ERS Congress 2025: highlights from the Sleep Disordered Breathing Assembly

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Shareable abstract (@ERSpublications)
Sleep medicine is shifting towards precision care, using pathophysiology, continuous data and personalised biomarkers to address disease heterogeneity. Future practice will be data-driven, gender-aware, environment-informed and patient-centred. https://bit.ly/44W914E

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The European Respiratory Society (ERS) Congress 2025, held in Amsterdam in September, highlighted remarkable progress in sleep disordered breathing research: the sessions underscored the multifactorial nature of obstructive sleep apnoea (OSA), highlighting the impact of disease variability, novel biomarkers, sex-specific differences and innovative therapeutic strategies aimed at improving patient outcomes. Emerging diagnostic tools and individualised treatment approaches are shaping a more accurate and patient-centred future.

Understanding disease variability and its impact on the management of OSA

The understanding of disease variability in OSA has evolved substantially, challenging the traditional static concept of disease severity. Recent evidence presented at the ERS Congress highlighted that OSA is not a fixed trait but a dynamic condition influenced by environmental, behavioural and technical factors: in particular variations in ambient temperature, circadian timing, alcohol use, and comorbidities can alter the severity and physiological impact of respiratory events. These findings suggest that apnoea–hypopnoea index (AHI) can fluctuate across nights and contexts: in this sense, the notion of "social apnoea", describing worsening of respiratory events during weekends or irregular sleep schedules, underscores the importance of lifestyle and circadian influences [1]. Beyond individual behaviour, climate-related stressors such as heatwaves have emerged as potential amplifiers of nocturnal hypoxaemia, linking OSA pathophysiology to global environmental changes [2]. Such variability has practical implications: single-night diagnostic studies may misclassify up to one-third of patients, calling for longitudinal monitoring and adaptive treatment strategies [3]. From a therapeutic perspective, variability also extends to the response and adherence to continuous positive airway pressure (CPAP) therapy [4]. Device-derived data show that residual AHI can vary markedly across nights due to mask leaks, cardiac decompensation, or opioid and alcohol intake [5–7]. Consequently, positive airway pressure telemonitoring should not merely quantify usage but contextualise nightly variability and identify technical or physiological causes of treatment failure [8, 9]. These insights encourage a shift from a one-size-fits-all threshold approach towards a multidimensional and patient-centred interpretation of CPAP data. Importantly, recent pooled analyses of cardiovascular outcome trials reinforce the need to integrate disease variability into OSA phenotyping. CPAP appears to confer cardiovascular protection primarily in high-risk phenotypes characterised by a greater hypoxic burden and stronger heart-rate response [10]. This supports a paradigm shift towards precision medicine in sleep apnoea where variability is not noise, but a defining feature of disease

expression and prognosis. Ultimately, recognising and quantifying night-to-night variability represent essential steps towards personalised management [11]. Incorporating longitudinal data, environmental influences and physiological markers could refine OSA risk stratification, improve adherence strategies, and enhance cardiovascular outcome prediction.

Central sleep apnoea: from pathophysiology over phenotypes to updated treatment recommendations

Just as OSA is not considered a fixed trait, central sleep apnoea (CSA) is increasingly recognised as a multifaceted disorder, extending beyond its classical association with heart failure. Recent discussions have redefined CSA as a spectrum of ventilatory control instabilities rather than a single entity. This refined understanding has prompted a more mechanistic approach to therapy: optimising cardiac function and circulatory dynamics can reduce the delay that contributes to ventilatory instability [12]. Pharmacological modulation of chemosensitivity and CO_2 reserve, through agents such as acetazolamide or serotonin agonists, has demonstrated the potential to stabilise breathing patterns. Adjusting the arousal threshold with cautious sedative use represents another dimension of personalised management, aiming to prevent recurrent arousal-induced ventilatory oscillations [13].

