

COMMENTARY

Underestimation of Sleep Apnea With Home Sleep Apnea Testing Compared to In-Laboratory Sleep Testing

Commentary on Bianchi and Goparaju. Potential underestimation of sleep apnea severity by at-home kits: rescoring in-laboratory polysomnography without sleep staging. *J Clin Sleep Med*. 2017;13(4):551–555.

Arveity R. Setty, MD

Sanford Children's Hospital, West Fargo, North Dakota

Home sleep apnea testing (HSAT) has made it easier to assess sleep apnea because of its widespread availability and reduced cost compared to in-laboratory polysomnography (PSG). However, the biggest limitation of HSAT is its inability to document sleep stages, which potentially alters the value of the apnea-hypopnea index (AHI) during the sleep study. This occurs largely because HSAT measures total recording time (TRT) rather than total sleep time (TST). Because HSAT measures a patient's sleep differently than PSG, the American Academy of Sleep Medicine (AASM) prefers to use the term respiratory event index (REI) rather than AHI when patients undergo HSAT. When patients with high pretest probability undergo HSAT, it has been shown to have high sensitivity and specificity for detecting obstructive sleep apnea (OSA),¹ but it has been noted in previous studies that HSAT might underestimate overall severity of sleep apnea because of the measurement of TRT and not the TST.^{2,3} One HSAT device may be the exception—the Apnea Risk Evaluation System—because it utilizes limited frontal electroencephalographic (EEG) channels. The underestimation of sleep apnea severity might pose a significant issue for a clinician because successful management of OSA relies on the accurate measurement of the severity of the patient's sleep apnea.⁴ Furthermore, long-term complications of sleep apnea are directly correlated with the severity of sleep apnea.^{5–8}

In this issue of *Journal of Clinical Sleep Medicine*, Bianchi and Goparaju performed a retrospective analysis comparing TRT to TST in assessing the severity of sleep apnea, and how this would affect the outcome of severity of sleep apnea in a tertiary sleep center population.⁹ They analyzed a population of 833 who underwent PSG at their center irrespective of etiology for a duration of 2 years. There were 394 subjects with AHI < 5 and 444 with AHI > 5. They re-scored the AHI based on total time in bed in both groups representing TRT as would be measured by HSAT. Both the cohort groups were similar especially with respect to TST and TRT, which was the important part of this study. Reclassification was done only for the group with OSA and it resulted in a classification of one grade less severe in 26.4% of the patients. Further, it was noted that age was significantly correlated with the difference between the initial AHI and recalculated AHI.

An analysis was also performed to determine whether considering subjective TST (sTST) will solve the problems of underestimating assuming that sTST is closer to objective TST (oTST) than TRT as measured by HSAT. The results of those findings led to a reclassification of 10.5% of patients from none to any OSA. When the cohort from the OSA group was analyzed with sTST rather than oTST, at least one level higher severity was noted in 16.9% patients. This is probably because of underestimation of sleep duration by most individuals.

HSAT also cannot assess respiratory events resulting in arousals (respiratory effort-related arousals). This study also evaluated that issue and it was noted that among those who had an AHI < 5, the recalculation observed that 66.4% had a respiratory disturbance index (RDI) > 5 and 22.9% had RDI > 15.

HSAT is easy to perform, and providers should be aware that it can potentially underestimate the severity of sleep apnea. This, in turn, directly affects the management of sleep apnea. Although this study sheds light on the important differences in how PSG and HSAT measure a patient's sleep, it is by basic understanding and also by AASM clinical guidelines that we know HSAT is not the best way to assess sleep apnea severity. This is especially true for patients in whom there exists a strong suspicion of sleep apnea but HSAT has a negative result. It is then appropriate to conduct in-laboratory sleep testing for better understanding.

The study by Bianchi and Goparaju also noted that there were more periodic limb movements of sleep (PLMS) measured in the patients with in-laboratory studies. PLMS is a potential cause of insomnia or nonrefreshing sleep, and can be missed by HSAT. PLMS has also been linked to long-term complications.¹⁰

With advancements in science, it is likely that one day we will have HSAT that can provide data similar to the data that in-laboratory PSG can obtain. I concur with the authors' comment that it would be easier to add leads that pick up autonomous activity in sleep rather than adding EEG signals for sleep staging. Either way, adding more leads to current HSAT devices will provide sleep clinicians with more information. Until then, clinicians should be aware that HSAT can potentially underestimate the severity of sleep apnea and cannot rule out PLMS or parasomnia.

CITATION

Setty AR. Underestimation of sleep apnea with home sleep apnea testing compared to in-laboratory sleep testing. *J Clin Sleep Med*. 2017;13(4):531–532.

REFERENCES

1. Ward KL, McArdle N, James A, et al. A comprehensive evaluation of a two-channel portable monitor to “rule in” obstructive sleep apnea. *J Clin Sleep Med*. 2015;11(4):433–444.
2. Collop NA, Tracy SL, Kapur V, et al. Obstructive sleep apnea devices for out-of-center (OOC) testing: technology evaluation. *J Clin Sleep Med*. 2011;7(5):531–548.
3. Aurora RN, Swartz R, Punjabi NM. Misclassification of OSA severity with automated scoring of 8 home sleep recordings. *Chest*. 2015;147(3):719–727.
4. Epstein LJ, Kristo D, Strollo PJ Jr, et al. Clinical guideline for the evaluation, management and long term care of obstructive sleep apnea in adults. *J Clin Sleep Med*. 2009;5(3):263–276.
5. Walia HK, Li H, Rueschman M, et al. Association of severe obstructive sleep apnea and elevated blood pressure despite antihypertensive medication use. *J Clin Sleep Med*. 2014;10(8):835–843.
6. Lee CH, Khoo SM, Chan MY, et al. Severe obstructive sleep apnea and outcomes following myocardial infarction. *J Clin Sleep Med*. 2011;7(6):616–621.
7. Marshall NS, Wong KK, Cullen SR, Knuiman MW, Grunstein RR. Sleep apnea and 20-year follow-up for all-cause mortality, stroke, and cancer incidence and mortality in the Busselton Health 5 Study cohort. *J Clin Sleep Med*. 2014;10(4):355–362.
8. Lopez-Jimenez F, Sert Kuniyoshi FH, Gami A, Somers VK. Obstructive sleep apnea: implications 44 for cardiac and vascular disease. *Chest*. 2008;133(3):793–804.
9. Bianchi MT, Goparaju B. Potential underestimation of sleep apnea severity by at-home kits: rescoring in-laboratory polysomnography without sleep staging. *J Clin Sleep Med*. 2017;13(4):551–555.
10. Walters AS, Rye DB. Review of the relationship of restless legs syndrome and periodic limb 23 movements in sleep to hypertension, heart disease, and stroke. *Sleep*. 2009;32(5):589–597.

SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication March 9, 2017

Submitted in final revised form March 14, 2017

Accepted for publication March 14, 2017

Address correspondence to: Arveity R. Setty, MD, Sanford Health, Fargo, North Dakota; Email: arveitysetty@gmail.com

DISCLOSURE STATEMENT

Dr. Setty has indicated no financial conflicts of interest.