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1.0 INTRODUCTION

The central sleep apnea syndromes (CSAS) are characterized by sleep disordered breathing associated with diminished or absent respiratory effort, coupled with the presence of symptoms including excessive daytime sleepiness, frequent nocturnal awakenings, or both. However, no recent evidence-based guidelines have been published. The purpose of this practice parameter is to review the available data for the treatment and management of CSAS in adults. When possible, a relative determination was made as to the most effective treatment option.

2.0 BACKGROUND

The International Classification of Sleep Disorders (ICSD)—²² identifies 6 different forms of CSAS: (1) Primary Central Sleep Apnea, (2) Central Sleep Apnea Due to Cheyne Stokes Breathing Pattern, (3) Central Sleep Apnea Due to Medical Condition Not Cheyne Stokes, (4) Central Sleep Apnea Due to High-Altitude Periodic Breathing, (5) Central Sleep Apnea Due to Drug or Substance, and (6) Primary Sleep Apnea of Infancy. The final category will not be reviewed in this document, as these guidelines pertain to CSAS treatment in adults.

While the ICSD-2 classification system for CSAS will be used to systemize these practice parameters, it is important to recognize that the underlying pathophysiology of central sleep apnea is due to 1 of 2 mechanisms: hyperventilation or hypoventilation. Post-hypocapnia hyperventilation is the underlying pathophysiological mechanism for central apnea associated with congestive heart failure, high altitude sickness, and primary CSAS. These patients chronically hyperventilate in association with hypocapnia during wake and sleep and demonstrate increased chemoresponsiveness and sleep state instability. CSAS in the absence of an identifiable etiology is referred to as “primary CSAS.” The presence and prevalence of this entity is uncertain.

Central sleep apnea due to hypoventilation results from the removal of the wakefulness stimulus to breathe in patients with compromised neuromuscular ventilatory control. Chronic ven-

tilatory failure due to neuromuscular disease or chest wall disease may manifest with central apneas or hypopneas, at sleep onset or during phasic REM sleep. This is typically noted in patients with central nervous system disease (e.g., encephalitis), neuromuscular disease, or severe abnormalities in pulmonary mechanics (e.g., kyphoscoliosis³). The ventilatory motor output is markedly reduced and insufficient to preserve alveolar ventilation resulting in hypopneas. Thus, this type of central apnea may not necessarily meet the strict “central apnea” definition.

CSAS due to Cheyne Stokes breathing pattern (CSBP) or Cheyne-Stokes respiration (CSR) is characterized by an absence of air flow and respiratory effort followed by hyperventilation in a crescendo-decrescendo pattern. CSR most often occurs in patients with congestive heart failure (CHF). The prevalence is estimated to be approximately 30%⁴ to 40%⁵ in patients with CHF. However, this respiratory pattern can also be seen in patients with stroke or renal failure.

There is mounting evidence that CSAS/CSR may be an indicator of higher morbidity and mortality in CHF patients. Consequently, effective treatment of CSAS/CSR might improve the outcome of CHF patients with CSAS/CSR.

CSAS can occur in individuals with cardiac, renal, and neurological disorders but without a CSR pattern. This category is referred to CSAS Due to Medical Condition Not Cheyne Stokes.

CSAS associated with high altitude can be seen during the acclimatization period, during or after rapid ascent to high altitudes, typically 4000 meters or greater. Hyperventilation secondary to altitude-associated hypoxia is thought to be the trigger for high-altitude periodic breathing. Hence, individuals with a heightened or brisk response to hypoxia are more likely to develop CSAS Due to High-Altitude Periodic Breathing.²

The use of chronic opioid treatment for the management of chronic pain has increased over the last 10 years.⁶ Central Sleep Apnea Due to Drug or Substance is primarily a disorder related to opioid use. Patients who are on long-acting opioids for at least 2 months appear to be at increased risk for developing CSAS. In