The new ERS statement clarifies the role of adaptive servo-ventilation (ASV) following the findings from new evidence such as the ADVENT-HF-RCT and the FACE study [14, 15]. Modern ASV devices, equipped with different algorithms, are effectively and safely used not only for idiopathic, opioid-induced, and treatment-emergent CSA, improving sleep quality and quality of life [16], but also for CSA in heart failure. In heart failure, treatment selection now follows a more nuanced gradient: ASV remains appropriate in patients with predominant OSA even when left ventricular ejection fraction (LVEF) is $\leqslant$45%, should be used cautiously in those with predominant CSA and LVEF 30–45%, and is part of a palliative symptom-oriented concept in CSA in LVEF <30% [16]. Treatment-emergent CSA, defined as the emergence of central events (central apnoea index $\geqslant$5 events·h^{-1} and >50% of total events) after CPAP eliminates obstructive events, has gained renewed attention as a marker of ventilatory instability rather than a benign transient phenomenon. Determinants include older age, male sex, low body mass index, heart disease, opioid use, high arousal index, high central/mixed apnoea index, high titration pressures and mask leaks [17]. American Academy of Sleep Medicine guidelines recommend active management with ASV for persistent treatment-emergent CSA as first-line and acetazolamide as an adjunct, to enhance ventilatory control and adherence to therapy [18]. In idiopathic CSA, mirtazapine and zolpidem has been discussed to raise arousal threshold, but drug therapy (except for acetazolamide) has not been included in treatment recommendation so far [19, 20]. Finally, in selected refractory cases with symptomatic CSA in heart failure or idiopathic CSA (and absence of OSA), phrenic-nerve stimulation represents an additional option with sustained improvements in oxygenation and symptom burden [21]. Collectively, these updates mark a decisive shift towards a mechanism-driven, phenotype-oriented approach to CSA. Therapeutic strategies are no longer defined by apnoea indices alone, but by the underlying control-system dynamics, comorbidities, and individual physiological traits that shape the disorder.

New markers in OSA: do they reflect the burden of disease?

A major theme emerging from the discussions at the 2025 Congress was the urgent need to move beyond the AHI. Traditional event-counting fails to capture the physiological stress imposed by respiratory disturbances. Instead, new quantitative markers are now redefining disease assessment by integrating the depth, duration, and systemic consequences of nocturnal events. Among these, the hypoxic burden has gained the greatest momentum. By integrating the area under all desaturation curves related to respiratory events, hypoxic burden provides a continuous measure of cumulative hypoxaemia that more accurately reflects the dose of intermittent hypoxia experienced overnight [22, 23]. *Post hoc* analyses from large cardiovascular trials have shown that hypoxic burden, but not AHI, independently predicts major cardiovascular outcomes, particularly in patients with acute coronary syndromes [24]. These findings suggest that cumulative hypoxia, rather than event frequency, drives vascular risk and should be central to risk stratification models.

Complementary to hypoxic burden, the delta heart rate response (ΔHR), which represents the difference between the maximum pulse rate after airway reopening and the minimum pulse rate during respiratory events, captures the autonomic cardiovascular response in OSA. Individuals with OSA who demonstrate an elevated ΔHR are at increased risk of cardiovascular morbidity and mortality; as a result, patients with a high ΔHR derived cardiovascular benefit from CPAP, whereas those with blunted responses did not [25].

A third emerging biomarker is the pulse wave amplitude drops (PWAD) index, derived from photoplethysmography signals. PWAD quantifies transient vasoconstrictive responses to respiratory events,

providing a non-invasive measure of autonomic activation and vascular tone: lower PWAD values are consistently associated with systemic hypertension, diabetes mellitus, and endothelial dysfunction, linking autonomic impairment to cardiometabolic risk in OSA [26].

When integrated, the aforementioned multidimensional markers (hypoxic burden, ΔHR and PWAD) capture distinct yet complementary aspects of OSA pathophysiology: hypoxaemic load, autonomic reactivity, and vascular responsiveness. Preliminary data on cohort studies have confirmed that their combined evaluation predicts cardiovascular morbidity and all-cause mortality more accurately than AHI alone. Collectively, this shift towards composite physiological metrics marks a turning point in OSA research and clinical management. The future lies in quantifying burden rather than counting events, enabling more precise phenotyping, better prediction of cardiovascular outcomes, and truly personalised approaches to CPAP therapy and beyond.

Sex differences in sleep disordered breathing: how do they influence diagnostics and treatment recommendations?

