

LETTERS TO THE EDITOR

Hypopneas with arousals: an important feature of central nervous system sympathetic activation in posttraumatic stress disorder

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Javaheri and Gay¹ have discussed the very important findings of Budhiraja and colleagues² from the Sleep Heart Health Study data, which showed a strong association between the apneahypopnea index (AHI) and hypertension using the American Academy of Sleep Medicine definition of hypopneas (30% reduction in airflow associated with $\geq 3\%$ oxyhemoglobin desaturation and/or an arousal) and not the current Centers for Medicare and Medicaid Services (CMS) criterion of $a \ge 4\%$ oxyhemoglobin desaturation with no consideration for an arousal component. The authors¹ propose that hypopneas with arousals alone or obstructive sleep apnea (OSA) arousal syndrome should be considered by CMS as a valid event representing disordered breathing, as arousals are associated with serious health outcomes such as hypertension.^{1,2} In one study the odds of hypertension increased approximately 20% per 5-unit increase in arousal index; the authors note that arousals are associated with sympathetic activation with consequent increased elevated blood pressure and long-term sleep fragmentation.¹ Javaheri and Gay¹ refer to the seminal work of Christian Guilleminault,³ who had first described the upper airway resistance syndrome and the importance of treating arousals alone.

It is very important to consider arousals in the definition of hypopneas in OSA in psychiatric disorders such as posttraumatic stress disorder (PTSD) that are associated with sympathetic activation. PTSD is associated with a higher prevalence of OSA⁴ and increased cardiovascular morbidity including increased risk for early incident hypertension by more than 30%.⁵ The sympathetic activation in PTSD can lead to increased cortical activation and directly affect AHI.⁶ Furthermore the inclusion of arousals in the definition of OSA also has potential implications for the management of OSA, for example, consideration of interventions to decrease cortical activation with treatments such as anticonvulsant mood stabilizers and neurofeedback. This may be especially relevant in patients with PTSD in whom the adherence to standard positive airway pressure therapy can be problematic.⁴

CITATION

Gupta MA. Hypopneas with arousals: an important feature of central nervous system sympathetic activation in posttraumatic stress disorder. *J Clin Sleep Med.* 2020;16(2):335.

REFERENCES

- Javaheri S, Gay PC. To die, to sleep- to sleep, perchance to dream... without hypertension: Dreams of the visionary Christian Guilleminault revisited. J Clin Sleep Med. 2019;15(9):1189–1190.
- Budhiraja R, Javaheri S, Parthasarathy S, Berry RA, Quan SF. The association between obstructive sleep apnea characterized by a minimum 3 percent oxygen desaturation or arousal hypopnea definition and hypertension. *J Clin Sleep Med*. 2019;15(9):1261–1270.
- Guilleminault C, Stoohs R, Clerk A, Cetel M, Maistros P. A cause of daytime sleepinesss: the upper airway resistance syndrome. *Chest.* 1993;104(3):781–787.
- Zhang Y, Weed JG, Ren R, Tang X, Zhang W. Prevalence of obstructive sleep apnea in patients with posttraumatic stress disorder and its impact on adherence to continuous positive airway pressure therapy: a meta-analysis. *Sleep Med.* 2017;36:125–132.
- Burg MM, Soufer R. Post-traumatic stress disorder and cardiovascular disease. Curr Cardiol Rep. 2016;18(10):94.
- Gupta MA. Effect of varying definitions of hypopnea on the calculation of the apnea-hypopnea index may depend upon the level of sympathetic activation: results from a patient with posttraumatic stress disorder. J Clin Sleep Med. 2019;15(10):1555.

SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication October 27, 2019 Submitted in final revised form November 6, 2019 Accepted for publication November 6, 2019

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DISCLOSURE STATEMENT

Off-label or investigational use: yes (mention of anticonvulsant mood stabilizers). The author reports no conflicts of interest.