COMPUMEDICS LIMITED MARKETING

Clinics in Chest Medicine 1998;19(1):99-113.

Sleep apnea in congestive heart failure.

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Sleep-related breathing disorders, including obstructive sleep apnea (OSA) and Cheyne-Stokes respiration with central sleep apnea (CSR-CSA), commonly occur in patients with congestive heart failure (CHF). In this setting they can have adverse pathophysiologic effects on the cardiovascular system. OSA may lead to development or progression of left ventricular (LV) dysfunction by increasing LV afterload through the combined effects of elevations in systemic blood pressure and a generation of exaggerated negative intrathoracic pressure, and by activating the sympathetic nervous system through the influence of hypoxia and arousals from sleep. Abolition of OSA by continuous positive airway pressure (CPAP) can improve cardiac function in patients with CHF. In contrast to OSA, CSR-CSA is likely a consequence rather than a cause of CHF. Here, pulmonary congestion causes hyperventilation by stimulating pulmonary irritant receptors. This leads to reductions in PaCO2 below the apneic threshold during sleep, precipitating posthyperventilatory central apneas. CSR-CSA is associated with increased mortality in CHF, probably because of sympathetic nervous system activation caused by recurrent apnea-induced hypoxia and arousals from sleep. Treatment of CSR-CSA by supplemental O2, theophylline, and CPAP can alleviate central apneas. Of these treatments, however, only CPAP has been shown to improve cardiac function and symptoms of heart failure. We conclude that effective treatments of OSA and CSR-CSA may prove to be useful adjuncts to the standard pharmacologic therapy of patients with CHF.