Sex differences in OSA have emerged as one of the most compelling themes in recent research, reshaping the understanding of disease pathophysiology [27], clinical expression, and therapeutic response [28]. Premenopausal women exhibit a lower prevalence of OSA. However, the prevalence and severity of OSA increases post-menopause [29]. Beyond prevalence, the pattern of respiratory disturbance differs substantially. Women tend to present with lower arousal threshold, a predominance of hypopnoeas rather than apnoeas, and respiratory-effort-related arousals with minimal oxygen desaturation: these features often culminate in rapid eye movement OSA, upper airway resistance syndrome and flow limitations patterns; forms easily overlooked by conventional scoring systems and contributing to persistent underdiagnosis [30]. Clinically, the female OSA phenotype is characterised by fatigue, insomnia, mood disturbance, and morning headaches rather than loud snoring or witnessed apnoeas [31]. This atypical symptom profile often leads to delayed diagnosis or misclassification as insomnia or depression. Women generally require lower CPAP pressures and may exhibit poorer adherence, partly due to less symptomatic sleepiness and greater sleep fragmentation [29].

These differences call for treatment strategies guided by symptom burden and functional impairment rather than by AHI thresholds alone.

Sex-based disparities extend beyond OSA. In obesity hypoventilation syndrome (OHS), female patients remain underdiagnosed and undertreated. Recent analyses reveal that women are less likely to receive timely non-invasive ventilation, reflecting potential diagnostic bias and reduced referral to specialised sleep centres [32, 33]. Heightened awareness of sex-related phenotypes could therefore improve recognition and optimise timing of non-invasive ventilation initiation in female patients. Additionally, CSA also exhibits sex-dependent expression. While men predominate in heart-failure-related forms and other high loop gain CSA, women are more affected by opioid-induced and insomnia-associated CSA, where ventilatory-drive suppression, hyperarousal and autonomic instability prevail [34, 35]. In these contexts, central events tend to be deeper and more prolonged, amplifying sympathetic activation and cardiovascular risk [36]. Overall, sex differences shape the full spectrum of sleep disordered breathing: from pathophysiology to clinical management. Recognising these distinctive phenotypes is pivotal to achieving equitable, personalised care, moving towards a precision-medicine approach that truly integrates sex as a biological variable in respiratory sleep medicine.

Emerging insights in diagnosis and treatment of sleep disordered breathing

A theme that emerged during the ERS Congress oral presentation session was the expected global rise in the prevalence of OSA. While previous predictions had already suggested an increase in OSA prevalence in America, the oral session presented new data predicting a similar trend for Europe [37]. Specifically, the prevalence of OSA among adults aged 30–69 years is projected to increase from 36% in 2020 to 59% by 2050. Although the absolute number of individuals affected by OSA may not increase in some countries due to population decline, the growth in prevalence is due to an accumulation of risk factors. Therefore, it is crucial to take preventive measures to control this rise and prevent OSA from becoming one of Europe's major health burdens. The session also discussed the potential impact of rising global temperatures on the prevalence of OSA. This association was estimated using data collected from an under-mattress monitoring device, suggesting that environmental changes may exacerbate the problem [38].

In the same session, several therapeutic approaches for specific subgroups of patients with sleep-related breathing disorders were also discussed. A *post hoc* analysis of the ADVENT trial examined the cycle length of Cheyne–Stokes respiration and identified a phenotype characterised by longer cycle lengths [39].

These patients had more advanced heart failure and higher mortality rates. Importantly, this subgroup showed a mortality benefit when treated with peak flow-triggered ASV. Another study focused on patients with OHS, who are known to have a suppressed ventilatory drive and blunted responses to hypercapnia: a randomised trial evaluated a combination therapy of acetazolamide (a carbonic anhydrase inhibitor that stabilises respiratory patterns, increases ventilatory drive and reduces hypoventilation) and atomoxetine (which increases pharyngeal muscle tone). The combination improved CO_2 levels, oxygenation and AHI, while also increasing chemosensitivity in treatment-naïve OHS patients [40]. These findings open new perspectives for pharmacological therapy targeting the pathophysiology of OHS.

Different clinical aspects of sleep and OSA according to gender, anthropometrics and symptoms

Preliminary studies presented during one of the poster sessions explored how gender, anthropometric measures, symptoms and comorbidities influence the clinical presentation, diagnosis and therapeutics of OSA. Several studies offered a deeper look into the symptomatology of OSA, particularly daytime sleepiness, focusing on its progression over time, and on factors affecting its severity [41, 42]. The presented data demonstrated the use of different OSA screening tools based on variables such as gender, neck circumference, and body mass index, highlighting the clear advantages of using these variables as predictors with established diagnostic accuracy. These conclusions were further supported by comparative analyses of the sensitivity and specificity of different screening tools, which underscored the superiority of anthropometry-based screening approaches [43, 44].

