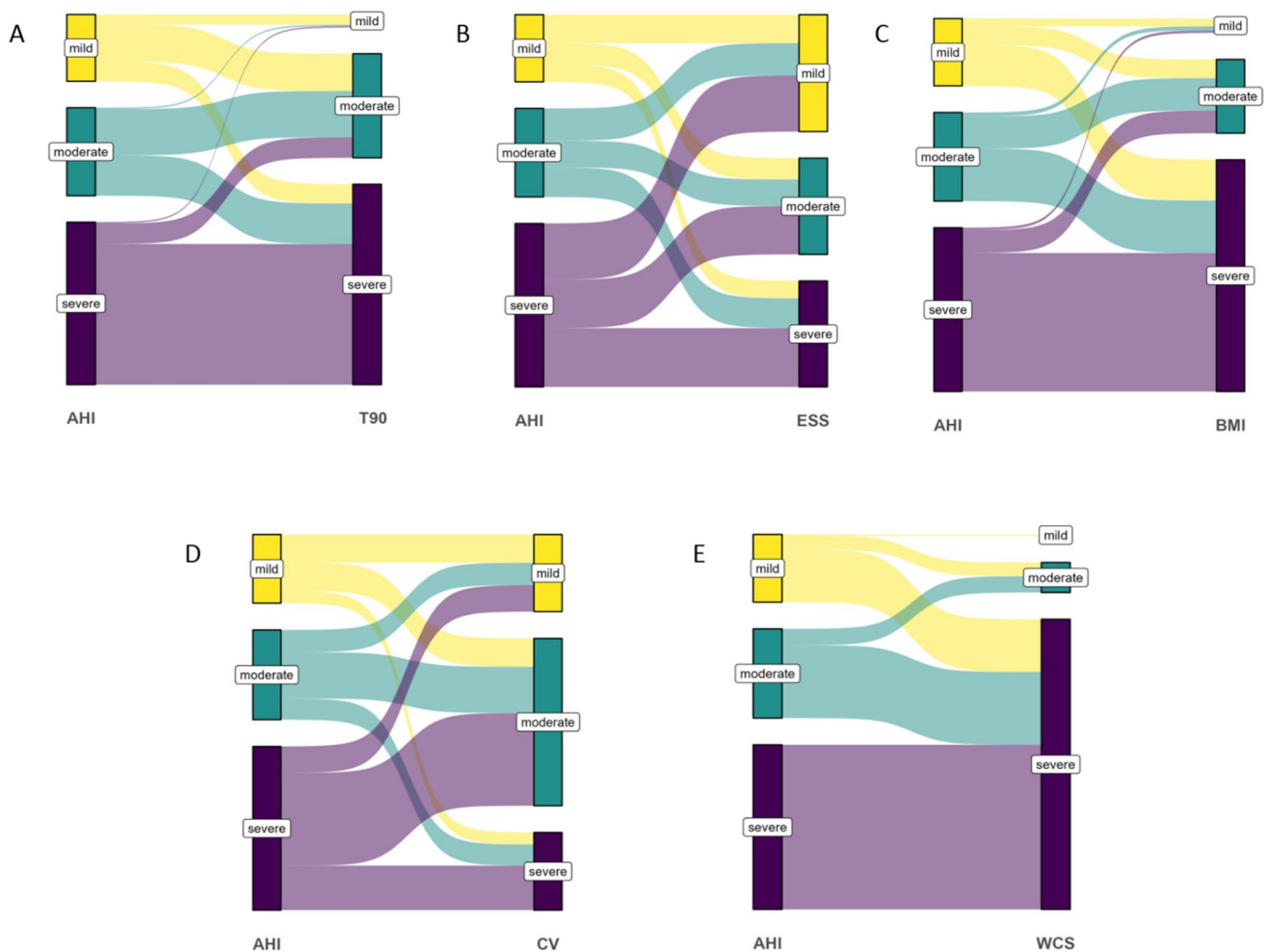


Fig. 2 Concordance between A: AHI-T90%, B: AHI-ESS, C: AHI-BMI, D: AHI-CV, E: AHI-WCS. Abbreviation list: Apnea-Hypopnea Index (AHI), Cardiovascular History (CV), Epworth Sleepiness Scale

severity criteria ($p > 0.1$ for all). Kappa values stratified by sex showed slightly higher agreement in females (kappa=0.20, 95% CI: 0.11–0.29) than in males (kappa=0.03, 95% CI: –0.05–0.12).

