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A Perspective on Sleep Disordered Breathing in Cardiovascular Diseases

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Introduction

Patients with atrial fibrillation, heart failure, and hypertension are more likely to experience sleep disordered breathing (SDB), which is linked to an increased risk of mortality, cardiovascular (CV) disease, and arrhythmia. The apnea hypopnea index discovered during one or two unattended overnight sleep tests is the primary basis for the current assessment of the severity of SDB. As more evidence has emerged linking sleep irregularities to symptoms, CV outcomes, and improvements in sensor technology, scoring systems for the complex phenomenon of SDB have undergone significant revisions in recent years. These developments have improved inter-scorer agreement [1].

Cardiovascular responses to central respiratory events and reactions to obstructive respiratory events can vary greatly. While central apneas are caused by central dysregulation of respiratory control and are characterised by periodic episodes of hyper- and hypoventilation resulting in transient changes in tidal volume and CO_2, obstructive respiratory events are primarily brought on by mechanical obstructions of the upper airway during sleep [2]. Dysregulation of respiratory control may result from increased sensitivity of peripheral and central chemoreceptors, lung congestion, and longer circulation times and event lengths.

Description

The cardiovascular system has been shown to be harmed by sleep disordered breathing, which is characterised by numerous intermittent bouts of desaturation followed by reoxygenation. The cyclical changes of hypoxia with reoxygenation in sleep apnea are comparable to ischemia-reperfusion injury and contribute to increased production of reactive oxygen species and oxidative stress, vascular inflammation, and blood pressure elevation; all of which may be involved in myocardial dysfunction.

Chronic continuous hypoxia can promote adaptive and maladaptive remodelling by differential modulation of the transcription factors hypoxia-inducible factor 1 and 2. While mechanical blockage of the upper airways during sleep is primarily, though not always, the source of obstructive respiratory events, periodic episodes of hyper- and hypoventilation is the primary cause of central apneas. The dysregulation of respiratory control and longer-lasting events are both related to increased sensitivity of peripheral and central chemoreceptors, pulmonary congestion, and prolonged circulation times.

A healthy childhood depends on sleep, and sleep-disordered breathing, which is the disruption of normal breathing rhythms and ventilation while sleeping, is linked to a number of behavioural and physical health problems. The full impact of SDB on health is still underappreciated by many clinicians, despite the fact that there is now compelling evidence that chronic, untreated obstructive sleep apnea (OSA) can cause hypertension, cardiovascular disease, metabolic disorders, obesity, and neuropsychiatric and developmental problems [3].

Sleep disordered breathing severity in individuals with cardiovascular illness, especially in those who have heart failure. Instead of serving as a risk factor for heart failure, a high percentage of central respiratory episodes identified in a single sleep study may reflect the underlying cardiac disease and mirror the degree of the temporal dynamics of pulmonary congestion. As a pathophysiological biomarker, dynamics in SDB burden as shown by short- and long-term variations may be helpful in guiding CV treatment regimens and launching timely intervention in the management of heart failure as a chronic disease [4].

Oxidative stress, systemic inflammation, and sympathetic nervous system stimulation can all contribute to endothelial dysfunction. SDB has an impact on these variables, including intermittent hypoxia, sleep deprivation, and arousals. It's possible that occasional hypoxia in OSA activates inflammatory pathways that aid in the emergence and development of atherosclerosis.

According to reports, endothelial dysfunction, as measured by flow-mediated dilatation, or arterial stiffness, as determined by the cardio-ankle vascular index, are related to the severity of SDB. SDB-associated oxidative stress and systemic inflammation support increased intima-media thickness in the cerebral artery. SDB-related hypoxia and intima-media thickness are correlated with markers of oxidative stress and inflammation [5].

Conclusion

According to evidence, those with precapillary pulmonary hypertension are more likely to experience sleep disorders, including sleep apnea and hypoxia. The incidence of new calcification was examined in accordance with baseline sleep metrics, and coronary artery calcification over time was objectively measured using computed tomography. However, when examining reduced sleep quality, sleep disturbed breathing, which is extremely prevalent in those with coronary artery disease, is a potentially confusing factor. The presence of sleep apnoea was not taken into account while establishing connections; therefore it is hard to say whether short sleep duration on its own causes an increase in mortality.

Conflict of Interest

The author declares that there is no conflict of interest associated with this manuscript.

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