

Prevalence and Clinical Impact of Opioid-Induced Central Sleep Apnea in Chronic Pain Patients

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DESCRIPTION

Central sleep apnea is a relatively uncommon but clinically significant form of sleep-disordered breathing characterized by repeated episodes of reduced or absent respiratory effort during sleep. Unlike obstructive sleep apnea, where the airway is physically blocked despite respiratory effort, central sleep apnea originates from a failure of the brain's respiratory control centers to send appropriate signals to the respiratory muscles. This distinct pathophysiology makes CSA not only more complex to understand but also more challenging to diagnose and manage effectively. Despite being less prevalent than obstructive forms of apnea, CSA is associated with significant morbidity, especially in individuals with underlying cardiovascular or neurological conditions.

One of the most recognized forms of CSA is Cheyne-Stokes respiration, often observed in patients with congestive heart failure or stroke. This breathing pattern is characterized by a crescendo-decrescendo pattern of tidal volume followed by central apneas or hypopneas. The pathophysiology of Cheyne-Stokes respiration is thought to involve prolonged circulation time, heightened chemosensitivity, and reduced buffering capacity of carbon dioxide, leading to respiratory overshoot and undershoot. This form of CSA is particularly concerning because it has been associated with increased mortality and worsened cardiac outcomes in patients with heart failure.

The clinical presentation of CSA can be subtle and overlaps significantly with other sleep disorders, particularly obstructive sleep apnea. Common symptoms include fragmented sleep, frequent nocturnal awakenings, excessive daytime sleepiness, fatigue, and poor concentration. Some patients may also report insomnia, palpitations, or morning headaches. Unlike obstructive sleep apnea, loud snoring is less common in CSA, and bed partners may report periodic breathing patterns rather than obstructive-type apneas. Because the symptoms are nonspecific, and because patients may not be aware of their nighttime breathing patterns, CSA is often underrecognized or misdiagnosed.

Diagnosis of central sleep apnea requires objective sleep testing, most commonly in the form of overnight polysomnography. This comprehensive test records multiple physiological parameters including airflow, respiratory effort, oxygen saturation, and brain activity. In CSA, polysomnography typically reveals episodes of absent respiratory effort, differentiating it from obstructive apneas where respiratory effort continues against a closed airway. The Apnea-Hypopnea Index (AHI) is used to quantify the severity of sleep apnea, and an AHI greater than five events per hour with predominantly central events supports a diagnosis of CSA. It is important to distinguish between different types of CSA, as this has implications for treatment and prognosis.

Treatment of central sleep apnea is complex and must be tailored to the underlying cause. In cases of heart failureassociated CSA, optimizing heart function through guidelinedirected medical therapy is paramount. This may include the use of beta-blockers, ACE inhibitors, diuretics, and in some cases, device-based therapy such as cardiac resynchronization therapy. Improvement in cardiac function can lead to resolution or significant improvement in CSA symptoms. In opioid-induced CSA, reducing or discontinuing opioid use is the primary approach. For high-altitude periodic breathing, acclimatization or descent to lower altitudes is usually sufficient, though acetazolamide can be used prophylactically in some cases.

One of the traditional treatments for CSA has been the use of continuous positive airway pressure (CPAP). While CPAP is effective in treating obstructive sleep apnea, its efficacy in CSA is variable and condition-dependent. CPAP may help stabilize the upper airway and reduce arousals, particularly in patients with coexisting obstructive and central events. However, in some individuals with CSA, especially those with Cheyne-Stokes respiration, CPAP can paradoxically worsen the condition by promoting hyperventilation and hypocapnia. Adaptive Servo-Ventilation (ASV) has emerged as a more sophisticated alternative, providing variable pressure support in response to the patient's breathing pattern. ASV has shown efficacy in stabilizing ventilation and improving sleep quality in many patients with CSA.

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