Introduction and Objective

Obstructive Sleep Apnea (OSA) is a chronic sleeping disorder with serious health consequences. Currently, OSA is the most common sleep-related breathing disorder showing an estimated prevalence of 20–30% in males and 10–15% in females in North America, with rates increasing with the rising obesity rate in the United States [1,2]. The most important risk factors for developing OSA are older age, with a plateau appearing in the 6th–7th decade, male gender, and obesity [3,4,5]. Other important risk factors include nasal congestion, smoking, menopause, and a family history of snoring or OSA [6,7,8].

OSA is characterized by recurrent, cyclical collapse of the velopharyngeal and/or oropharyngeal airway during sleep. This collapse results in obstruction of airflow and disturbances in gas exchange sufficient enough to cause arterial hypoxemia and hypercapnea. As a result of airflow obstruction and intermittent hypoxemia, patients typically experience transient arousals from sleep and interrupted sleep, leading to compensatory responses from the autonomic nervous system [9]. These compensatory mechanisms have been shown to produce serious long-term sequelae such as systemic hypertension through enhanced chemoreceptor sensitivity and excessive sympathetic vasoconstriction, dysfunctional insulin sensitivity and glucose regulation, and negative neurocognitive effects (impaired memory and concentration) due to hypoxia-induced neurotoxicity [9].

Patients classically present with a chief complaint of snoring and daytime sleepiness; however, the most specific symptom appears to be nocturnal choking or gasping [10]. This shift could be beneficial for patients wishing to undergo testing in the comfort of their homes. Furthermore, out-of-center sleep studies are financially superior as most HSAT devices are less costly and the presence of a sleep technologist is not required. Some disadvantages include data loss and misinterpretation, therefore, further research is needed in order to determine the ideal testing and diagnostic parameters of HSAT [11].

Current literature is varied in identifying recording time necessary to diagnose OSA using HSAT, reporting times ranging from 250 to over 390 minutes [12,13]. These compensatory mechanisms have been shown to produce serious long-term sequelae such as systemic hypertension through enhanced chemoreceptor sensitivity and excessive sympathetic vasoconstriction, dysfunctional insulin sensitivity and glucose regulation, and negative neurocognitive effects (impaired memory and concentration) due to hypoxia-induced neurotoxicity [9].

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Results

TRTs of at least 6 h were achieved during 199 (81%) of the HSATs and TRTs of at least 7 h were captured in 123 (54%). The diagnosis of OSA was made in 165 (67%) patients based on initial REI.

Significant differences were found between the REI at 60 min (P < 0.0001), 120 min (0.0002), 180 min (0.0001) and 240 min (0.0002) with low concordance, signifying these intervals are inadequate diagnostic correlates for the REI. REIs determined at 300, 360, and 420 min were not significantly different from the REI at and had very significant CCCs, 0.979, 0.990, and 0.996, respectively. The Fleiss’ test showed almost perfect agreement between the REI at 360 and 420 min.

Methods

A retrospective review was conducted of HSATs done from January-October 2017. Data of 246 consecutive patients with a high pre-testing index of suspicion for OSA who completed 2 consecutive nights of testing were included. The Sleep Technologists certified by the American Board of Sleep Medicine, and automated algorithm for REI analysis. Records were over scored by Registered Polysomnographic Technicians. OSA was diagnosed if the number of apnea-hypopnea events per hour of sleep (AHI) was greater than 5.

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References

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