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COMMENTARY

Does a Low Arousal Threshold Equal Low PAP Adherence?

Commentary on Zinchuk et al. Prevalence, associated clinical features, and impact on continuous positive airway pressure use of a low respiratory arousal threshold among male United States veterans with obstructive sleep apnea. *J Clin Sleep Med*. 2018;14(5):809–817.

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The propensity for respiratory derangements to disturb sleep, and the development of obstructive sleep apnea (OSA), is governed by anatomic and non-anatomic factors.[1] One of the non-anatomic factors is a low arousal threshold (ArTH). Patients with a low ArTH are more likely to be aroused from sleep by a given respiratory disturbance, which may contribute to sleep fragmentation and lead to an increase in hypopneas.[2]

Positive airway pressure (PAP) remains the primary treatment for OSA. It is 100% effective for restoring pharyngeal patency during sleep, but that is all it does. The low ArTH is not directly addressed. For those patients whose OSA is predominantly driven by a low ArTH, there is a biologically plausible explanation for poor PAP adherence. PAP may not fix their sleep-related respiratory disturbances, and conceivably, could make them worse.

Is there evidence to support a relationship between the ArTH and PAP adherence? There is now. In this issue of the *Journal of Clinical Sleep Medicine*, Zinchuk et al. studied a large cohort of veterans who were prescribed PAP for OSA.[3] They measured the ArTH, indirectly, and found it was related to ethnicity, age, medications, body mass index, and comorbid disease. They also identified an association with lower adherence among nonobese patients. Perhaps this data confirms our physiologic hypothesis: thinner patients, presumably with less contribution from upper airway collapsibility (Pcrit), do not use PAP if they have a low ArTH.

At Walter Reed National Military Medical Center (WRNMMC) we have long suspected this relationship exists. Despite a robust education program, frequent follow-up, and prescription of non-benzodiazepine sedative hypnotics (NBSHs), our adherence is well below average.[4,5] The prevalence of the low ArTH is greater than 80% in our patient population, and our patients are generally younger and thinner because we only see active duty service members.[6] Does the low ArTH drive our poor adherence, at least in part? We do not know because the prevalence is so high, we cannot generate an internal comparison group to study. Our suspicion is that it does.

Assuming the relationship between low ArTH and adherence truly represents cause and effect, what should we do about it? Sleep aids can increase the arousal threshold, but their effects are modest and unpredictable for individual patients.[7] One small study found that eszopiclone improved the apnea-hypopnea index in patients with a low ArTH identified using an epiglottic pressure monitor.[8] Our group used NBSHs to improve adherence in patients prescribed PAP, but we did not identify the prevalence of the low ArTH in that cohort.[9] In that study we included dependents and retirees, who are older and heavier than active duty service members, so we cannot assume a high prevalence of low ArTH by extrapolating data from our active duty WRNMMC patients. In short, we do not have much data to support using sleep aids in lieu of or in addition to PAP.

We believe the glass is half full though. The Zinchuk group identified the low ArTH using variables collected on routine polysomnography (PSG). They found a subgroup that is thin, has a low ArTH, and poor PAP adherence. Furthermore, they found several non-PSG factors that predict the low ArTH. These can be used to determine pretest probability for OSA, before PSG, by modeling the likelihood that non-anatomic factors are present.

The physiologic factors that drive the propensity for apneas and hypopneas, including the low ArTH, exist along a continuum. They interact in complex ways in the individual patient.[10] Using physiologic knowledge to impact diagnosis and treatment is not going to be simple and clean. That said, PAP adherence is poor, and education, follow-up, and desensitization are expensive and labor intensive.[11] The way forward is to use readily available data to identify the physiologic contributors to OSA and PAP adherence, and then use this data to individualize therapy. We are not there yet, but the study by Zinchuk et al. gets us one step closer.

CITATION

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DISCLOSURE STATEMENT

The authors report no conflicts of interest.