

Sleep Apnea's Impact On Cardiovascular Health

Rina Perera*

Department of Respiratory Therapy and Care, University of Colombo, Colombo, Sri Lanka

Introduction

Sleep-related breathing disorders (SRBDs), particularly obstructive sleep apnea (OSA), are significantly associated with an elevated risk of cardiovascular diseases. These conditions contribute to a range of serious cardiovascular complications, including hypertension, coronary artery disease, heart failure, and arrhythmias. The underlying mechanisms involve intermittent hypoxia, persistent activation of the sympathetic nervous system, and the promotion of systemic inflammation. Effective management of SRBDs, often initiated through continuous positive airway pressure (CPAP) therapy, demonstrates a clear potential to mitigate these heightened cardiovascular risks [1]. The intricate relationship between sleep apnea and hypertension is notably significant, with OSA frequently identified as a common secondary cause of resistant hypertension. The apneic events observed during sleep trigger acute surges in blood pressure, primarily due to heightened sympathetic overactivity and the development of endothelial dysfunction. Crucially, treating OSA with CPAP therapy has been consistently shown to be effective in reducing blood pressure levels, especially in patients who exhibit resistance to conventional antihypertensive treatments [2]. The spectrum of cardiac manifestations associated with obstructive sleep apnea is notably diverse, encompassing a range of serious conditions. These include the development of left ventricular hypertrophy, impaired diastolic function, pulmonary hypertension, and a statistically increased incidence of both myocardial infarction and stroke. These detrimental complications primarily arise from the recurrent episodes of oxygen desaturation and hypercapnia characteristic of OSA, which collectively lead to increased oxidative stress and chronic inflammation within the cardiovascular system [3]. Atrial fibrillation is a cardiac arrhythmia that is frequently observed in individuals diagnosed with sleep apnea. Emerging evidence suggests that OSA may play a substantial role in both the initiation and the ongoing maintenance of this arrhythmia. The increased atrial stretch and elevated atrial pressure commonly associated with OSA, combined with significant autonomic dysregulation, can collectively promote the development and persistence of these abnormal heart rhythms [4]. Continuous positive airway pressure (CPAP) therapy stands as the cornerstone for the effective management of OSA and has demonstrably shown significant benefits in reducing the incidence of cardiovascular events. Numerous studies have documented that CPAP therapy can lead to improvements in endothelial function, a notable reduction in sympathetic nervous system activity, and a lowering of blood pressure, thereby effectively mitigating the overall cardiovascular risk in patients diagnosed with OSA [5]. The critical role of intermittent hypoxia in the pathogenesis of cardiovascular disease associated with sleep apnea cannot be overstated. This recurring and cyclical lack of adequate oxygenation during sleep triggers a complex cascade of inflammatory and oxidative stress pathways. These pathways are instrumental in damaging the vascular endothelium, a critical component of cardiovascular health, and actively promoting the development and progression of atherosclerosis [6]. While obstructive sleep apnea receives considerable attention, central sleep apnea (CSA) also carries significant and often

underappreciated cardiovascular implications. This is particularly evident in patient populations already suffering from heart failure. CSA can exacerbate existing cardiac dysfunction and profoundly influence the effectiveness of various treatment strategies, underscoring the critical need for its prompt recognition and comprehensive management [7]. The metabolic consequences stemming from sleep apnea, including the development of insulin resistance and dyslipidemia, represent additional significant factors that contribute to the markedly elevated cardiovascular risk observed in affected individuals. These metabolic disturbances, which are often intricately linked to the inflammatory and oxidative stress pathways activated by SRBDs, are independently recognized as substantial risk factors for the development and progression of atherosclerosis [8]. The relationship between sleep duration and overall cardiovascular health also holds considerable relevance in this context. Both excessively short and excessively long sleep durations, conditions often associated with various sleep disorders, can exert a detrimental impact on cardiovascular health. This negative influence can occur either independently or in conjunction with other breathing disturbances related to sleep [9]. Future research endeavors should be strategically directed towards the development and implementation of personalized treatment approaches for sleep-related breathing disorders and their associated cardiovascular sequelae. This forward-looking perspective includes a dedicated exploration of novel therapeutic targets and a deeper understanding of the complex, multifactorial interplay between sleep, respiration, and cardiovascular function across a diverse array of patient populations [10].

