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POSITIONAL CENTRAL SLEEP APNEA

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Introduction: Obstructive sleep apnea (OSA) is well known to often improve with non-supine positioning as opposed to supine positioning. The prevalence of OSA that may improve on proper positioning is 50-60% and the prevalence of OSA that appears when supine and disappears when non-supine is 25-30%. Sleeping in a lateral positioning is thought to reduce pressure on the airway, shift the directional effect of gravity on airway structures, and counteract physiologic genioglossus collapse that occurs when supine. On the other hand, the effect of positional changes on the severity of central sleep apnea (CSA) is not well documented aside from Cheyne-stokes breathing in congestive heart failure.
Report of Cases: We present two cases of positional CSA. One patient is a 52-year-old male with a history of traumatic brain injury, hypogonadism, hypothyroidism and Parkinson's Disease. He underwent split-polysomnography (PSG) for dream enactment behavior, was found to have severe CSA, which occurred almost exclusively supine (Supine Apnea-Hypopnea Index (AHI) 66/hr, Non-Supine 0.8/hr). A positional belt was recommended to the patient. The second patient is an 89-year-old male with history of chronic obstructive pulmonary disease and chronic kidney disease who underwent PSG for symptoms of sleep apnea. He was found to have severe obstructive and central sleep apnea with periods of Cheyne-stokes breathing. This also occurred almost exclusively supine (Supine AHI 69/hr, Non-Supine 0/hr) and improved on continuous positive airway pressure (CPAP) independent of position (AHI 6/hr). CPAP or positional belt was recommended to the patient.
Conclusion: Positional CSA unrelated to congestive heart failure is an uncommon phenomenon with poorly understood pathophysiology. Treatment of CSA is often challenging and based on elucidating and addressing the underlying cause such as optimizing treatment of heart failure. Positional therapy is characteristically thought of as a potential treatment option for OSA. However, our findings further support the presence of a phenotype of central sleep apnea that may respond to positional therapy.
Support (If Any):

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WORKUP OF NOCTURNAL HYPOXEMIA LEADS TO DIAGNOSIS OF PATENT FORAMEN OVALE

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Introduction: Patent foramen ovale (PFO) in adults often remains asymptomatic until clinical manifestations such as cryptogenic stroke, migraine headache, air embolism, hypoxemia, or platypnea-orthodeoxia syndrome occur. In this case, prior workup of cryptogenic stroke failed to identify a PFO that was diagnosed after further investigation of polycythemia which revealed nocturnal hypoxemia on polysomnogram.
Report of Cases: A 60-year-old male with history of recurrent venous thromboembolic events (VTE), secondary polycythemia, and cryptogenic strokes was referred for a polysomnogram during the evaluation of polycythemia. Over the span of six years, he had multiple cryptogenic strokes and VTEs in the setting of polycythemia and normal hypercoagulability labs. Further evaluation suggested secondary polycythemia after serum erythropoietin and JAK2 mutation testing were negative. The patient was a never smoker without evidence of malignancy or renal disease leading to suspicion that his polycythemia was due to hypoxemia despite normal oxygen saturations during point of care evaluations. He frequently reported shortness of breath during appointments. During a polysomnogram, the patient was found to have a total apnea-hypopnea index of fewer than 5 events per hour but demonstrated hypoxemia with 49.7% of total sleep time spent below an oxygen saturation of 90%. He was referred to pulmonology for further evaluation that showed normal resting oxygen saturation, no desaturation on a six-minute walk test, mild restrictive defect on pulmonary function testing, and a normal chest computed tomography with angiography. Prior transthoracic echocardiogram had demonstrated an atrial septal aneurysm without communication between the atrial chambers. No evidence suggestive of a right-to-left shunt was found on lateral imaging of the brain during a ventilation-perfusion scan. Further evaluation of the aneurysmal atrial septum on transesophageal echocardiogram with an agitated saline bubby study demonstrated a patent foramen ovale with a right-to-left shunt. In the setting of hypoxia leading to secondary polycythemia, the patient was scheduled for PFO closure.
Conclusion: Hypoxemia out of proportion to sleep-disordered breathing on polysomnogram should prompt further evaluation. Despite multiple prior strokes, our patient's PFO had gone undiagnosed until polycythemia prompted a polysomnogram that demonstrated isolated nocturnal hypoxemia and prompted a further workup. Clinically significant hypoxemia is an indication for PFO closure.
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A PEDIATRIC CASE OF MYOCLONIC SEIZURES PRECIPITATING CENTRAL SLEEP APNEA AND SEQUENCES OF PERIODIC BREATHING DURING SLEEP

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Introduction: Myoclonic seizures are known to be influenced by the sleep-wake cycle. Although myoclonic seizures have been shown to alter sleep architecture, little is known about their influence on sleep-disordered breathing.
Report of Cases: A 7-year-old male with a history of generalized epilepsy of unknown etiology with absence as well as myoclonic seizures, poor impulse control, and fine motor delay was referred for restless sleep and frequent nocturnal awakenings. A routine electroencephalography (EEG) performed 33 months prior to presentation, at an outside institution, reportedly showed 3.0 Hz generalized frontally dominant spike and wave discharges lasting less than two seconds. Video EEG (vEEG) revealed diffuse bilateral poly-spike and slow wave discharges, predominantly during sleep, and myoclonic seizures with abrupt whole-body jerks. Polysomnography, at this time, was characterized by mild central sleep apnea (AHI: 3.7; OAHI: 0.0; CAI: 2.8; REM AHI: