



From Cause to Treatment: Obstructive Sleep Apnoea as a Driver of Hypertension and a Target for Prevention

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AT THE EUROPEAN Respiratory Society (ERS) Congress 2026 in Barcelona, Spain, specialists from across the globe came together to collaborate and showcase the latest breakthroughs in respiratory medicine. This informative session, chaired by Winfried Randerath, Solingen, Germany; Matteo Ciciliano, Rome, Italy; and Sophie E. Schiza, Heraklion, Greece, featured four insightful talks centred around the association between obstructive sleep apnoea and hypertension.

PATHOPHYSIOLOGY TIES OSA TO HYPERTENSION

Esther Irene Schwarz, Department of Pulmonology, University of Zurich, Switzerland, opened the session by exploring the pathophysiological link between obstructive sleep apnoea (OSA) and hypertension, driven by sympathetic overactivity and vascular dysfunction.

A higher proportion of patients with OSA experience hypertension than in the general population. In sleep clinic cohorts with OSA, defined by an Apnoea-Hypopnea Index

(AHI) $\geq 5/h$, 40–45% develop hypertension, 60% develop nocturnal hypertension or non-dipping blood pressure, and 10–20% have resistant hypertension. Similar patterns were observed across patients with severe OSA (AHI $\geq 30/h$), rapid eye movement–OSA, OSA with obesity, and OSA with substantial nocturnal hypoxaemia.^{1–3}



A higher proportion of patients with OSA experience hypertension than in the general population



When sleeping with OSA and intermittent hypoxia, the sympathetic nervous system surges and stimulates abnormal vasoconstriction, which leads to elevated blood pressure.^{4,5} If this is sustained, it can cause endothelial dysfunction, characterised by oxidative stress, inflammation, and decreased nitric oxide. This has been further supported by studying patients withdrawing from continuous positive airway pressure (CPAP), which has demonstrated increases in urinary catecholamines, markers of sympathetic activity, and blood pressure. At the same time, the flow-mediated dilation was shown to decrease, representing impaired endothelial function.^{6,7}

Interestingly, age affects this physiological response, with younger and middle-aged individuals demonstrating a stronger association between OSA and nocturnal hypoxaemia and increased sympathetic activity. The risk of hypertension in individuals with OSA may diminish with age.^{8,9} Although the role of sex has not yet been studied systematically, Schwarz highlights that intermittent hypoxia increases sympathetic activity in both sexes, but the risk of hypertension in women with OSA is likely to be higher.

Schwarz then described how an increase in blood pressure due to OSA can blunt the natural nocturnal dipping pattern and develop into sustained hypertension. As the blood pressure is still lower than normal during this period, the hypertension is masked. Additionally, arousal-associated repetitive blood pressure surges, sustained daytime hypertension, and impaired baroreflex function should be carefully monitored.

MOVING BEYOND CPAP TOWARDS OSA THERAPIES

The session then continued with Peter A Cistulli, Professor of Sleep Medicine, University of Sydney, Australia, who explores the effectiveness of OSA therapies for blood pressure control beyond CPAP, which has been the gold-standard treatment. He emphasises that a large amount of heterogeneity exists, given that systolic and diastolic blood pressure data following CPAP vary across populations.¹⁰

The results from the ANDANTE meta-analysis collaboration, which involved studying 9,434 patients across 36 RCTs,

showed that CPAP only lowers blood pressure where it is uncontrolled, which aligns with results from the previous HIPARCO study.^{11,12}

Patients with OSA who undergo CPAP therapy are thought to reduce their risk of incident hypertension by almost 30%, according to research by Marin et al.,¹³ and nonadherence to CPAP therapy was found to be associated with an increase in 30-day all-cause and cardiovascular-cause readmission in patients with OSA.^{13,14}

Moving on to CPAP alternatives, Cistulli describes how oral appliances typically have lower efficacy yet demonstrate a higher level of compliance from patients, therefore providing a similar overall effectiveness.¹⁵ He highlighted the CRESCENT study, a comparison of 24-hour blood pressure between mandibular advancement devices (MAD) and CPAP. MAD therapy showed greater reductions in 24-hour blood pressure at the non-inferiority primary endpoint of 6 months, while nearly 60% of patients using MAD therapy used it for more than 6 hours per night, compared with 23% for patients with CPAP, showing a marked difference.¹⁶





