



# Effects of Obstructive Sleep Apnea and CPAP on Cognitive Function

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## Abstract

**Purpose of Review** Obstructive sleep apnea (OSA) is characterized by repetitive episodes of complete or partial upper airway obstruction during sleep. Studies indicate that OSA is an independent risk factor for cognitive decline in older patients. The purpose of this paper is to critically review the recent literature on the cognitive effects of untreated OSA and the benefits of treatment across cognitive domains.

**Recent Findings** OSA's greatest impact appears to be on attention, vigilance, and information processing speed. Furthermore, the presence of OSA seems to have a significant impact on development and progression of mild cognitive impairment (MCI). Impact of OSA treatment, particularly with CPAP, appears to mitigate and slow the rate of cognitive decline and may reduce the risk of dementia.

**Summary** Larger properly controlled studies, of a prospective nature, are required to further elucidate the degree of treatment effect. More studies are needed on other treatments for OSA such as oral mandibular devices and hypoglossal nerve stimulation.

**Keywords** Obstructive sleep apnea · Cognition · Continuous positive airway pressure (CPAP) · Executive function · Attention · Memory

## Introduction

Obstructive sleep apnea (OSA) is the most common sleep-related breathing disorder. It is characterized by repetitive episodes of complete or partial upper airway obstruction during sleep. Global estimates of OSA using the definition of five or more obstructive respiratory events per hour in individuals aged 30–69 years suggest a worldwide estimate of 936 million [1]. Nocturnal respiratory events in OSA are associated with intermittent blood oxygen desaturations, transient sympathetic surges, and fragmented sleep due to repetitive arousals. Risk factors for OSA include obesity (BMI > 35 kg per m<sup>2</sup>), male sex, age 40–70 years, postmenopausal women not on hormonal therapy, family history of OSA, craniofacial abnormalities, and smoking. Clinically, patients present with one or more of the following symptoms: excessive daytime sleepiness,

episodes of choking or gasping when sleeping, sleep maintenance insomnia, nocturia, loud snoring, witnessed apneas, nonrestorative sleep, morning headache, and/or fatigue. Common physical exam findings are elevated body mass index (BMI), large neck greater than 17 inches in men and 16 inches in women, large waist circumference, and a crowded oropharyngeal airway (e.g., elongated uvula, macroglossia, tonsillar hypertrophy, high arched or narrow palate, retrognathia, deviated nasal septum, or nasal polyps) [2, 3]. Treatments for OSA include weight loss, positional therapy, oral appliances, positive upper airway pressure, oromaxillofacial surgery, hypoglossal nerve stimulation, and bariatric surgery [4]. Of these, continuous positive airway pressure (CPAP) is the most commonly prescribed treatment.

Animal studies, large cross-sectional and epidemiological studies, and randomized trials show that OSA leads to systemic hypertension [5–8]. OSA is associated with a 2- to 3-fold increased risk of cardiovascular and metabolic disease [4]. Other than hypertension, metabolic and cardiovascular conditions associated with OSA include obesity, type 2 diabetes, pulmonary hypertension, coronary artery disease, cardiac dysrhythmias such as atrial fibrillation, congestive heart failure, and sudden cardiac death [9, 10]. OSA is associated with an increased incidence of stroke or death from any cause, and this association is independent of other cardiovascular and

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cerebrovascular risk factors, including hypertension [11]. Furthermore, a systemic review found that the pooled prevalence of depressive and anxious symptoms in OSA patients was 35% [12]. A recent meta-analysis found that patients with Alzheimer's disease (AD) have a 5-fold increased risk of presenting with OSA compared to age-matched controls and that about 50% of AD patients experience OSA after their initial diagnosis [13].

OSA and neurocognitive impairment have been investigated in multiple studies [14]. There are a number of potential factors that could account for cognitive impairment in patients with OSA with chronic, intermittent hypoxia being the most likely explanation [15••]. Other factors which may contribute to cognitive impairment include anxiety or depression, quality of sleep, sleep fragmentation, and excessive daytime sleepiness [16, 17]. Studies indicate that OSA is an independent risk factor for cognitive decline in older patients [18••]. OSA is prevalent in 27% of patients with MCI [19]. The purpose of this paper is to critically review the recent literature on the cognitive effects of untreated OSA and the benefits of treatment across cognitive domains.