Discussion

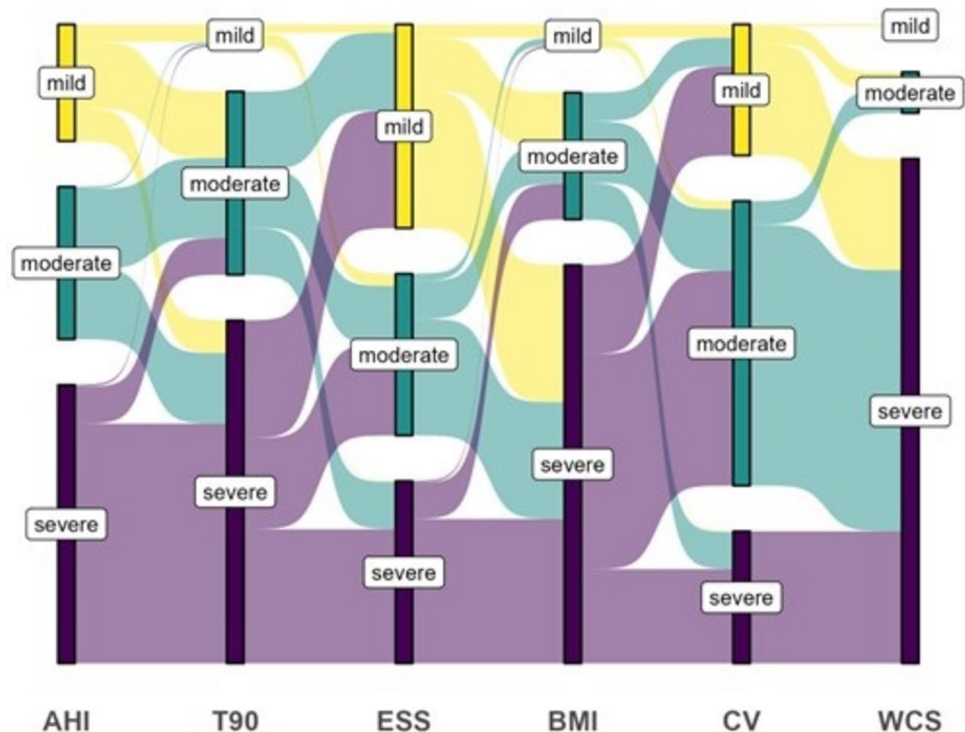
The observed differences in OSA severity classification across criteria highlight the limitations of relying solely on AHI. Each metric captures distinct physiological and clinical dimensions of the disease. AHI, while a widely used metric, primarily quantifies the frequency of respiratory events, whereas T90% assesses nocturnal hypoxemia, and BMI reflects the mechanical and inflammatory impact of obesity on airway collapsibility. The increase in severe cases observed under SEPAR criteria suggests that AHI alone may underestimate disease burden in certain patient subgroups.

(ESS), Body mass index (BMI), total time with oxyhemoglobin saturation below 90% (T90%), worst case scenario (WCS)

These findings further support that OSA is a complex condition with distinct physiological dimensions. T90% and BMI-based classification identified more severe cases, likely due to their ability to capture chronic hypoxia and obesity-related airway collapsibility, both of which are associated with increased cardiovascular risk.