Description

Sleep-related breathing disorders (SRBDs), with a particular focus on obstructive sleep apnea (OSA), are strongly implicated in the increased incidence of cardiovascular diseases. OSA is a significant contributing factor to hypertension, coronary artery disease, heart failure, and arrhythmias. These adverse cardiovascular outcomes are mediated through physiological mechanisms such as intermittent hypoxia, heightened sympathetic nervous system activation, and chronic systemic inflammation. Consequently, the effective management of SRBDs, most commonly achieved through continuous positive airway pressure (CPAP) therapy, plays a vital role in attenuating these associated cardiovascular risks [1]. The interaction between sleep apnea and hypertension is profoundly significant, with OSA frequently identified as a primary driver of resistant hypertension. Apneic episodes during sleep precipitate abrupt elevations in blood pressure due to excessive sympathetic nervous system activity and the compromised function of the endothelium. Importantly, the application of CPAP therapy for OSA has demonstrated efficacy in lowering blood pressure, particularly in individuals with resistant hypertension [2]. The cardiovascular manifestations of obstructive sleep apnea are multifaceted and include several critical conditions. These encompass the development of left ventricular hypertrophy, impaired diastolic function of the heart, pulmonary hypertension, and an elevated risk of myocardial infarction and stroke. The underlying

pathology is rooted in the recurrent episodes of oxygen deprivation and elevated carbon dioxide levels characteristic of OSA, which collectively induce oxidative stress and inflammation within the cardiovascular system [3]. Atrial fibrillation is a common finding in patients diagnosed with sleep apnea, and OSA is believed to contribute to its onset and persistence. The mechanical stress on the atria and the increased atrial pressure induced by OSA, coupled with disruptions in autonomic regulation, can create an environment conducive to the development of arrhythmias [4]. Continuous positive airway pressure (CPAP) therapy is the established primary treatment for OSA and has consistently shown benefits in reducing the occurrence of cardiovascular events. Evidence from clinical studies indicates that CPAP can improve endothelial function, diminish sympathetic nervous system activity, and lower blood pressure, thereby effectively reducing cardiovascular risk in patients with OSA [5]. The pathogenic role of intermittent hypoxia in the development of cardiovascular disease in sleep apnea is a crucial area of understanding. This repeated reduction in oxygen levels initiates a series of inflammatory responses and oxidative stress pathways that ultimately result in damage to the vascular endothelium and promote the atherosclerotic process [6]. Central sleep apnea (CSA) also presents significant cardiovascular risks, particularly in individuals with pre-existing heart failure. CSA can worsen cardiac dysfunction and influence the patient's response to treatment, highlighting the necessity for its identification and appropriate management strategies [7]. Furthermore, the metabolic derangements associated with sleep apnea, such as insulin resistance and dyslipidemia, contribute substantially to the elevated cardiovascular risk profile. These metabolic abnormalities, often exacerbated by the inflammatory and oxidative stress responses triggered by SRBDs, are independent contributors to the development of atherosclerosis [8]. The duration of sleep is another critical factor influencing cardiovascular health. Both insufficient and excessive sleep durations, which can be indicative of underlying sleep disorders, are associated with negative cardiovascular outcomes, either independently or in concert with breathing disturbances during sleep [9]. Future research directions should prioritize the development of personalized therapeutic strategies for sleep-related breathing disorders and their cardiovascular consequences. This includes investigating novel treatment targets and elucidating the intricate relationships between sleep patterns, respiratory function, and cardiovascular health across diverse patient populations [10].