## Cognitive Function and Obstructive Sleep Apnea

Two recent reviews examined cognitive function and OSA. Caporale et al. conducted a descriptive review of studies investigating structural brain alteration and cognitive impairment in OSA [20]. Reviewing 17 studies, they found that compared to healthy controls patients, those with OSA had worse performance in attention, memory, and executive function. Furthermore, cognitive impairment was also related to OSA severity, and treatment could improve certain cognitive aspects. Zhu et al. conducted a meta-analysis of 19,940 participants from 6 cohort studies to examine the association between adults with sleep disordered breathing (SDB) and cognitive decline [18••]. They found that adults with SDB were at significantly higher risk of cognitive decline with a greater risk in females compared to males.

To characterize MCI in a sleep-clinic population, Beaudin et al. studied cognitive function in 1084 adults referred to three academic sleep centers for suspected OSA who had home sleep apnea testing or polysomnography [21••]. Patients completed sleep and medical history questionnaires, the Montreal Cognitive Assessment Test (MoCA) of global cognition, the Rey Auditory Verbal Learning Test (RAVLT) of memory, and the WAIS-IV Digit Symbol Coding (DSC) subtest of information processing speed. The main findings were the following: (a) ~48% of all patients met the validated threshold MoCA score of < 26 to indicate MCI, increasing to > 55% in patients with moderate-to-severe OSA; (b) a MoCA < 26 was predominantly observed in older males with more severe

OSA, nocturnal hypoxemia, and vascular comorbidities; (c) moderate and severe OSA were associated with > 70% higher odds of having MCI compared to patients with no OSA after adjusting for known confounders; (d) episodic memory and information processing speed were lower in OSA patients compared to age-matched normal values, and processing speed decreased with increasing OSA severity; and (e) total MoCA and DSC scores were associated with more severe OSA and nocturnal hypoxemia. A study limitation was the lack of polysomnography (PSG) data for all patients.

Executive functioning has also been evaluated in patients with OSA. It is a multidimensional function where an individual consciously controls effort to guide the operation of various cognitive processes, thereby regulating cognition. Olathe and Bucks conducted a meta-analysis on the effects of OSA and role of CPAP on executive function [22]. Using models of executive functioning from Miyake et al. [23], Fish and Sharp [24], and Adrover-Roig et al. [25], executive functioning was conceptualized as having five subcomponents: (a) shifting between tasks or mental sets, (b) updating and monitoring of working memory representations, (c) inhibition of dominant or pre-potent responses, (d) generativity which is the efficiency of access to long-term memory, and (e) fluid reasoning, an overarching system of reasoning and problem-solving. Olathe and Bucks' [22] meta-analysis evaluated the results from studies examining the impact of OSA on executive functioning compared to controls (21 studies) and before and after treatment (19 studies); their analyses had 5 studies that met inclusion in both categories. They found that all 5 components of executive function were impaired with OSA and that executive function had small to medium improvements with CPAP treatment. Furthermore, they found that age and OSA severity did not modulate the effects of changes in executive function.

Evaluating the effects of OSA on attention, Angelelli et al. studied 32 patients with moderate-to-severe OSA using an extensive computerized battery (test of attentional performance, TAP) that evaluated intensive (i.e., alertness and vigilance) and selective (i.e., divided and selective) dimensions of attention and returned different outcome parameters (i.e., reaction time, stability of performance, and various types of errors) [26••]. OSA patients manifested deficits in both intensive and selective attention processes. The study was limited by a small sample size that was not representative of women; therefore, a large-scale study is recommended to confirm the findings. Another limitation is that they did perform a sleep study to assess the quality and architecture of sleep, so it was unclear whether sleepiness was related to sleep fragmentation or other factors. Finally, the study was not corroborated by a neuroimaging study, which could elucidate the dysfunctional neural mechanisms of cognitive impairment.

