

Mild Obstructive Sleep Apnea: Beyond the AHI

Joyce Lee-Iannotti MD

James M Parish MD

Division of Pulmonary Medicine (Dr Parish) Center for Sleep Medicine
Department of Neurology (Dr Lee-Iannotti), Center for Sleep Medicine
Mayo Clinic Arizona
Scottsdale, Arizona

A common conundrum faced by sleep medicine practitioners is how to manage the large group of patients with mild sleep apnea. Many patients are referred for sleep evaluation, with symptoms thought to be due to obstructive sleep apnea (OSA). Often polysomnography demonstrates only mild sleep apnea, and the clinician and patient are faced with the dilemma of whether to use continuous positive airway pressure (CPAP) therapy or an oral appliance. In making this important decision the clinician incorporates the commonly used definition of mild sleep apnea as an apnea-hypopnea index of between 5 and 14 apneas or hypopneas per hour of sleep. Moderate sleep apnea is defined as 15-29 events per hour, and severe is 30 and above events per hour. These arbitrary thresholds originated in the early 1980s when knowledge of this condition was in its infancy and little was known about the long term health effects. The definition was based on the finding of apneas, defined by the complete cessation of airflow for at least 10 seconds. The concept of hypopnea and respiratory-effort related arousal (RERA) came later and with frequently changing definitions that have been the subject of significant controversy throughout the last 30 years. Many sleep centers include these RERA's in the definition of respiratory disturbance index, which is incorrectly used interchangeably with AHI. While the sleep literature has demonstrated the untoward effects of moderate to severe sleep apnea, there has been considerable debate about the clinical significance of mild sleep apnea, that is, an AHI between 5 and 15.

The current paper by Quan, et al (1) is a significant contribution to the literature in sleep medicine addressing this important clinical question. This paper reports data drawn from the APPLES study, a large multi-center, well-conducted study designed to determine if CPAP therapy improves sleepiness, mood disorder, or cognitive function in patients with OSA, that has subsequently produced several important publications (2-6). As part of the study, extensive data was obtained on each of these neurocognitive parameters including the Epworth Sleepiness Scale, Stanford Sleepiness Scale, Hamilton Rating Scale for Depression, Profile of Mood States, and Sleep Apnea Quality of Life Index, all validated questionnaires used frequently in the sleep literature. In this part of the study, 199 patients with an $AHI > 5$ but ≤ 15 were compared to 40 patients enrolled in the study, but with an $AHI \leq 5$. The mean AHI was 10 per hour in the mild OSA group, and was 3 per hour in the non-OSA group. Size of the study was statistically large enough to determine significant differences. Remarkably, there

was no significant difference in any rating of sleepiness, mood, or quality of life between the two groups. This study produces an important challenge to the traditional thresholds of disease severity, and raises the question of whether mild sleep apnea based on AHI alone is a disease, and whether it truly requires treatment. Since many patients seen at sleep medicine clinics fall into this category, this is an extremely important question to address.

Several previous studies have attempted to elucidate the issue of mild sleep apnea. Barnes, et al (7) in a randomized controlled trial of CPAP in mild OSA (defined in their study as an AHI 5-30 events per hour) reported that CPAP improved self-reported symptoms of snoring, restless sleep, daytime sleepiness, and irritability, but did not improve objective measure of sleepiness (multiple sleep latency test) or any test of neurobehavioral function, quality of life, mood scores, or 24-hour blood pressure. Weaver, et al (8) reported results from the CATNAP study, a randomized, sham-CPAP controlled study of self reported sleepy patients with mild OSA (defined as AHI 5-30 events per hour) that CPAP significantly improved scores on the Functional Outcomes of Sleep Questionnaire. Both of these trials differ from the current study by defining mild OSA as an AHI up to 30 per hour, whereas the major controversy involves those patients in the AHI 5-15 range. The CATNAP study also selected patients who complained of excessive sleepiness.

The findings from this study emphasize the need to differentiate “obstructive sleep apnea” from “obstructive sleep apnea syndrome.” Obstructive sleep apnea has been traditionally defined solely by the AHI, whereas OSA syndrome incorporates the subjective and clinical components to the diagnosis (sleepiness, mood disturbance, fatigue, etc.) An abnormal AHI in the mild range without symptoms may not warrant treatment with CPAP, whereas an excessively sleepy patient with an AHI of 7 would require at least a trial of CPAP with close monitoring. Fatigue, although traditionally associated with mood disorders, is a common symptom in sleep medicine and may be a manifestation of untreated sleep apnea. Future studies could incorporate a fatigue scale (e.g. Fatigue Severity Score) as an adjunct to the Epworth sleepiness score to assess the importance of fatigue as a symptom of OSA.

The current study has an important limitation in that subjects were enrolled based on a referral to a sleep center for some clinical indication related to OSA, and therefore do not represent the general population. It would be possible that individuals drawn randomly from the general population would have lower scores on these tests than a group of subjects referred to a sleep center, which would result in the mild OSA group having significantly different scores on these tests than the general population. In addition the no-OSA group in this study included only 40 patients, and it is possible that a larger group of true no-OSA patients without symptoms causing referral to a sleep center would yield a slightly different result. However, if the untoward effects of mild OSA are indeed significant, it should be relatively easy to find significant abnormalities in mood,

sleepiness, and quality of life, and the inability to demonstrate differences in this study group leads one to conclude that the differences, if they exist, are likely to be very small.

Besides the mood and quality of life effects of sleep apnea, cardiovascular disease is known to be a significant consequence of obstructive sleep apnea (9). Stroke, heart failure, myocardial infarction, and atrial fibrillation are known to occur more commonly in untreated OSA than in normal individuals (10). There have been several studies on the cardiovascular effects of mild sleep apnea. The Sleep Heart Health study found a small but significant increase in cardiovascular disease in mild sleep apnea (11). In another study, Buchner et al (12) found CPAP reduced the risk of subsequent cardiovascular events in patients with mild to moderate (AHI 5-30 per hour) OSA. Therefore, the clinician must look at not only at the AHI, but the larger picture inclusive of presenting symptoms and cardiovascular and cerebrovascular risk factors when deciding on treatment.

Ultimately, this paper challenges the sleep community to look beyond the AHI and improve management algorithms for patients with mild obstructive sleep apnea, with or without symptoms. We propose that an obstructive sleep apnea score be developed, similar to the CHADS-2 score used to determine the need for anticoagulation in patients with non-valvular atrial fibrillation as a means of secondary stroke prevention (13). The “OSA score” could incorporate the AHI, the Epworth sleepiness scale, a quality of life score, a fatigue severity scale, and known cardiovascular and cerebrovascular co-morbidities. A point system could be generated to determine the need for CPAP or alternative therapies.

Hence, this study is likely to be a sentinel study in the sleep medicine literature. Further research in how to “score” patients who need treatment is needed in order to provide best value in management of sleep apnea.

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