https://doi.org/10.5664/jcsm.9042

JCSM | Journal of Clinical Sleep Medicine

LETTERS TO THE EDITOR

Obstructive sleep apnea in coronary artery disease: the role of nocturnal hypoxic burden

Response to Azarbarzin A, White DP. Reply to "Impact of obstructive sleep apnea on left ventricular mass index in men with coronary artery disease". *J Clin Sleep Med*. 2021;17(2):357. doi:10.5664/jcsm.8968

Zhihua Huang, MD[1,2,*]; Ling Wang, MD[1,*]; Yuanhui Liu, MD[1]; Guang Li, MD[1]; Jiyan Chen, MD, PhD[1]

*[1]Department of Cardiology, Guangdong Cardiovascular Institute, Guangdong Provincial Key Laboratory of Coronary Heart Disease Prevention, Guangdong Provincial People's Hospital, Guangdong Academy of Medical Sciences, Guangzhou, China; [2]Shantou University Medical College, Shantou, China; *Contributed equally.*

The authors are grateful to Dr. Azabarzin and colleagues[1] for bringing the issue of "hypoxic burden" measurement and its related findings to our attention. Hypoxic burden, a recently described parameter by Azabarzin et al, captures the duration, the depth, and the frequency of respiratory events.[2,3] It is a better predictor of cardiovascular mortality across populations than the apnea-hypopnea index or oxygen desaturation index[2] and predicts incident heart failure.[3] Computation of this novel metric in our study[4] was mostly based on the method proposed by Azabarzin et al,[2,3] yet there were minor differences. For each individually identified apnea or hypopnea, the oxygen saturation signals (SpO_2) were traced prior to and after the end of the event until 2 SpO_2 peaks on each side were identified. In our population of coronary artery disease (CAD), SpO_2 signals exhibited unstable baseline levels and lacked a clear start and end for a given respiratory event. The average desaturation curve for each participant was determined by overlaying SpO_2 signals with respect to the end of events. This criterion yielded a search window for calculation of hypoxic burden. However, the pre-event baseline saturation in our study was defined as the maximum SpO_2 over a participant-specific search window in comparison with that during the 100 seconds prior to the end of the event proposed by Azabarzin et al.[2] This may, to some extent, underestimate the individual hypoxic burden in our population of patients with CAD. The integrated area under the baseline value was calculated over an individual-specific search window for each event. The hypoxic burden was then obtained by adding these single desaturation areas and dividing the total recording time.

Notably, another influencing factor accounting for the variation in hypoxic burden in our studies with similar sleep apnea severity is the difference of the study population. Compared with the whole population, patients with CAD may be less tolerant of hypoxemic events. Even a short respiratory event and oxygen desaturation may elicit a microarousal and another respiratory cycle in patients with CAD. The average respiratory event duration in the shortest quartile in our CAD population was 12.7 seconds vs 15.9 seconds in the Sleep Heart Health Study,[5] which supported the above assumption. Presumably, patients with CAD may have augmented autonomic nervous reactions and increased ventilatory instability, rendering short respiratory events a potential discernable biomarker of low arousal threshold. However, the aforementioned assumptions should be further examined by future studies.

Our study highlighted the importance of various sleep apnea parameters in the risk estimation for left ventricular hypertrophy in patients with CAD. Sleep apnea is a complex entity with multiple dimensions and other parameters may be part of its management plan, particularly in patients with comorbid cardiovascular disease. More in-depth prospective studies are warranted to explore whether parameters of hypoxemic sequalae or autonomic responses of sleep apnea influence target organ damage or long-term prognosis in patients with CAD.

CITATION

Huang Z, Wang L, Liu Y, Li G, Chen J. Obstructive sleep apnea in coronary artery disease: the role of nocturnal hypoxic burden. *J Clin Sleep Med*. 2021;17(2):359–360.

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SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication November 25, 2020
Submitted in final revised form November 25, 2020
Accepted for publication November 25, 2020

Address correspondence to: Guang Li, MD, and Jiyan Chen, MD, PhD, Department of Cardiology, Guangdong Cardiovascular Institute, Guangdong Provincial People's Hospital, Zhongshan Road #106, Yuexiu District, Guangzhou 510080, Guangdong, China; Email: drliguang@hotmail.com (G.L.) and gdchenjiyan@126.com (J.C.)

DISCLOSURE STATEMENT

All authors contributed substantially to the work and agreed to submit the manuscript for publication. Work for this study was performed in the Department of Cardiology, Guangdong Cardiovascular Institute, Guangdong Provincial Key Laboratory of Coronary Heart Disease Prevention, Guangdong Provincial People's Hospital, Guangdong Academy of Medical Sciences, Guangzhou, China. This study was funded by a grant from the Science and Technology Special Funding of Guangdong Provincial People's Hospital (no. 2017bq03). The funders had no role in the study design, data collection and analysis, the decision to publish, or the preparation of the manuscript. The authors report no conflicts of interest.