# Measurement of the severity of sleep-disordered breathing: a moving target

Measurement of the severity of sleep disordered breathing – a moving target.
Response to: Gupta*, et al. Hypopneas With Arousals: An Important Feature of Central Nervous System Sympathetic Activation in Posttraumatic Stress Disorder (PTSD).*

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To the editor:

We thank Dr. Gupta for her comments on the importance of hypopneas with arousals causing sympathetic nervous system activation and consequent elevations in blood pressure[1]. Dr. Gupta highlights the importance of incorporating arousals in the hypopnea definition, particularly in patients with posttraumatic stress disorder (PTSD) and other psychiatric conditions that are already associated with increased sympathetic activity[1,2]. Given the high prevalence of OSA among individuals with PTSD[2], further studies are needed to evaluate whether arousal index and hypopneas resulting in arousals are indeed higher in this vulnerable population.

We also thank Drs. Javaheri and Gay for their comments on the potential adverse pathophysiologic consequences of arousals and chronic sleep fragmentation including increased sympathetic activation, inflammation, dysbiosis, and adverse structural vascular changes[3]. Although some data suggest that hypoxia may be more predictive of adverse cardiovascular events in patients with sleep disordered breathing[4], further studies in larger populations are needed to determine whether hypoxia, sympathetic activation, or other pathophysiological mechanisms in obstructive sleep apnea are the primary drivers for subsequent cardiovascular disease. The etiology is likely multifactorial and may vary among different populations such that different measures of sleep disordered breathing are more relevant. For example, in patients with heightened baseline sympathetic activation such as those with PTSD, hypopneas with arousals may be more relevant whereas in patients with heart failure, the time spent with oxygen saturation <90% may be a better predictor of subsequent morbidity.

It is possible that the current definition of apnea-hypopnea index (AHI) may still not be the optimum one. The current definitions of AHI do not incorporate duration of apneas and

hypopneas or duration of hypoxic events, which may be important in patients with comorbid cardiovascular disease (CVD) or more predictive of the development of incident CVD in certain populations. Future metrics incorporating these variables may potentially be more predictive of the adverse outcomes of OSA. It is also possible that different measures of sleep disordered breathing severity are relevant to different populations. Ultimately, more studies are needed to elucidate the potential phenotypes of sleep disordered breathing in diverse populations and to further understand the mechanisms underlying the associations between sleep disordered breathing and subsequent cardiovascular disease. However, pending new studies or metrics, we believe that universal acceptance of a hypopnea definition that includes both 3% desaturation and arousals is a step in the right direction and will expand appropriate access to much needed treatment of sleep disordered breathing.

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