POSITION PAPER

Hypopnea in Sleep-Disordered Breathing in Adults

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INTRODUCTION

ACCUMULATING EVIDENCE OVER THE PAST 15 YEARS HAS LINKED SLEEP-DISORDERED BREATHING TO EXCESS MORTALITY AND MORBIDITY. Not surprisingly, some of the consequences of sleep-disordered breathing appear to be more likely as the intensity of disordered breathing increases. The major polysomnographic measurement of the intensity of sleep-disordered breathing has been assessment of the frequency of respiratory events per hour of sleep. Although the original terminology for obstructive sleep-disordered breathing, the obstructive sleep apnea syndrome, suggested that apnea was the unique respiratory event, most clinical investigators have long recognized that apneas and hypopneas have a similar if not indistinguishable impact on expression of the disease. This position paper will review the impact of recent clinical research on the evolving definition of hypopnea in the adult patient and the role of hypopnea in assessing the intensity of sleep-disordered breathing.

Disease is treated to improve patients' quality of life, health, and life span. Secondary benefits of disease treatment may include increased individual productivity to society and arrested disease progression or complication, and decreasing overall medical expenditures. Obstructive sleep apnea is a prevalent disease, and an increasingly recognized public health issue. Recent population studies suggest that the frequency of hypopneas and apneas during sleep is associated with cardiovascular disease in a dose-dependant fashion.

Polysomnography is routinely indicated for measuring sleep stages and sleep-disordered breathing, for detection of the obstructive sleep apnea syndrome (OSA).[1] Sleep-disordered breathing, which may include apneas, hypopneas and respiratory effort related arousals (RERAs), is the hallmark of OSA. It is currently standard in clinical practice and epidemiologic studies to assess the severity of sleep-disordered breathing by combining the number of apneas and hypopneas per hour of sleep in an index called the apnea-hypopnea index (AHI) or the respiratory disturbance index (RDI). This index has been shown to be both a reproducible measurement and a predictor of associated cardiovascular disease. The severity of oxygen desaturation and sleep fragmentation during polysomnography are combined with clinical symptoms to assess the immediate consequences to the individual from the sleep-disordered breathing.

Apnea, characterized by a cessation of airflow for 10 seconds or more, is easily recognized and agreed upon. In contrast, the definition of hypopnea, characterized by a reduction without cessation in airflow or effort, is not consistent.[2] A duration criteria of at least 10 seconds is generally agreed upon in adults. More variable definition features include the degree of airflow or respiratory effort reduction, inclusion and degree of oxygen desaturation, and inclusion of arousal from sleep.[2] To compound the problem further, each of these is dependent on the method of detection. The implications of differing hypopnea definitions and methods of detection have been extensively explored.[3] The importance of outcome data to support a definition of hypopnea and methodology for detection has been stressed. RERAs, the most recently described event in the spectrum of sleep-disordered breathing, generally refers to a reduction of airflow culminating in EEG arousal but not meeting criteria for apnea or hypopnea.[4] Prevalence and disease outcome data are scarce for RERAs, and are beyond the scope of this discussion.

One approach to the definition of hypopnea would be to adopt a definition that correlates with symptoms such as excessive sleepiness. Unfortunately, sleepiness has not been shown to correlate very well with the frequency of disordered breathing events, and is frequently present in those without disease.[5] If the definition of hypopnea, which then governs treatment, cannot be formulated on symptoms alone, then improved health and life span are alternative goals. Thus, a definition based upon disease outcome, in this case primarily cardiovascular, is most reasonable at this time.

Recent population studies correlating AHI with cardiovascular disease have helped to hone the definition of hypopnea. The Sleep Heart Health Study (SHHS) is a large, multicenter trial designed to relate cardiovascular disease with polysomonographic findings. Hypopnea is defined in this study as a decrease in airflow or chest wall movement to an amplitude that was smaller than approximately 70% (i.e., a 30% or greater reduction) of baseline movement for at least 10 seconds and accompanied by oxygen desaturation of 4% or greater.[6] Tsai et al. compared different hypopnea definitions including a discernable reduction in respiratory effort combined with >4 % desaturation alone, 4% desaturation or arousal, or arousal alone.[7] The best reproducibility of definitions of hypopnea occurred when these definitions included oxygen desaturation. A high degree of interscorer reli-

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ability was shown when at least a 30% reduction in airflow or effort was combined with oxygen desaturation of at least 4%, with an intraclass correlation coefficient of 0.99.[8] Airflow and respiratory effort were measured qualitatively rather than quantitatively, methodology similar to that used in many clinical sleep laboratories and in prior outcome studies.