While the AHI remains the most widely used metric for OSA severity, its limitations are recognized, particularly in moderate cases. Rapoport (2016) noted that in adults with moderate obstructive sleep apnea, the correlation between AHI and clinically relevant outcomes such as sleepiness or cardiovascular risk is often weak in this group, supporting the need to explore complementary markers [15]. Likewise, Punjabi (2016) acknowledges that although the AHI has served as the standard for diagnosis and severity grading, it represents a crude summary of OSA pathophysiology, overlooking critical dimensions such as event duration, hypoxemia severity, event clustering, and respiratory effort [16]. Both authors emphasize that the field should move toward a

Fig. 3 Concordance between all criteria



multidimensional characterization of OSA, integrating multiple physiological and clinical factors. In this context, our study does not aim to replace AHI, but to explore whether adding T90%, BMI, and cardiovascular comorbidities could offer a broader assessment of disease severity. This approach requires further validation against clinical outcomes before being adopted in routine practice.

OSA is increasingly recognized as a systemic disorder rather than merely a condition defined by the AHI. Its pathophysiology involves intermittent hypoxia, sympathetic nervous system overactivation, and systemic inflammation, which contribute to oxidative stress, endothelial dysfunction, and structural cardiovascular remodeling [17–19]. In adult patients with OSA, these mechanisms are strongly associated with an increased risk of hypertension, coronary artery disease, heart failure, and cerebrovascular events, independently of AHI severity [17, 19–21]. Furthermore, the growing recognition of OSA as a multisystemic disease reinforces the limitations of a classification system based solely on AHI, as it may not fully capture the condition's clinical heterogeneity and systemic burden. Multidimensional approaches—such as the SEPAR classification—considering parameters beyond event frequency, including hypoxemia, comorbidities, and symptoms, are better aligned with the complex pathophysiology and prognostic variability observed in OSA patients.

The growing recognition of distinct clinical phenotypes in OSA—such as those characterized by excessive daytime sleepiness, insomnia symptoms, or prominent

cardiometabolic comorbidities—underscores the limitations of relying solely on event-based indices like the AHI. While the SEPAR classification does not formally categorize phenotypes, its inclusion of hypoxemia, obesity, and cardiovascular burden aligns with key clinical dimensions that underlie phenotypic diversity. For instance, Morris et al. (2021) identified sex-specific symptom profiles in mild OSA, highlighting how demographic and phenotypic variability can influence clinical presentation and severity perception [22]. These insights reinforce the value of multidimensional tools that accommodate patient heterogeneity, even if not structured explicitly around phenotypic clusters.

The Sankey diagrams (Figs. 2 and 3) clearly depict how the application of T90% and BMI reclassified a significant proportion of moderate cases into the severe category. This transition highlights that OSA severity is highly dependent on the physiological dimension being evaluated, reinforcing the notion that a single metric is insufficient to fully capture the disease burden.

Prior studies have explored alternative classifications for OSA, including phenotype-based approaches. However, many of these models lack external validation and do not fully address disease heterogeneity across demographic and clinical subgroups [23]. Our findings suggest that integrating multiple physiological markers may offer a more robust framework for severity assessment.

Furthermore, Randerath W et al. (2018) emphasized the need for phenotype-based approaches in OSA classification [24]. However, traditional PSG-based classification systems

have shown limitations in identifying patients at higher cardiovascular risk [25], reinforcing the importance of incorporating clinical comorbidities—such as cardiovascular disease—as proposed by the SEPAR classification. This is particularly important given that OSA is a medical condition that is characterized by severe cardiovascular impairment [26] and closely related to a set of comorbidities, including stroke, diabetes, hypertension, and heart failure [27]. These comorbidities are associated with an increased risk of mortality [28].

T90% emerged as a key determinant of increased severity in our analysis. Given its established role as a predictor of cardiovascular risk, its incorporation into OSA classification may have important prognostic implications [29]. A higher T90% was independently associated with the risk of hypertension in patients with severe OSA [30]. Importantly, this metric can be readily obtained from various diagnostic sleep studies, such as HSAT and PSG.

Likewise, BMI-based classification led to a substantial shift toward higher severity categories, consistent with prior research indicating that obesity exacerbates airway collapsibility and worsens OSA outcomes.

