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Obstructive sleep apnea and hypertension: epidemiology, mechanisms and treatment effects.

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Obstructive sleep apnea (OSA) is a highly prevalent disorder of breathing during sleep. A growing body of evidence suggests that OSA is independently associated with an increased risk of cardiovascular disease, although the extent of this risk and underlying mechanisms remain to be elucidated. However, there is clearer evidence from epidemiological and pathophysiological research of a causal link between OSA and hypertension. The acute hemodynamic and autonomic perturbations that accompany obstructive apneas during sleep, with associated repeated arousals and intermittent hypoxemia, appear to result in sustained hypertension. In addition to the metabolic and humoral effects from obesity, OSA appears to predispose individuals to autonomic imbalance characterized by sympathetic overactivity and altered baroreflex mechanisms as well as alterations to vascular function. Treatment of OSA restores normal sleep architecture and generally mitigates the acute hemodynamic effects of OSA. Treatment of symptomatic OSA, particular at the severe end of the spectrum, appears to be associated with improvements in blood pressure, both during sleep and wakefulness, and there may also be additional gains in subjects who are hypertensive and/or resistant to antihypertensive medications. The severe group appears to be particularly at risk for developing fatal and non-fatal cardiovascular events and treatment with continuous positive airway pressure appears to markedly reduce that risk. Future treatment studies will need to be extended for greater than the current average of 1-2 months in order to more fully evaluate any time dependent improvements in blood pressure, and consequent cardiovascular risk.

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