Findings from the SHHS reported so far have supported an association of OSA, as defined by AHI, with a variety of cardiovascular disease manifestations. Mean systolic and diastolic pressure and prevalence of hypertension was correlated with AHI even after adjusting for confounding variables such as body mass index, alcohol intake, smoking, age and ethnicity.[9] Further support for a relationship comes from studies showing effective treatment of OSA reduces daytime and nocturnal blood pressure.[10,11] Another report from the SHHS associated OSA and self-reported angina, myocardial infarction, stroke and/or congestive heart failure. Even mild elevations of AHI (≥5/hour of sleep) were associated with cardiovascular disease, an association that remained significant after adjustment for race, age, sex, smoking status, diabetes, hypertension, and cholesterol.[6] These findings confirm OSA, defined by AHI criteria, is a risk factor for a wide range of cardiovascular disease.

Evidence supports the inclusion of hypopnea along with apnea in defining OSA and its severity. Hypopnea as defined in the SHHS, a ≥30% reduction of amplitude in thorocoabdominal movement or airflow as compared to baseline with a ≥4% oxygen desaturation, has a high inter- and intrascorer reliability. It is similar to definitions used in prior studies of disease prevalence and outcome. Most importantly, a significant association between AHI, utilizing the above definition, and cardiovascular disease has been shown in a large, multicenter cohort of middle age and older adults. Support is overwhelming for the inclusion of hypopnea in the spectrum of OSA. Unfortunately, this definition will not include all patients who may benefit from treatment. But, based on current available evidence, it is an improvement over the current position that apnea alone defines OSA.

CONCLUSION

- Hypopnea in adult patients is defined as an abnormal respiratory event lasting at least 10 seconds with at least a 30% reduction in thorocoabdominal movement or airflow as compared to baseline, and with at least a 4% oxygen desaturation.
- The definition proposed has a high degree of intra- and interscoring reliability.
- An association between a wide range of cardiovascular disease and AHI, with hypopnea so defined, has been demonstrated.
- Evidence supports that hypopnea should be included with apnea in the clinical assessment of sleep-disordered breathing.
- Even mild elevations of the apnea-hypopnea index (AHI) of ≥5 are associated with increased risk of cardiovascular disease.

REFERENCES

1. Standards of Practice Committee of the American Sleep Disorders Association. Practice parameters for the indications for polysomnography and related procedures. Sleep 1997:20:406-22.
2. Moser NJ, Phillips B, Berry DTR, Harbison L. What is hypopnea anyway? Chest 1994:105:426-28.
3. Redline S, Sanders M. Hypopnea, a floating metric: implications for prevalence, morbidity estimates and case finding. Sleep 1997:20(12):1209-17.
4. Loube DI, Gay PC, Strohl KP, Pack AI, White DP, Collop NA. Indications for positive airway pressure treatment of adult obstructive sleep apnea patients. Chest 1999:115:863-66.
5. Gottlieb DJ, Whitney CW, Bonekat WH, Iber C, James GD, Lebowitz M, Nieto J, Rosenberg CE. Relation of sleepiness to respiratory disturbance index. Am J Respir Crit Care Med 1999:159:502-7.
6. Shahar E, Whitney CW, Redline S, Lee ET, Newman AB, Nieto FJ, O'Connor GT, Boland LL, Schwartz JE, Samet JM. Sleep-disordered breathing and cardiovascular disease. Am J Respir Crit Care Med 2001:163:19-25.
7. Tsai WH, Flemons W, Whitelaw WA, Remmers JE. A comparison of apnea-hypopnea indices derived from different definitions of hypopnea. Am J Respir Crit Care Med 1999:159:43-8.
8. Whitney CW, Gottlieb DJ, Redline S, Norman RG, Dodge RR, Shahar E, Surovec S, Nieto FJ. Reliability of scoring respiratory disturbance indices and sleep staging. Sleep 1998:21:749-57.
9. Nieto FJ, Young TB, Lind BK, Shahar E, Samet JM, Redline S, D'Agostine RB, Newman EB, Lebowitz MD, Pickering TG. Association of sleep-disordered breathing, sleep apnea and hypertension in a large community-based study. JAMA 2000:283:1829-36.
10. Suzuki M, Otsuka K, Guilleminault C. Long-term continuous positive airway pressure administration can normalize hypertension in obstructive sleep apnea patients. Sleep 1993:16:545-49.
11. Hoffstein V, Chan CK, Slutsky AS. Sleep apnea and systemic hypertension. Am J Med 1991:91:190-96.