In this study, the agreement between AHI and ESS was slight. Although excessive daytime sleepiness is a common symptom in patients with OSA, ESS alone is not strongly associated with OSA severity. According to Guilleminault et al. (2001), excessive daytime sleepiness is typically a multifactorial phenomenon, often attributed to chronic sleep deprivation alone or in conjunction with other sleep disorders such as OSA [31]. For instance, in a national OSA cohort in Sweden, AHI was only weakly correlated with daytime sleepiness [32].

According to our results, a fair agreement was found between AHI and BMI. Obesity is a common feature in patients with OSA, with the incidence of OSA rising with higher BMI and age [33]. Obesity induces changes in both upper airway anatomy and neuromuscular control, thereby affecting the mechanical function of the upper airway and increasing susceptibility to OSA [34].

People with OSA have a 2 to 3 times higher risk of developing cardiovascular disease and metabolic disorders [35]. The development of cardiovascular compromise is associated with oxidative stress, a pro-inflammatory state, intermittent hypoxemia, and endothelial deregulation. These factors can trigger a cascade of events that adversely affect cardiovascular health, increasing the risk of recurrent cardiovascular events and mortality [36]. In contrast, adherence to treatment strategies, such as continuous positive airway pressure, can reduce major adverse cardiac and cerebrovascular events [37].

The recently published International Consensus Document on OSA advocates the inclusion of additional factors

in assessing the severity of patients with OSA. This guideline considers four levels (mild, moderate, severe, and very severe) for each parameter: T90%, daytime somnolence, measured through ESS, BMI, cardiovascular disease, and AHI [11].

On the other hand, we combined the severe and very severe categories in the analyses of this study because the AASM criteria only consider three levels of severity of OSA (mild, moderate, and severe). In addition, the SEPAR classification does not specify the selection of criteria for individual cases, considering the different severity levels of various metrics for the same individual. We included the worst-case scenario in the comparison to address this issue, as the SEPAR document does not mention how to choose between them. It is also important to note that potential collinearity between SEPAR's components may influence the interpretation of results. Future studies should explore this aspect to refine the classification system.

The study, conducted at a tertiary referral center, employed a convenience sample of Hispanic/Latino subjects. This may have introduced selection bias, limiting the generalizability and external validity of the findings. Furthermore, HSAT was used for diagnosis, which may underestimate the AHI due to the inability to measure total sleep time and detect arousals, leading to the under-scoring of hypopneas [38]. Both factors contribute to a potential underestimation of AHI. HSAT also does not capture arousals, sympathetic activation, or sleep fragmentation patterns. Although these factors do not directly impact AHI calculation, they have been identified as physiologically relevant traits in OSA, contributing to cardiovascular risk and potentially informing phenotypic characterizations in research settings [21, 39]. Their absence may limit the ability to characterize the multidimensional burden of OSA in clinical practice fully.

Although HSAT provides oximetry signals from which HB can be computed, this metric was not analyzed in our study due to the additional signal processing required, which was beyond the scope of this analysis. Similarly, other markers such as event duration could not be assessed. These factors may have further limited our sample's physiological characterization of OSA severity. Another potential limitation is recall bias, inherent to the retrospective nature of the study.

Given the heterogeneous clinical manifestations of OSA, it is likely that a single indicator is insufficient to fully capture the range of complications associated with this disorder, including cardiovascular and cerebrovascular events, cognitive decline, and mortality. Therefore, it is necessary to consider multiple factors, such as symptoms, biomarkers, and cardiometabolic comorbidities. Although our analysis focused on SEPAR variables, future research could explore the added value of other physiological markers—such as

arousal intensity, apnea–hypopnea event duration, and hypoxic burden—which may provide complementary prognostic insight in multidimensional models [10]. Conducting prospective assessments of supplementary parameters alongside AHI, such as hypoxemia, would enable the identification of additional criteria for evaluation and help establish optimal thresholds for diagnosis and management. Integrating such information should lead to a more personalized approach to the diagnosis and treatment of these patients. Our findings demonstrate that a multidimensional classification system significantly alters the distribution of OSA severity, identifying a more significant proportion of severe cases than AHI-based criteria alone. This suggests that traditional classifications may underrepresent disease burden in patients with substantial nocturnal hypoxemia or obesity. Given the clinical relevance of these factors, integrating additional parameters beyond the AHI, such as T90% and BMI, into routine severity assessment may provide a more comprehensive evaluation of OSA risk and treatment needs. Future research should investigate whether these alternative classifications correlate better with clinical outcomes and response to therapy.

