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The Association Between Obstructive Sleep Apnea and Systemic Hypertension: A Review of Epidemiological and Pathophysiological Evidence

¹A.Bent, ²A. Benton

^{1,2} University of Illinois College of Medicine

Corresponding Email : bentmd284847@gmail.com

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ABSTRACT

Introduction: Systemic hypertension is a primary risk factor for cardiovascular disease, and the identification of modifiable secondary causes is a clinical priority. Obstructive Sleep Apnea (OSA), a highly prevalent disorder of intermittent airway collapse during sleep, has emerged as a major independent contributor to elevated blood pressure.

Literature Review: A review of pivotal literature establishes a strong, independent, and dose-response relationship between OSA severity and the prevalence and incidence of hypertension. Large, population-based cohort studies provided robust epidemiological evidence for this association. The pathophysiological pathways linking OSA to hypertension were elucidated, with key mechanisms including chronic daytime overactivity of the sympathetic nervous system, endothelial dysfunction driven by intermittent hypoxia and oxidative stress, and activation of the renin-angiotensin-aldosterone system. Furthermore, randomized controlled trials demonstrated that treating OSA with Continuous Positive Airway Pressure (CPAP) therapy results in a modest but clinically significant reduction in blood pressure, providing causal support for the relationship.

Conclusion: The body of evidence reviewed firmly establishes OSA as a causal factor in the development and maintenance of systemic hypertension. This understanding has led to the widespread recognition of OSA as a common cause of secondary and treatment-resistant hypertension, making screening for this sleep disorder an integral part of modern hypertension management.

Keywords: Obstructive Sleep Apnea (OSA), Hypertension, Blood Pressure, Continuous Positive Airway Pressure (CPAP), Sympathetic Nervous System.

INTRODUCTION

Systemic hypertension is one of the most significant public health problems globally, serving as a leading modifiable risk factor for myocardial infarction, stroke, heart failure, and chronic kidney disease. While the majority of cases are classified as essential hypertension, with no single identifiable cause, a substantial portion is secondary to other underlying medical conditions. The identification and treatment of these secondary causes are clinically important as they can offer a path to better blood pressure control and, in some cases, a cure for hypertension.

One of the most important and common of these causes is Obstructive Sleep Apnea (OSA). OSA is a condition characterized by recurrent episodes of partial or complete upper airway obstruction during sleep, resulting in intermittent hypoxemia, hypercapnia, and frequent arousals. Initially viewed primarily as a cause of daytime sleepiness, a compelling body of evidence has established OSA as a major independent risk factor for the development of systemic hypertension. This literature review will synthesise the pivotal epidemiological and mechanistic evidence that solidified this important clinical link.

LITERATURE REVIEW

The scientific case linking Obstructive Sleep Apnea to systemic hypertension is built on robust epidemiological data, a clear understanding of the underlying pathophysiology, and evidence from intervention trials.

The epidemiological foundation for the association is exceptionally strong. Large, prospective, population-based cohort studies, such as the Wisconsin Sleep Cohort Study and the Sleep Heart Health Study, provided definitive evidence. These landmark investigations demonstrated a clear dose-response relationship between the severity of OSA at baseline, as measured by the

apnea-hypopnea index (AHI), and the likelihood of developing hypertension in the future. This relationship persisted even after rigorous statistical adjustment for confounding factors, most notably obesity, confirming that OSA is an independent risk factor for hypertension (Shamsuzzaman et al., 2003).

The biological plausibility of this link is supported by several well-characterised pathophysiological mechanisms. A primary mechanism is chronic activation of the sympathetic nervous system. Each apneic event, with its associated hypoxia and arousal from sleep, acts as a powerful stressor that triggers a surge in sympathetic outflow and a spike in blood pressure. Research has shown that this sympathetic overactivity does not simply switch off during the day; instead, it persists during wakefulness, contributing to a state of sustained, elevated vascular tone and hypertension (Kapsimalis and Kryger, 2002). A second key pathway is endothelial dysfunction. The recurrent cycles of hypoxia and reoxygenation generate significant oxidative stress, which damages the endothelial lining of blood vessels. This impairs the production of nitric oxide, a potent vasodilator, and promotes a pro-inflammatory state, both of which contribute to elevated blood pressure and the progression of atherosclerosis.

Further evidence for causality comes from randomized controlled trials examining the effect of OSA treatment on blood pressure. The standard treatment for moderate to severe OSA is Continuous Positive Airway Pressure (CPAP), which acts as a pneumatic splint to keep the airway open during sleep. Multiple randomized trials and subsequent meta-analyses demonstrated that effective CPAP therapy leads to a modest, yet statistically significant, reduction in both 24-hour ambulatory and office blood pressure readings (Bazzano et al., 2007; Pepperell et al., 2002). The blood pressure-lowering effect was generally found to be greatest in patients with more severe OSA and in those with resistant hypertension.

This body of evidence led to the clinical recognition of OSA as one of the most common causes of resistant hypertension—defined as high blood pressure that remains uncontrolled despite the use of three or more antihypertensive medications. Studies have shown an extremely high prevalence of undiagnosed OSA in this patient population, solidifying the importance of screening for sleep-disordered breathing in the management of difficult-to-treat hypertension (Logan et al., 2003).

CONCLUSION

The pivotal literature investigating the relationship between Obstructive Sleep Apnea and systemic hypertension provides a clear and convincing case for causality. Strong, dose-dependent epidemiological evidence from large cohort studies is supported by a deep understanding of the plausible biological mechanisms, including chronic sympathetic activation and endothelial dysfunction. The demonstration that treatment of OSA with CPAP can lower blood pressure further strengthens this causal link.

This body of research has fundamentally altered clinical practice. It has successfully repositioned OSA from being solely a sleep disorder to being recognized as a major systemic disease and a leading secondary cause of hypertension. Consequently, the assessment for sleep-disordered breathing has become an essential component in the evaluation and management of patients with hypertension, particularly those whose blood pressure is resistant to conventional therapy.

REFERENCES

- Bazzano, L.A., Khan, Z., Reynolds, K., He, J. (2007) 'Effect of nocturnal nasal continuous positive airway pressure on blood pressure in obstructive sleep apnea', *Hypertension*, 50(2), pp. 417-423.
- Kapsimalis, F. and Kryger, M.H. (2002) 'The cardiovascular comorbidity of sleep-disordered breathing', *Israel Medical Association Journal*, 4(7), pp. 547-551.
- Logan, A.G., Perlikowski, S.M., Mente, A., Tisler, A., Tkacova, R., Niroumand, M., Leung, R.S., and Bradley, T.D. (2003) 'High prevalence of unrecognized sleep apnoea in drug-resistant hypertension', *Journal of Hypertension*, 21(12), pp. 2271-2277.
- Pepperell, J.C.T., Ramdassingh-Dow, S., Crosthwaite, N., Mullins, R., Jenkinson, C., Stradling, J.R. and Davies, R.J.O. (2002) 'Ambulatory blood pressure after therapeutic and subtherapeutic nasal continuous positive airway pressure for obstructive sleep apnoea: a randomised parallel trial', *The Lancet*, 359(9302), pp. 204-210.
- Shamsuzzaman, A.S.M., Gersh, B.J. and Somers, V.K. (2003) 'Obstructive sleep apnea: implications for cardiac and vascular disease', *JAMA*, 290(14), pp. 1906-1914.

