Commentary

iMedPub Journals
www.imedpub.com

Journal of Neurology and Neuroscience
ISSN 2171-6625

2023
Vol.14 No.S6:004

Causes of Obstructive Sleep Apnea and Associated Comorbidities Involved in Cognitive Impairment of Patients

Lawrence McCloskey*

Department of Behavioral Health, Wellspan Health System, York, Pennsylvania, USA

Corresponding author: Lawrence McCloskey, Department of Behavioral Health, Wellspan Health System, York, Pennsylvania, USA, E-mail: lmccloskey@wellspan.org

Received date: 29-May-2023, Manuscript No. IPJNN-23-13786; **Editor assigned:** 31-May-2023, PreQC No. IPJNN-23-13786 (PQ); **Reviewed:** 14-Jun-2023, QC No IPJNN-23-13786; **Revised:** 21-Jun-2023, Manuscript No. IPJNN-23-13786 (R); **Published:** 29-Jun-2023, DOI: 10.4172/2171-6625.23.S6.004

Citation: McCloskey L (2023) Cognitive Impairment in the Obstructive Sleep Apnea and its Causes. J Neurol Neurosci Vol.14 No.S6:004

Description

As anyone with an interest in this subject knows, the obstructive Sleep Apnea Syndrome (OSA) is a very common condition in middle-aged and older adults which is commonly associated with serious outcomes like reduced quality of life and increased all-cause mortality. Despite some dissent, there has emerged a rough consensus after decades of research that OSA is also associated with mild cognitive impairments across a range of domains. Although different reviewers have stressed different ones, my own conclusion after perusal of this vast literature is that Attention/Working Memory (A/WM), Executive Function (EF), Episodic Memory (EM) and Speed of Information Processing (SIP) may be most vulnerable, as they are in depression and many other illnesses that are often comorbid with OSA, including obesity, coronary atherosclerosis, Hypertension (HTN) and Diabetes Mellitus (DM), in accord with what Gaquoine in called "Frontal-Subcortical Ischemia" [1-3]. Therefore, some authorities have suspected that such comorbidities may account for these impairments instead of OSA itself. Moreover, the inconsistent correlations, summing to nil in several meta-analyses, between the severity of OSA and impairment in cognition further undercuts causality [1]. Commented here on the very scant literature which addresses this important question directly.

Borges, et al. asserted truly in that EF is "a multifaceted construct" whose impairment might not "be shown when following the widely held practice" which holds widely across other cognitive domains in this literature, of measuring it by only one or a couple of tests [4]. Matched by age, gender, education, intelligence and body-mass index, 22 treatment-naïve OSA patients and 22 controls, all without obesity, psychopathology, HTN or DM, did not differ on tests of attention, working memory, planning, updating, set-shifting, and dual-tasking, inhibition, flexibility, and verbal fluency. Acknowledging their "limited sample size," the authors concluded that "no clinically significant executive effects are found in moderate and severe (OSA) patients who are otherwise healthy".

Seemingly unaware that they were partially replicating the study above, Gnoni, et al. in early "undertook a proof of the concept to define the cognitive pattern in a (rare) group of male, middle-aged patients with untreated OSA who present without comorbidities" of any kind [5]. Compared to 7 "healthy people-education-matched" controls, 11 patients with severe OSA performed often substantially worse on tests of "executive functioning, visuospatial short-term memory vigilance and psychomotor control" which were "largely in keeping with previous studies of OSA patients with associated multiple comorbidities". They also reported," for the first time, diminished social cognition". Revealing a threshold effect, patients with mild OSA "performed better than those with severe OSA on most of those same tasks and rarely worse than controls".

Unaware in turn of the contemporaneous study above, I sought also in early 2023 to approach causality from a different direction by controlling comorbidities through multiple regressions [1]. After partialing the presence or absence of HTN and DM among 39 patients with mild to severe OSA, the Apnea-Hypopnea Index (AHI; the most common measure of severity) but not the Nadir Of Oxygen Saturation (NOS; the most common measure of deoxygenation) predicted demographically corrected, composite scores of several tests apiece which tapped A/WM, EF and EM, accounting for 10-13 percent of the variance. I interpreted this dose-response relationship as evidence that OSA may cause mild deficits in these three cognitive domains beyond those attributable to at least two of its most frequent comorbidities.

These three germane studies (all that I could find) suffer by tiny samples (as must be expected in proofs of concept), so all stand in dire need of replication. Two of them sought the advantage of a theoretical test of OSA's unique cognitive effects by recruiting more or less "pure" patients at the cost of generalizability beyond the sliver of OSA patients without contributory comorbidities [4,5]. This strategy prevented the discovery of effects which might arise only in synergy with comorbidities, however, and they contradicted each other sharply besides. The third sought generalizability by accepting "all comers" at the risk of the pitfalls of statistical control (like overfitting), while covarying only two of OAS's most frequent comorbidities.

The next step to follow the latter (my own) strategy would be:

- To recruit a large, diverse sample to yield power enough to control *via* multiple regression for more comorbidities (preferably coded as continuous variables, like blood-pressure readings, rather than simply present or absent);

- To control for demographic confounds while conserving precious degrees of freedom by administering cognitive tests with demographically-corrected norms; and
- To form composite measures of several tests in each cognitive domain to cancel error and to map "multifaceted constructs" like EF. More investigators also might consider polysomnographic variables other than the traditional AHI and NOS, including markers of sleep fragmentation (like the arousal index) and sleep microstructure (like sleep spindles). Yet their relation to mentation has been inconsistent, too [6,7].

Conclusion

The particular cognitive impairments in OSA do seem to support the oft-repeated hypothesis of frontal-subcortical ischemia, inasmuch as they do occur as well in patients with documented dysfunction in those areas. Their pathophysiology in OSA remains highly speculative, however, and their geography unconfirmed. In a meta-review of morphometric and functional-connectivity studies, for example, Yeung concluded that "no brain region…was consistently reported across the meta-analyses as significantly atrophied". I wonder therefore whether the cognitive tests which are taken to be the most "frontal" or executive are merely the most sensitive to diffuse or variegated cerebral dysfunction because they tap into broad networks.

Acknowledgement

None

Conflict of interest

None

References

1. McCloskey LC (2023) Mentation tracks severity but not oxygenation in obstructive sleep apnea. Percept Mot Skills 130:1-13.
2. Lee RSC, Hermans DR, Porter MA (2012) A meta-analysis of cognitive deficits in first-episode major depressive disorder. J Affect Disord 140:113-124.
3. Gasquoine PG (2011) Cognitive impairment in common, noncentral nervous system medical conditions of adults and the elderly. J Clin Exp Neuropsychol 33:486-496.
4. Borges JG, Ginani GE, Hachul H (2013) Executive functioning in obstructive sleep apnea syndrome patients without comorbidities: Focus on the fractionation of executive functions. J Clin Exp Neuropsychol 35:1094-1107.
5. Gnoni V, Mesquita RJ, O'Regan DO (2023) Distinct cognitive changes in male patients with obstructive sleep apnea without comorbidities. Front Sleep 1-11.
6. Gu Y, Gagnon JF, Kaminska M (2023) Sleep eletroencephalography biomarkers in obstructive sleep apnea. J Sleep 1-50.
7. Yeung AWK (2019) Morphometric and functional connectivity changes in the brain of patients with obstructive sleep apnea. A meta-analysis. J Sleep Res 28: e12857.