Conclusion

This study demonstrates that applying a multidimensional classification system alters OSA severity distribution compared to the traditional AHI-based classification. Including T90% and BMI in the SEPAR criteria resulted in a higher proportion of severe cases, underscoring the importance of considering nocturnal hypoxemia and obesity in severity assessment. These findings suggest that AHI alone may not fully capture disease burden, particularly in patients at higher cardiovascular risk. Moving toward a more comprehensive classification framework could improve risk stratification and inform more personalized treatment strategies. Future research should explore whether these alternative classifications provide better predictive value for clinical outcomes, such as hospitalization, mortality, and therapeutic response.

Acknowledgements We would like to express our deepest gratitude to Sandra Herrera Vásquez, former head of the Library at Hospital Clínico La Florida, for her unwavering support to our team from the outset. Additionally, our sincere appreciation extends to hospital staff of the Adult Neurology Unit at this institution.

Data availability The datasets generated and analyzed during the current study are available from the corresponding author upon reasonable request.

Declarations

Disclosure of potential conflicts of interest One of the authors (GL) declares potential conflicts of interest. He has received research funding or support from the following institutions: Universidad de Concepción (Chile), CHEST Foundation, AASM, NIH, Sleep Research Society, and the ResMed Foundation. All other authors declare that they have no conflicts of interest.

Ethics and data management The study was conducted following the ethical standards of the institutional and national research committees, as well as the 1964 Helsinki Declaration and its subsequent amendments. The Ethics Scientific Committee of the participating institution approved the protocol (ID: 12–09–2023). It waived the requirement for informed consent due to the protocol's retrospective design and the use of anonymized data. All data used in this study were anonymized before analysis, handled in compliance with institutional data protection regulations, and accessed exclusively by the research team.

References

1. Kapur VK, Auckley DH, Chowdhuri S et al (2017) Clinical practice guideline for diagnostic testing for adult obstructive sleep apnea: an american academy of sleep medicine clinical practice guideline. *J Clin Sleep Med* 13:479–504. <https://doi.org/10.5664/jcsm.6506>
2. Miller MA, Cappuccio FP (2021) A systematic review of COVID-19 and obstructive sleep apnoea. *Sleep Med Rev* 55:101382. <https://doi.org/10.1016/j.smrv.2020.101382>
3. Senaratna CV, Perret JL, Lodge CJ et al (2017) Prevalence of obstructive sleep apnea in the general population: a systematic review. *Sleep Med Rev* 34:70–81. <https://doi.org/10.1016/j.smrv.2016.07.002>
4. Patil SP, Ayappa IA, Caples SM et al (2019) Treatment of adult obstructive sleep apnea with positive airway pressure: an american academy of sleep medicine clinical practice guideline. *J Clin Sleep Med* 15:335–343. <https://doi.org/10.5664/jcsm.7640>
5. Benjafield AV, Ayas NT, Eastwood PR et al (2019) Estimation of the global prevalence and burden of obstructive sleep apnoea: a literature-based analysis. *Lancet Respir Med* 7:687–698. [https://doi.org/10.1016/S2213-2600\(19\)30198-5](https://doi.org/10.1016/S2213-2600(19)30198-5)
6. Ng SSS, Chan T-O, To K-W et al (2009) Validation of a portable recording device (ApneaLink) for identifying patients with suspected obstructive sleep apnoea syndrome. *Intern Med J* 39:757–762. <https://doi.org/10.1111/j.1445-5994.2008.01827.x>
7. Collop NA, Anderson WM, Boehlecke B et al. Clinical guidelines for the use of unattended portable monitors in the diagnosis of obstructive sleep apnea in adult patients. *J Clin Sleep Med*, 7:737–747. <https://doi.org/10.17615/7194-MB44>
8. Epstein LJ, Kristo D, Strollo PJ et al (2009) Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. *J Clin Sleep Med* 05:263–276. <https://doi.org/10.5664/jcsm.27497>
9. Martinez-Garcia MA, Campos-Rodriguez F, Barbé F et al (2019) Precision medicine in obstructive sleep apnoea. *Lancet Respir Med* 7:456–464. [https://doi.org/10.1016/S2213-2600\(19\)30044-X](https://doi.org/10.1016/S2213-2600(19)30044-X)
10. Malhotra A, Ayappa I, Ayas N et al (2021) Metrics of sleep apnea severity: beyond the apnea-hypopnea index. *Sleep* 44:zsab030. <https://doi.org/10.1093/sleep/zsab030>
11. Mediano O, González Mangado N, Montserrat JM et al (2022) Documento internacional de consenso sobre apnea obstructiva

