

Obesity, Oxygen Desaturation, and the Need for a Phenotypic Classification of Sleep Disordered Breathing

Commentary on Ling et al. Interrelationships between body mass, oxygen desaturation, and apnea-hypopnea indices in a sleep clinic population. *SLEEP* 2012;35:89-96.

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Since the advent of reliable therapy for obstructive sleep apnea syndrome (OSAS), sleep medicine has been challenged to find cost-effective ways to identify patients who could benefit from therapy. Particularly lacking have been strategies to subdivide large numbers of patients using a stratified approach in a population-based medical management model. Instead we have spent years comparing various modalities to standard polysomnography (PSG) in an attempt to find an acceptable alternative to attended multichannel PSG with little success. This one-test-fits-all approach has resulted in underrecognition¹ of a serious and complex medical disorder.

Ling and colleagues in this issue of *SLEEP*² have provided us with a robust analysis of the potential value of oximetry as a population screening tool for OSAS. They analyzed oximetry data from attended PSG studies in 11,448 patients over nine years. In their retrospective analysis, the authors found that the oxygen desaturation index at 3% (ODI 3%) had the best performance statistically when compared with AHI. This study did not look at unattended oximetry in the home, however, which may have altered the findings and which should be evaluated in prospective studies to test their hypothesis. The ODI 3% performance did not hold true in those with BMIs less than 25 kg/m². The authors go on to propose that oximetry could be used to identify a population who, having an ODI 3% > 15 and a BMI ≥ 25, might be referred directly to therapy, thus beginning a discussion of population stratification based upon phenotypic differences and minimal screening physiologic measurements. Netzer and associates, in a review of oximetry in the past,³ compared the sensitivity and specificity of oximetry across the broad population with the conclusion that a trial of therapy could be considered in anyone with a high pre-test probability and ODI > 15 events/hour. Ling,² ten years later, has suggested that we identify a subpopulation in which oximetry alone may have sufficient specificity and sensitivity to allow for clinical intervention to proceed without further study.

The reliance upon the degree of oxygen desaturation as a criterion for the diagnosis of sleep disordered breathing remains an unsettled controversy. The paper by Ling et al. compares the ODI with the AHI scored by the “Chicago” guidelines,⁴ which utilize

3% desaturations and respiratory arousal events without desaturation to define hypopneas; this is the most sensitive scoring protocol in use. This is an interesting choice, as oximetry alone is likely to differ considerably from PSG scoring guidelines that score non-desaturation events as in this study.² The AHI derived by the Chicago method does not, however, correlate well with AHI requirements for CPAP therapy in some countries (e.g., USA), where disease definitions are tied to oxygen desaturation at the 4% level. Payers, at least in the USA, have determined that hypoxia determined by PSG (i.e., ODI) is the parameter they are most interested in. Oximetry therefore is a logical tool for identifying patients with sleep-dependent hypoxia.

The extent of hypoxia in a sleeping patient with OSA is dependent upon respiratory mechanics and arousal threshold differences in addition to the degree or frequency of upper airway obstruction. Patients with higher BMIs have more desaturations and are therefore more likely to be identified by that property. In the Ling study, women were more likely to be either normal weight or very obese, and they were underrepresented in the 25 to 35 kg/m² BMI range. These findings support the recognition that there are distinct phenotypes of patients with OSA, which explains why we have had difficulty in creating a single unifying definition for OSA. Do we believe that OSA is a disease that requires hypoxia as a necessary element, or do we include those who have sleep disruption from sleep disordered breathing without hypoxia?

As pointed out by Ling and colleagues, instead of insisting upon one testing modality for all patients, we are ready to prioritize resources and stratify populations by phenotype. Moderately overweight males would seem to be a population who could be approached by oximetry first. Other populations, such as those with normal BMIs and females who are not morbidly obese, would be best served by a study that can identify events that are physiologically important but not associated with oxygen desaturation. The PSG using the Chicago criteria⁴ would suit those populations better. Prospective studies looking at populations with phenotype-based management models and comparing a variety of diagnostic and therapeutic pathways will need to be done to determine if this approach improves care, increases access, and saves money. Only through intelligent population stratification will we be able to increase recognition and therapy with strained resources for this disease, which is a major public health problem.

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DISCLOSURE STATEMENT

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