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EDITORIAL (INVITED)

Hypopnea: 'rule of thumb' or ruler and plumb?

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Invited commentary on BaHammam AS. et al. "A comparison between the AASM 2012 and 2007 definitions for detecting Hypopnea" sleep and breathing. Feb 2014

The need to establish clinically meaningful indices of sleep disorders are driven by the necessity for equity in access to healthcare resources and clinical treatment as well as the desire to understand the relationship between pathological markers, pervasive symptoms and comorbid consequences of disease. The controversy that surrounds the definition of hypopnea springs from a contrast between symptomatology and clinical significance that may not be fully captured by current metrics required by payers for reimbursement of treatment [1, 2]. Sleep apnea clinical thresholds defining disease, treatment recommendations, and prevalence estimates are heavily influenced by hypopneas since obstructive apneas contribute only an estimated 5 to 25 % of the spectrum of sleep disordered breathing [3]. Inclusion of hypopneas in the indices of disease spectrum is intended to reflect ventilatory insufficiency by defining periods of reduced respiration. Developing more accurate measurements of inspired and expired airflow, and then providing more precise definitions for hypopneas, will be paramount for determining how mild, moderate and severe ventilation insufficiency are associated with clinically identifiable consequences such as magnitude of oxygen desaturation or duration of arousal from sleep.

The recent study by Dr. BaHamman and colleagues, examines the impact of altering the hypopnea definition on the indices of sleep breathing [4] as described for previous updates to scoring consensus [5]. In the current manuscript, the authors examine how changing the requisite percentage for reduction in airflow, and threshold level for oxygen desaturation and association with arousal, alters the distribution and significance of the clinical thresholds for disease. Agreement between the AASM 2012 [1] and 2007 (recommended and alternate) [2] scoring guidelines were assessed using Bland Altman plots that display the difference between two scores plotted versus their average. Comparing criteria of a 50 % (2007) versus 30 % (2012) reduction in airflow both with 3 % desaturation or arousal, demonstrates approximately 10 events per hour less, are detected when the threshold for reduced airflow is decreased by 20 %. In this population, approximately one fifth of the sample have solely hypopneas with a greater than 50 % reduction in airflow, while the majority of the sample is evenly distributed with individuals that have hypopneas both above and below 50 % (Fig. 1; also see the original article Figure 2-C [4]). This raises the question of whether changing percent reduction in airflow to define hypopneas obscures information important to an individuals' ventilatory capacity. On the one hand, a greater reduction in airflow may reflect a greater degree of airflow obstruction [6]; however, on the other hand may reflect increased ventilatory reserve [7] or lower arousal threshold [8].

The analysis of BaHamman et. al. demonstrates systematic bias of agreement by applying comparisons of the indices of 30 % reduction in airflow for events classified with 4 % desaturation versus 3 % or arousal. While the contrast of these criteria highlight the difference in hypopnea classification, we are unable to resolve whether the disparity is mostly due to arousal or desaturation criteria. Previous investigators using an outcome-based approach to determine the risk of cardiovascular disease from hypopneas found that events with a desaturation of at least 4 % were independently associated with disease; however, these effects disappeared when events were defined by 3 % desaturation or arousals [9]. In contrast, it is my opinion that little attention is paid to the fact that

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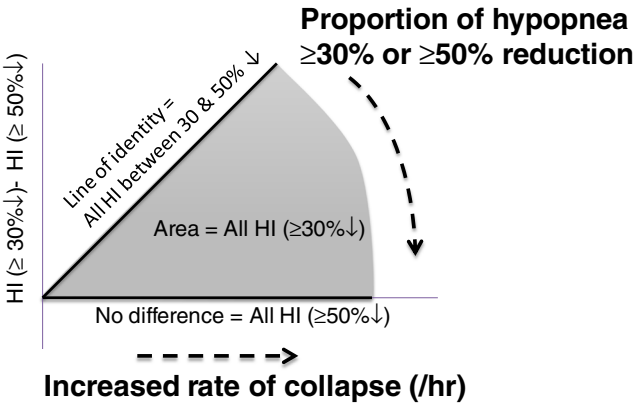

Fig. 1 A schematic summary of the Bland-Altman analysis comparing hypopnea 30 % versus 50 % reduction. The *y* axis is the difference between the hypopnea airflow reduction criteria and the *x* axis represents the average of the two hypopnea index. The line of identity (LOI) and no difference bound the 'area' of possible individual values. This scheme provides the framework examine the differences in criteria are hypopnea rate-dependent

hypopneas classified by association with 3 or 4 % desaturation may have varying baseline O_2 levels. While it is clear that less stringent criteria increases the number of patients diagnosed with sleep disordered breathing, it is also clear that the multifactorial definition of the hypopnea indices makes it difficult to define the clinical sequelae associated with ventilatory insufficiency and inadequate gas exchange.

Studies to date have predominantly utilized either naso-oral thermistor or nasal cannula-based measurements of airflow [10]. While pseudo-flow-based measurements are pervasive in the literature, empiric evidence is still required to determine the contribution of quantitative measurements such as tidal volume, inspiratory timing, and rate in contributing to sleep-related respiratory event classification [11, 12]. Moreover, in the absence of a quantitative measurement of ventilation, the hypopnea definition suffers the vagary of a reduction from the "baseline" level of breathing [13]. During sleep, the baseline breathing level is determined by various factors such as metabolic demand, airway patency, respiratory rate, and route of breathing [14]. Indeed, very little has been systematically described to document baseline breathing as it constitutes a risk factor for consequential changes. Therefore, it should stand that if methodically measured and reported, ventilatory variables such as tidal volume, rate, and respiratory timing could establish a foundation that provide addition explanatory aspects for the modulation of gas exchange and ventilatory reserve . The need to more accurately define ventilation and its component during polysomnography will help to clarify some of the variance in the spectrum of sleep disordered breathing.

It is beyond the scope of this commentary to detail all of the potential factors that influence the performance and sensitivity of the variety of nasal cannula and thermistor that are used to determine the relative percent change in airflow [13]. The most problematic appear to be: standardization in the placement of sensors, their performance relative to facial morphology and body position, detection but more importantly quantification of oral breathing, and inappropriate sampling or filtering (smoothing). Although guidelines attempt to maximize reproducibility and consistency of the technical component of airflow signal acquisition, we are still left with arbitrary indicators that lack a definite magnitude of any physical quantity.

While consensus and standardization of sleep disordered breathing definitions may lead to consistency, neither should be at the expense of accuracy. More respiratory-related detail, not less, combined with quantifiable (not relative) values are required to (1) more precisely understand the impact of sleep disordered breathing on clinical outcomes, (2) unravel the complexities of current multifactorial indices of breathing events, and (3) define the phenotypes to uncover primary etiologies of upper airway collapse, reduced ventilatory drive, increased sensitivity to CO_2, and decreased capacity for gas exchange.

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