- del sueño. Arch Bronconeumol 58:52–68. <https://doi.org/10.1016/j.arbres.2021.03.017>
12. Gómez GM, Deck GB, Santelices BP et al (2020) Adaptación transcultural y validación de la escala de somnolencia de Epworth en la población chilena. Rev Otorrinolaringol Cir Cabeza Cuello 80:434–441. <https://doi.org/10.4067/S0718-48162020000400434>
 13. Cohen J (1960) A coefficient of agreement for nominal scales. Educ Psychol Meas 20:37–46. <https://doi.org/10.1177/001316446002000104>
 14. Landis JR, Koch GG (1977) The measurement of observer agreement for categorical data. Biometrics 33:159. <https://doi.org/10.2307/2529310>
 15. Rapoport DM (2016) POINT: is the apnea-hypopnea index the best way to quantify the severity of sleep-disordered breathing? Yes. Chest 149:14–16. <https://doi.org/10.1378/chest.15-1319>
 16. Punjabi NM (2016) COUNTERPOINT: is the apnea-hypopnea index the best way to quantify the severity of sleep-disordered breathing? No. Chest 149:16–19. <https://doi.org/10.1378/chest.14-2261>
 17. Thareja S, Mandapalli R, Shaik F et al (2024) Impact of obstructive sleep apnea on cardiovascular health: a systematic review. Cureus. <https://doi.org/10.7759/cureus.71940>
 18. Iannella G, Magliulo G, Greco A et al (2022) Obstructive sleep apnea syndrome: from symptoms to treatment. Int J Environ Res Public Health 19:2459. <https://doi.org/10.3390/ijerph19042459>
 19. De Vito A, Woodson BT, Koka V et al (2021) OSA upper airways surgery: a targeted approach. Medicina (Mex) 57:690. <https://doi.org/10.3390/medicina57070690>
 20. Liu L, Wang Y, Hong L et al (2023) Obstructive sleep apnea and hypertensive heart disease: from pathophysiology to therapeutics. Rev Cardiovasc Med 24:342. <https://doi.org/10.31083/j.rcm2412342>
 21. Maniaci A, Lavalley S, Parisi FM et al (2024) Impact of obstructive sleep apnea and sympathetic nervous system on cardiac health: a comprehensive review. J Cardiovasc Dev Dis 11:204. <https://doi.org/10.3390/jcdd11070204>
 22. Morris JL, Mazzotti DR, Gottlieb DJ, Hall MH (2021) Sex differences within symptom subtypes of mild obstructive sleep apnea. Sleep Med 84:253–258. <https://doi.org/10.1016/j.sleep.2021.06.001>
 23. Zinchuk A, Yaggi HK (2020) Phenotypic subtypes of OSA. Chest 157:403–420. <https://doi.org/10.1016/j.chest.2019.09.002>
 24. Randerath W, Bassetti CL, Bonsignore MR et al (2018) Challenges and perspectives in obstructive sleep apnoea: Report by an *ad hoc* working group of the sleep disordered breathing group of the European Respiratory Society and the European Sleep Research Society. Eur Respir J 52:1702616. <https://doi.org/10.1183/13993003.02616-2017>
 25. Jorquera J, Dreyse J, Salas C et al (2023) Clinical application of the multicomponent grading system for sleep apnea classification and incident cardiovascular mortality. Sleep Sci 16:e446–e453. <https://doi.org/10.1055/s-0043-1776770>
 26. Tong J, Yu Q, Li Y et al (2023) Obstructive sleep apnea and cardiovascular events in acute coronary syndrome: a meta-analysis. Coron Artery Dis 34:177–184. <https://doi.org/10.1097/MCA.0000000000001207>