BIOMARKERS CARVING THE PATH TO PERSONALISED MEDICINE

For the following presentation, Silke Ryan, University College Dublin, Ireland, explored how biomarkers and risk models can be used to create personalised treatment strategies for OSA. She opened the talk by highlighting the role of OSA as both an independent contributor to hypertension and a contributor to the complex gene-environment interactions involved in the pathophysiology of hypertension. She reinforces the significance of OSA muting the blood pressure overnight dipping pattern, which supports performing 24-hour ambulatory blood pressure monitoring (ABPM) as both a biomarker and clinical endpoint.^{23,24}

Ryan outlines how this can serve as a biomarker for CPAP response. Studies have shown that patients with nocturnal hypertension and a non-dipping blood pressure profile are more likely to respond positively to CPAP.²⁵

Key markers in pathophysiological mechanisms, such as sympathetic activation, can further serve as biomarkers for hypertension. Hypoxic burden has demonstrated an association with both adverse outcomes and a positive response to CPAP.²⁶ However, there is limited data on its association with hypertension. In a multi-ethnic study of atherosclerosis, increased hypoxic burden was associated independently with increased blood pressure, particularly in patients who were not receiving hypertension medication.²⁷ Furthermore, the HeartBEAT study demonstrated that a greater reduction in hypoxic burden following CPAP treatment was independently associated with systolic blood pressure improvements.²⁸ Other pathophysiological biomarkers may include those of systemic inflammation, oxidative stress, endothelial dysfunction, and vascular remodelling. However, Ryan emphasises that many biomarkers from these areas may have limited specificity due to their wide involvement in other conditions.

He then outlines the impacts of weight loss on hypertension as an upstream driver of OSA. A trial demonstrated that after 6 months, weight loss coupled with CPAP led to a significant improvement in systolic blood pressure, compared with either intervention alone.¹⁷ This is supported by the SURMOUNT-OSA study, demonstrating that tirzepatide is effective for obesity-related OSA.^{18,19}

Emerging pharmacological OSA therapies include acetazolamide and sulthiame, both carbonic anhydrase inhibitors, aroxylbutynin, and atomoxetine.²⁻²²

Cistulli summarised his presentation by describing the effect of CPAP on blood pressure as modest and heterogeneous. MAD, together with weight management and emerging drugs, may expand the selection of options; however, differential blood pressure outcomes must be considered.

Ryan turned the discussion to molecular markers such as microRNAs (miRNA): small, non-coding RNAs involved in the regulation of gene expression. A cluster of miRNAs has shown an association with a favourable blood pressure response to CPAP.³⁰ Similarly, researchers have shown that exosomes of patients with reverse dipping blood pressure, in which the blood pressure dips during the day, showed differentially expressed miRNA that promoted vascular damage and led to increased permeability of an endothelial cell monolayer *in vitro*.³⁰

She concluded her presentation by suggesting that an ideal biomarker may not be possible due to the complexity of the condition: one that has a high sensitivity, high specificity, high reproducibility, early detection, and is easy to obtain and assay. Instead, multicomponent models should be developed and evaluated in large cohorts and subsequently in RCTs.

PHENOTYPING OF PATIENTS WITH OSA

Jean-Louis Pépin then began his presentation by describing the many faces of OSA hypertension that can be observed from ABPM measurements, with varying dipping patterns and surges. The interplay between hypertension phenotypes and OSA phenotypes can be complex when considering comorbidities. He reinforced a point made by previous speakers: ABPM and identification of hypertension phenotypes should be standardised practice. Pépin outlined complex clinical cases that demonstrate this and further reinforced the association between a non-dipping nocturnal blood pressure profile and CPAP effectiveness. However, nocturnal hypertension is not only related to OSA but also to insomnia and restless leg movement. Therefore, improving sleep quality should be prioritised to lower blood pressure.³¹ Additionally, the OMAN trial showed that a greater reduction in blood pressure can be achieved when blood pressure medication is taken at bedtime.³² However, this has not yet been proven in patients with OSA.

