Autonomic Circulatory Regulation in Obstructive Sleep Apnea

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Normal sleep is characterized by a structured neural circulatory response to different sleep stages with reduced muscle sympathetic nerve activity (MSNA) and slower heart rate during slow wave sleep. During REM sleep, when dreams are more likely to occur, there are intermittent surges in sympathetic activity and marked fluctuations in heart rate.

This sleep stage-related autonomic response is disrupted in obstructive sleep apnea (OSA), where chemoreflex-mediated responses to hypoxemia and hypercapnia dominate the neural circulatory profile. Sympathetic activity increases progressively and markedly during apneic events with striking surges in blood pressure at the end of apnea, which can sometimes result in an overall increased blood pressure during sleep compared to wakefulness. This high level of sympathetic drive carries over into daytime wakefulness so that otherwise healthy patients with OSA have very high levels of daytime MSNA even during normoxic daytime wakefulness. These patients also exhibit faster heart rates, diminished heart rate variability, and increased blood pressure variability, all of which may predispose to increased risk for hypertension.

Because of the diving reflex, OSA patients may sometimes manifest increases in cardiac vagal activity, simultaneous with the increases in sympathetic traffic to blood vessels. The heightened vagal drive may induce bradyarrhythmias ranging from first-degree heart block to asystole. Simultaneous sympathetic and vagal activation together with hypoxemia, increases in afterload and other stresses may elicit atrial fibrillation, a common co-morbidity of OSA. Some data suggest that selective ablation of cardiac ganglia may prevent the development of apnea-induced atrial fibrillation. Autonomic responses to OSA may also be implicated in ventricular arrhythmias, acute myocardial infarction and sudden death occurring during sleep.