Sleep Apnea and Stoke

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Prospective observational cohort studies demonstrate that sleep apnea is independently associated with an increased risk of stoke. These independent associations imply that there are mechanisms, specific to sleep apnea, that confer this risk. Such mechanisms may include intermittent hypoxic vascular inflammation, sympathetic hyperactivity, alterations in hemodynamics, metabolic dysfunction, and endothelial dysfunction. In the short-term, positive airway pressure (PAP) safely and effectively treats sleep apnea, and attenuates these aberrant physiologic sequelae. Increased CPAP compliance, (i.e. longer duration of effective CPAP use) appears to be directly related to these short-term improvements. Long-term observational cohort studies examining cardiovascular outcomes in the context of CPAP compliance suggest that compliance with CPAP therapy may attenuate cerebrovascular risk, but these studies could be confounded by a variety factors (e.g., CPAP adherence may be a surrogate for adherence with recommended medical interventions, leading healthier lifestyles, etc.) which precludes definitive conclusions. To date, no long-term randomized trials have demonstrated reduction in cardiovascular risk associated with CPAP therapy, and, important ethical/safety considerations have been raised regarding the conduct of such trials (whereby patients with known symptomatic sleep apnea would be randomized to a control group over the longer term that does not receive therapeutic CPAP). The high prevalence of sleep apnea in the general population confers a high population attributable risk for these adverse cardiovascular health outcomes, and whether CPAP may reduce these outcomes has enormous public health implications both for reducing a leading cause of mortality in the and possible initiation of larger scale screening. A central question persists, in the context of an existing patient's cerebrovascular risk profile and associated treatment: Does diagnosing and treating sleep apnea over the long- term confer cerebrovascular benefit? Target populations at high risk for sleep apnea and high risk for cerebrovascular outcome events are well suited for such randomized trials evaluating diagnosis and treatment interventions.

The objectives of this talk are to:
1. Describe the relation between sleep apnea and cerebrovascular disease in populations and in clinic cohorts;
2. Review the variety of potential mechanisms underlying the association of sleep apnea and cardiovascular disease;
3. Examine what is known from randomized clinical trials about the cerebrovascular benefits of sleep apnea treatment.