Pépin goes on to recommend that the impact of CPAP therapy should be monitored for the first 6 months of care. A study by Coiffier O et al.³³ showed that 6 months of CPAP may lead to a higher reduction in evening blood pressure compared with morning blood pressure, which is more difficult to control.³³ Pépin then referred to the key findings of the SURMOUNT-OSA trial, as presented by Cistulli, and concluded by reinforcing that in patients living with obesity, combining CPAP with weight loss strategies is essential to lower blood pressure.^{19,20}

THE PATH FORWARD

Overall, the session highlighted the complex relationship between OSA and hypertension, demonstrating how intermittent hypoxia, sympathetic activation, and vascular dysfunction can contribute to adverse blood pressure profiles. While CPAP remains an important treatment, its effects on blood pressure are modest and heterogeneous, reinforcing the need for individualised approaches. Alternative therapies, including mandibular advancement devices, weight management, and emerging pharmacological treatments, may broaden therapeutic options. The growing role of ABPM, hypoxic burden, and molecular biomarkers also offers opportunities to identify patients most likely to benefit from specific interventions. Ultimately, integrating OSA phenotypes, hypertension profiles, and patient characteristics may support more personalised and effective management strategies.

“Patients with nocturnal hypertension and a non-dipping blood pressure profile are more likely to respond positively to CPAP”