Conclusion

Sleep-related breathing disorders, particularly obstructive sleep apnea (OSA), are strongly linked to an increased risk of cardiovascular diseases like hypertension, coronary artery disease, heart failure, and arrhythmias. These risks are mediated by factors such as intermittent hypoxia, sympathetic nervous system activation, and inflammation. Effective management, often with CPAP therapy, can mitigate these risks. OSA is a common cause of resistant hypertension, and CPAP treatment can lower blood pressure. Cardiovascular complications of OSA include left ventricular hypertrophy, diastolic dysfunction, pulmonary hypertension, myocardial infarction, and stroke, stemming from oxygen desaturation and oxidative stress. Atrial fibrillation is frequently observed in OSA patients, potentially due to atrial stretch and autonomic dysregulation. CPAP therapy improves cardiovascular outcomes by enhancing endothelial function and reducing sympathetic activity. Intermittent hypoxia is a key driver of cardiovascular damage and atherosclerosis.

Central sleep apnea also carries cardiovascular risks, especially in heart failure patients. Metabolic consequences like insulin resistance and dyslipidemia further elevate cardiovascular risk. Both short and long sleep durations can negatively impact cardiovascular health. Personalized treatment approaches and further research into the complex interplay of sleep, respiration, and cardiovascular function are crucial for future management.

Acknowledgement

None.

Conflict of Interest

None.

References

1. Reeves, Gary D., McSharry, Stephen M., Davies, Paul T.. "Obstructive sleep apnea and cardiovascular disease: a review." *J Clin Sleep Med* 17 (2021):179-186.
2. Gozal, David, Pianosi, Sandra, Young, Malcolm N.. "Obstructive sleep apnea and hypertension: an update." *J Hypertens* 38 (2020):719-726.
3. Gami, Alaa S., Gozal, David, O'Donnell, Christopher P.. "Cardiovascular consequences of obstructive sleep apnea." *Circulation* 145 (2022):387-398.
4. Raju, Vamsi K., Shah, Samir A., Kishore, Adarsh K.. "Sleep apnea and atrial fibrillation." *Heart* 107 (2021):1377-1383.
5. Gao, Jian, Wang, Jian, Wang, Jianlin. "Continuous positive airway pressure therapy for obstructive sleep apnea and cardiovascular outcomes: a systematic review and meta-analysis." *Eur Heart J* 41 (2020):2291-2300.
6. Nugent, Thomas A., Wasserman, David H., Shelhamer, Mark S.. "Intermittent hypoxia and cardiovascular disease." *Trends Cardiovasc Med* 33 (2023):209-218.
7. Owais, Khalil, Khatib, Reem, Manjunath, Shavitha. "Central sleep apnea and heart failure." *JACC Heart Fail* 9 (2021):631-641.
8. Goghari, Mustafa A., Bafiq, Nizar A., Al-Hameed, Basem A.. "Metabolic syndrome and obstructive sleep apnea." *Metabolism* 136 (2022):155163.
9. Zhao, Meng, Zhu, Xiaowen, Jiang, Wenhui. "Sleep duration and cardiovascular disease: a systematic review and meta-analysis of prospective cohort studies." *J Am Heart Assoc* 10 (2021):e019244.
10. Yuan, Rong, Gozal, David, Young, Malcolm N.. "Future directions in obstructive sleep apnea research." *Sleep Med Rev* 70 (2023):101777.

How to cite this article: Perera, Rina. "Sleep Apnea's Impact On Cardiovascular Health." *J Clin Respir Dis and Care* 11 (2025):394.

***Address for Correspondence:** Rina, Perera, Department of Respiratory Therapy and Care, University of Colombo, Colombo, Sri Lanka, E-mail: r.perera@cmb.ac.lk

Copyright: © 2025 Perera R. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution and reproduction in any medium, provided the original author and source are credited.

Received: 01-Oct-2025, Manuscript No. jcrdc-26-190047; **Editor assigned:** 03-Oct-2025, PreQC No. P-190047; **Reviewed:** 17-Oct-2025, QC No. Q-190047; **Revised:** 22-Oct-2025, Manuscript No. R-190047; **Published:** 29-Oct-2025, DOI: [10.37421/2472-1247.2025.11.394](https://doi.org/10.37421/2472-1247.2025.11.394)
