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Obstructive sleep apnea: implications for cardiac and vascular disease.

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CONTEXT: Obstructive sleep apnea (OSA) has been increasingly implicated in the initiation and progression of cardiovascular diseases. OBJECTIVE: To systematically review the interactions of OSA with cardiovascular pathophysiology and diseases. DATA SOURCES AND STUDY SELECTION: The MEDLINE database from January 1966 to March 2003 was searched using the Medical Subject Headings sleep, sleep apnea, obesity, hypertension, heart failure, cardiac arrhythmia, coronary artery disease, stroke, sympathetic activity, endothelium, inflammation, and continuous positive airway pressure (CPAP) to identify peer-reviewed studies of OSA. Priority was given to large prospective cohort studies and to randomized controlled trials. DATA EXTRACTION: We identified 154 original investigations and reviews of sleep-related breathing disorders. Data from these studies were examined for relevance and extracted by one of the authors. DATA SYNTHESIS: Approximately 1 in 5 adults has at least mild OSA (apnea-hypopnea index [ie, the number of apneic and hypopneic events per hour], 5-15), and 1 in 15 adults has at least moderate OSA (apnea-hypopnea index, 15-30). Repetitive apneic events disrupt the normal physiologic interactions between sleep and the cardiovascular system. Such sleep fragmentation, as well as abnormalities evident in patients with OSA (eg, increased sympathetic activation, vascular endothelial dysfunction, increased oxidative stress, inflammation, increased platelet aggregability, metabolic dysregulation), may be implicated in the initiation and progression of cardiac and vascular disease. Persuasive data implicate OSA in the development of hypertension, and OSA also may contribute to cardiac ischemia, congestive heart failure, cardiac arrhythmias, and perhaps also to cerebrovascular disease and stroke. CONCLUSIONS: Obstructive sleep apnea is common, readily diagnosed, and usually treatable. It frequently coexists undiagnosed in patients with cardiovascular disease, activates disease mechanisms known to elicit cardiac and vascular damage, and may be implicated in progression of cardiovascular disease and resistance to conventional therapeutic strategies. In the absence of definitive evidence from large-scale trials and a better understanding of potential cost-effectiveness, the likely benefits of diagnosis and treatment of OSA are presently best appraised on an individualized patient basis.

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