References

1. Tkacova R et al. Nocturnal intermittent hypoxia predicts prevalent hypertension in the European Sleep Apnoea Database cohort study. *Eur Respir J*. 2014; 44(4):931-41.
2. Hedner J et al. The European Sleep Apnoea Database (ESADA): report from 22 European sleep laboratories. *Eur Respir J*. 2011;38(3):635-42.
3. Show KL et al. Global prevalence of nocturnal hypertension: systematic review and meta-analysis. *BMC Global Health*. 2026;26(1):2358.
4. Gruber B et al. Impact of obstructive sleep apnoea on nocturnal blood pressure: mechanisms and treatment responses – a narrative review. *Eur Respir Rev*. 2026; 35(181):26019.
5. Lévy P et al. Obstructive sleep apnoea syndrome. *Nat Rev Dis Primers*. 2015; 1:15015.
6. Kohler M et al. Effects of continuous positive airway pressure therapy withdrawal in patients with obstructive sleep apnea: a randomized controlled trial. *Am J Respir Crit Care Med*. 2011;184(10):1192-9.
7. Schwarz EI et al. Physiological consequences of CPAP therapy withdrawal in patients with obstructive sleep apnoea—an opportunity for an efficient experimental model. *JTD*. 2017;10(Suppl 1):S24-32.
8. Puzino K et al. 0488 Poor Sleep and Daytime Sleepiness Increase the Risk of Hypertension Associated with Mild-to-Moderate Obstructive Sleep Apnea: Age Effect. *Sleep*. 2019;42(Suppl 1):A195-6.
9. O'Connor GT et al. Prospective study of sleep-disordered breathing and hypertension: the sleep heart health study. *Am J Respir Crit Care Med*. 2009;179(12):1159-64.
10. Shiina K. Obstructive sleep apnea-related hypertension: a review of the literature and clinical management strategy. *Hypertens Res*. 2024;47(11):3085-98.
11. Pengo MF et al.; ANDNATE collaborators. Effect of CPAP therapy on blood pressure in patients with obstructive sleep apnoea: a worldwide individual patient data meta-analysis. *Eur Respir J*. 2025;65(1):2400837.
12. Martínez-García MA et al.; Spanish Sleep Network. Effect of CPAP on blood pressure in patients with obstructive sleep apnea and resistant hypertension: The HIPARCO randomized clinical trial. *JAMA*. 2013;310(22):2407-15.
13. Marin JM et al. Association between treated and untreated obstructive sleep apnea and risk of hypertension. *JAMA*. 2012;307(20):2169-76.
14. Truong KK et al. Nonadherence to CPAP associated with increased 30-day hospital readmissions. *J Clin Sleep Med*. 2018;14(2):183-9.
15. Sutherland K et al. Oral appliance treatment response and polysomnographic phenotypes of obstructive sleep apnea. *J Clin Sleep Med*. 2015;11(8):861-8.
16. Ou YH et al. Mandibular advancement vs CPAP for blood pressure reduction in patients with obstructive sleep apnea. *J Am Coll Cardiol*. 2024;83(18):1760-72.
17. Chirinos JA et al. CPAP, weight loss, or both for obstructive sleep apnea. *N Engl J Med*. 2014;370:2265-75.
18. Malhotra A et al. Tirzepatide for the treatment of obstructive sleep apnea and obesity. *N Engl J Med*. 2024;391:1193-205.
19. Malhotra A et al. Tirzepatide on obstructive sleep apnea-related cardiometabolic risk: secondary outcomes of the SURMOUNT-OSA randomized trial. *Nat Med*. 2026;32(2):653-9.
20. Eskandari D et al. Acetazolamide reduces blood pressure and sleep-disordered breathing in patients with hypertension and obstructive sleep apnea: a randomized controlled trial. *J Clin Sleep Med*. 2018;14(3):309-17.
21. Hedner J et al. A randomized controlled clinical trial exploring safety and tolerability of sulthiame in sleep apnea. *Am J Respir Crit Care Med*. 2022;205(12):1461-9.
22. Strollo PJ et al. Aroxycbutynin and atomoxetine (AD109) for obstructive sleep apnea: a randomized phase 3 trial (SynAIRgy). *Am J Respir Crit Care Med*. 2026;212(7):1569-84.
23. Boggia J et al.; International Database on Ambulatory blood pressure monitoring in relation to Cardiovascular Outcomes (IDACO) Investigators. Prognostic accuracy of day versus night ambulatory blood pressure: a cohort study. *Lancet*. 2007;370(9594):1219-29.
24. Sasaki N et al. Impact of non-dipping on cardiovascular outcomes in patients with obstructive sleep apnea syndrome. *Clin Exp Hypertens*. 2015;37(6):449-53.
25. Sapiña-Beltrán E et al.; on behalf of the Spanish Sleep and Breathing Group. Differential blood pressure response to continuous positive airway pressure treatment according to the circadian pattern in hypertensive patients with obstructive sleep apnoea. *Eur Respir J*. 2019;54(1):1900098.
26. Tasali E et al. Obstructive sleep apnea and cardiometabolic disease: obesity, hypertension, and diabetes. *Circ Res*. 2025;137(5):764-87.
27. Kim S et al. Relationship of obstructive sleep apnoea severity and subclinical systemic atherosclerosis. *Eur Respir J*. 2020;55(2):1900959.
28. Messineo L et al. Treatment of Sleep Apnea and Reduction in Blood Pressure: The Role of Heart Rate Response and Hypoxic Burden. *Hypertension*. 2024; 81(5):1106-14.
29. Sánchez-de-la-Torre M et al.; Spanish Sleep Network. Precision medicine in patients with resistant hypertension and obstructive sleep apnea: blood pressure response to continuous positive airway pressure treatment. *J Am Coll Cardiol*. 2015;66(9):1023-32.
30. Khalyfa A et al. Circulating plasma exosomes in obstructive sleep apnoea and reverse dipping blood pressure. *Eur Respir J* 2020;55(1):1901072.
31. Pépin JL et al. Hypertension and sleep: overview of a tight relationship. *Sleep Med Rev*. 2014;18(6):509-19.
32. Ye R et al. Morning vs bedtime dosing and nocturnal blood pressure reduction in patients with hypertension: the OMAN randomized clinical trial. *JAMA Netw Open*. 2025;8(7):e2519354.
33. Coiffier O et al. Home blood pressure trajectories during 6 months of continuous positive airway pressure therapy: results from the eMEUSE-SANTE and SLEEPCONNECT clinical trials. *Ann Am Thorac Soc*. 2026;23(1):117-24.