 27. Zhang X, Fan J, Guo Y et al (2020) Association between obstructive sleep apnoea syndrome and the risk of cardiovascular diseases: an updated systematic review and dose–response meta-analysis. Sleep Med 71:39–46. <https://doi.org/10.1016/j.sleep.2020.03.011>
 28. Roth GA, Mensah GA, Johnson CO et al (2020) Global burden of cardiovascular diseases and risk factors, 1990–2019. J Am Coll Cardiol 76:2982–3021. <https://doi.org/10.1016/j.jacc.2020.11.010>
 29. Oldenburg O, Wellmann B, Buchholz A et al (2016) Nocturnal hypoxaemia is associated with increased mortality in stable heart failure patients. Eur Heart J 37:1695–1703. <https://doi.org/10.1093/eurheartj/ehv624>
 30. Wang L, Wei D, Zhang J, Cao J (2022) Time under 90% oxygen saturation and systemic hypertension in patients with obstructive sleep apnea syndrome. Nat Sci Sleep 14:2123–2132. <https://doi.org/10.2147/NSS.S388238>
 31. Guilleminault C (2001) Excessive daytime sleepiness: a challenge for the practising neurologist. Brain 124:1482–1491. <https://doi.org/10.1093/brain/124.8.1482>
 32. Ulander M, Hedner J, Stillberg G et al (2022) Correlates of excessive daytime sleepiness in obstructive sleep apnea: results from the nationwide SESAR cohort including 34,684 patients. J Sleep Res 31:e13690. <https://doi.org/10.1111/jsr.13690>
 33. Kleindorfer DO, Towfighi A, Chaturvedi S et al (2021) 2021 Guideline for the prevention of stroke in patients with stroke and transient ischemic attack: A guideline from the American Heart Association/American Stroke Association. Stroke 52. <https://doi.org/10.1161/STR.0000000000000375>
 34. Schwartz AR, Patil SP, Squier S et al (2010) Obesity and upper airway control during sleep. J Appl Physiol 108:430–435. <https://doi.org/10.1152/jappphysiol.00919.2009>
 35. Gottlieb DJ, Punjabi NM (2020) Diagnosis and management of obstructive sleep apnea: a review. JAMA 323:1389. <https://doi.org/10.1001/jama.2020.3514>
 36. Gonzaga C, Bertolami A, Bertolami M et al (2015) Obstructive sleep apnea, hypertension and cardiovascular diseases. J Hum Hypertens 29:705–712. <https://doi.org/10.1038/jhh.2015.15>
 37. Sánchez-de-la-Torre M, Gracia-Lavedan E, Benitez ID et al (2023) Adherence to CPAP treatment and the risk of recurrent cardiovascular events: a meta-analysis. JAMA 330:1255. <https://doi.org/10.1001/jama.2023.17465>
 38. Barrett JA (2017) A restless farewell in changing times: advice for the field of sleep medicine. J Clin Sleep Med 13:655–656. <https://doi.org/10.5664/jcsm.6572>
 39. Kim J-W, Won T-B, Rhee C-S et al (2020) Polysomnographic phenotyping of obstructive sleep apnea and its implications in mortality in Korea. Sci Rep 10:13207. <https://doi.org/10.1038/s41598-020-70039-5>

Publisher's Note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Springer Nature or its licensor (e.g. a society or other partner) holds exclusive rights to this article under a publishing agreement with the author(s) or other rightsholder(s); author self-archiving of the accepted manuscript version of this article is solely governed by the terms of such publishing agreement and applicable law.