In parallel, investigators examined the severity of the symptomatic profile in patients with OSA in correlation to various predictors, including gender and anthropometric factors [45]. Based on these findings, an interpretable model incorporating specific physical and clinical variables was proposed to stratify patients according to disease severity [46]. These insights were further enriched by an exploration of the factors contributing to the underdiagnosis of OSA cases [47].

From a therapeutic perspective, the application of patient-reported outcome measures such as the Pittsburgh Sleep Quality Index, Beck Depression Inventory, and Insomnia Severity Index revealed distinct gender-based symptomatic patterns, suggesting potential benefits of sex-specific therapeutic approaches [48]. Another unique perspective was presented through an analysis of data from the European Sleep Apnoea Database (ESADA), which revealed a link between the spoken language and the severity of OSA symptoms [49].

Collectively, these findings establish the need for a habitus-based and gender-aware approach to sleep disordered breathing, beginning with symptom-driven suspicion and the use of appropriate diagnostic tools, and extending through to the management plan.

Association of sleep disordered breathing and different comorbidities

During the session on the association between sleep disordered breathing and various comorbidities, several studies underscored the strong and multifaceted links between OSA and cardiovascular as well as systemic conditions.

Across different cohorts, OSA frequently appeared in patients with cardiovascular disease, even when classic symptoms such as excessive daytime sleepiness were minimal, and its presence was often confirmed at high rates [50]. Evidence from large population studies (such as the HypnoLaus study) showed that increasing OSA severity was associated with a higher burden of cardiac arrhythmias and elevated long-term cardiovascular risk [51], particularly when OSA coexisted with insomnia [52]. Obesity also emerged as a key contributor, demonstrating a dose–response relationship with cardiovascular risk in combination with OSA [53]. Beyond cardiovascular outcomes, specific comorbidity profiles were described: patients with gastro-oesophageal reflux disease tended to be younger and more symptomatic without greater OSA severity [54]. Meanwhile, in obese patients, coexisting OHS worsened oxygen desaturation and arousal indices, indicating more severe ventilatory impairment [55]. In the ESADA cohort, stroke patients exhibited a heavier cardiometabolic burden with high rates of systemic hypertension, ischaemic heart disease and diabetes mellitus [56]. Finally, analyses in the same cohort also indicated that nervous-system medications did not substantially influence OSA severity [57].

Conclusion

The ERS Congress 2025 underscored a pivotal transition in the field of sleep disordered breathing from traditional diagnostic paradigms to a precision medicine approach rooted in pathophysiology, individual variability, and multidimensional biomarkers (figure 1). The growing recognition of disease heterogeneity

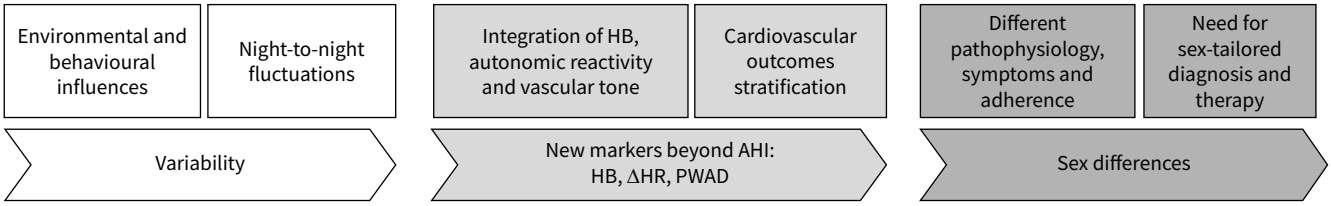

FIGURE 1 Conceptual summary of the European Respiratory Society 2025 Assembly 4 highlights. Advances in sleep medicine move from understanding variability in obstructive sleep apnoea expression, to the development of multidimensional markers that reflect disease burden and the recognition of sex-specific phenotypes requiring tailored diagnostic and therapeutic approaches. AHI: apnoea–hypopnoea index; HB: hypoxic burden; ΔHR: delta heart rate response; PWAD: pulse wave amplitude drops.

in OSA and CSA has transformed both research and clinical practice, encouraging the use of continuous and contextual data to better predict outcomes and tailor interventions.

Collectively, the insights presented delineate a clear trajectory for the future of sleep medicine: one that is data-rich, gender-aware, environmentally conscious and profoundly patient-centred. As precision sleep medicine continues to evolve, collaboration across disciplines will be essential to translate these scientific advances into tangible improvements in global respiratory health.

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