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LETTERS TO THE EDITOR

Cognitive profiles in obstructive sleep apnea and their relationship with intermittent hypoxemia and sleep fragmentation

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We read with great interest the article written by Olaithe et al[1] describing distinct neuropsychological performance profiles in moderate–severe obstructive sleep apnea (OSA) and exploring their relationship with OSA features. This article supports the multifactorial aspect of cognitive dysfunction in this population and characterizes protective (cognitive reserve) and detrimental (hypoxemia and comorbidities) factors.

The authors have calculated the apnea-hypopnea index based on alternative American Academy of Sleep Medicine criteria, where a hypopnea requires a 50% decrease in nasal airflow with a ≥3% oxygen desaturation. It is assumed that the apnea-hypopnea index closely reflects the oxygen desaturation index, although the information is not provided. It is puzzling that there was no association between cognitive profiles and apnea-hypopnea index. The apnea-hypopnea index (or oxygen desaturation index) would more closely reflect intermittent hypoxemia.[2] The mean oxygen saturation (SpO_2) may inform on the severity of intermittent hypoxemia. However, it may reflect a more sustained pattern of hypoxemia that can be observed with hypoventilation or chronic pulmonary diseases. The proportion of study participants who experienced such diseases or who were smokers is unknown and may have been a confounding factor. Intermittent hypoxemia has distinct downstream pathophysiologic consequences that differ from sustained chronic hypoxia, notably oxidative and proinflammatory effects.[3] Hence, assessing a wider spectrum of measures of hypoxemia, such as the oxygen desaturation index, percentage of time spent with a SpO_2 below 90% (T90), or the nadir SpO_2, may have provided additional answers to help more precisely delineate the role of OSA-related hypoxemia on cognition.

Last, the authors mention the glymphatic system as a factor implicated in cognitive changes. Glymphatic function is most active in sleep and is disrupted by sleep fragmentation. OSA-related sleep fragmentation has detrimental effects on cognitive function.[4] It would have been interesting to explore how OSA-related sleep fragmentation influenced cognitive function in their sleep clinic study population. By using scoring criteria for OSA that exclude hypopneas without oxygen desaturation but with arousal, the authors may have omitted an important element in the mechanisms of OSA-related cognitive changes.

In conclusion, the results of Olaithe and colleagues are very revealing but may require further clarification as to the mechanisms accounting for the heterogeneity of cognitive dysfunction in OSA.

CITATION

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DISCLOSURE STATEMENT

Both authors have seen and approved the manuscript. The authors report no conflict of interests.