Obstructive sleep apnea: the new cardiovascular disease

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Obstructive sleep apnea (OSA) is a respiratory disorder of sleep characterized by recurrent episodes of complete or partial upper airway obstruction and increasingly recognized as a novel cardiovascular risk factor. OSA has an estimated prevalence of 9–24% in middle-aged individuals [1,2] and is increasingly emerging as a cardiovascular risk factor [3–6]. Several etiological factors in OSA overlap with those of cardiovascular diseases creating difficulty in distinguishing the direct cardiovascular consequences of OSA from its role in exacerbating concomitant cardiovascular disease. Nevertheless, an independent role for OSA in cardiovascular morbidity and mortality is now well supported [4–6]. OSA mediates sympathetic activation, increased respiratory workload, and intermittent hypoxia in the immediate term and is implicated in the pathogenesis of hypertension, left ventricular dysfunction, cardiac arrhythmia, coronary artery disease and stroke. Endothelial dysfunction, oxidative stress, and inflammation are directly linked to intermittent hypoxia and critical pathways in the pathogenesis of cardiovascular disease. In otherwise healthy individuals, OSA constitutes a significant risk factor for the development of cardiovascular disease or the progression of existent cardiovascular disorders toward heart failure, stroke, or death.

In summary, OSA is strongly associated with the incidence and poor outcome of hypertension, CAD, arrhythmia, heart failure, and stroke. In addition, obesity and aging, both on the rise in the general population, are risk factors for both OSA and heart failure. Treatment of OSA completely reverses its cardiovascular consequences. Therefore, OSA should be approached as an important modifiable cardiovascular risk factor.

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