To study the effects of OSA on sustained attention also known as vigilance, Huang et al. compared the psychomotor vigilance test reaction time (PVT RT), divided attention

steering simulator (DASS), and the test of attentional performance (TAP) in patients with moderate-to-severe OSA compared to controls [27]. They found that the untreated OSA group had impaired vigilance as indicated by the increase of PVT RT as well as the decreased tonic alertness in the TAP which correlated with an impairment of simulated driving performance. The study was limited by a small sample size. Furthermore, another study found that obesity was also a risk factor for impaired vigilance in patients with OSA. Patients with OSA and obesity defined as a BMI  $\geq 30$  compared to non-obese OSA patients had delayed reaction times in a psychomotor vigilance task and a decrease in working memory [28]. McCloy et al. studied polysomnographic risk factors for vigilance-related cognitive decline in patients with OSA using the psychomotor vigilance task (PVT) [29]. Factors associated with vigilance-related cognitive decline include OSA severity, change in oxygen desaturation, and sleep fragmentation as in the form of sleep arousals. In a retrospective study, Duce et al. found that EEG arousals greater than 5 s had a sensitivity of 70% and a specificity of 66.7% for neurocognitive impairment as measured by the PVT. [30]

ECG slowing has been associated with cognitive impairment. A study comparing patients with OSA and obesity hypoventilation syndrome (OHS) found that patients with OHS demonstrate greater slow frequency EEG activity (increased delta-alpha ratio during sleep and theta power during wake) but no differences in the psychomotor vigilance test pretreatment, compared with equally obese OSA patients with comparable apnea-hypopnea index (AHI) [31••]. Patients with OHS have greater slow frequency EEG activity during sleep and wake than equally obese patients with OSA. Greater EEG slowing was associated with worse vigilance and lower oxygenation during sleep. Quantitative EEG analysis has also been studied in OSA patients. EEG slowing during stage REM sleep was associated with slower PVT reaction times, more PVT lapses, and more driving simulator crashes. Furthermore, decreased spindle density in NREM sleep was also associated with slower PVT reaction times.

## CPAP and Cognitive Function

The majority of studies that has evidenced the effect of OSA treatment on cognitive function have evaluated use of CPAP. A landmark study by Kushida and colleagues known as the Apnea Positive Pressure Long-term Efficacy Study (APPLES) was a 6-month, randomized, double-blind, 2-arm, sham-controlled, and multicenter trial to determine the neurocognitive effects of CPAP therapy on patients with OSA [32••]. Using three neurocognitive variables, Pathfinder Number Test-Total Time (attention and psychomotor function), Buschke Selective Reminding Test-Sum Recall (learning and memory), and Sustained Working Memory Test-

Overall Mid-Day Score (executive and frontal-lobe function), the use of CPAP resulted in mild, transient improvement in the most sensitive measures of executive and frontal-lobe function for those with severe disease, suggesting a complex OSA-neurocognitive relationship. Wang et al. conducted a meta-analysis of randomized controlled trials (RCT) on the cognitive effects of CPAP for OSA [33]. They reviewed 14 RCT with a total of 1926 participants and found that CPAP treatment can partially improve cognitive impairment in the population of severe OSA with significant treatment effect on attention and speed of information processing.

To determine whether subjective improvements in daytime sleepiness, fatigue, and depression experienced by patients with OSA with CPAP therapy predict an objective improvement in vigilance, Bhat et al. studied 182 patients with mild-to-severe OSA with CPAP using the psychomotor vigilance task (PVT) [34]. A total of 182 patients underwent PVT testing and measurements of subjective daytime sleepiness, fatigue, and depression at baseline and after a minimum of one month of adherent CPAP use at an adequate pressure. They found no predictive relationship between subjective improvements in daytime sleepiness, fatigue and depression, and objective vigilance with CPAP use in patients with OSA. The implications of these findings are that improvements in subjective daytime sleepiness and fatigue in patients with OSA are not necessarily reflective of improvements in objective vigilance and vice versa. Unfortunately, the study was limited by a high dropout rate due to patients who declined CPAP, abandoned CPAP, or had suboptimal use. This may have created a self-selection bias where patients who did not benefit from CPAP elected not to continue treatment.

Recent studies examining short-term effects of CPAP had mixed findings. Turner et al. studied a sample of 16 patients with moderate-to-severe OSAS, who were assessed both prior to and after 3 months of CPAP treatment, using a neuropsychological battery and questionnaires to assess mood and anxiety disorders, irritability, quality of life, quality of sleep, and daytime sleepiness [35]. With 3 months of CPAP use, there were significant improvements in executive functions and memory with improvements in working memory, long-term verbal memory, and short-term visuospatial memory. There were no significant changes in mood, anxiety, aggressive behavior, and quality of life, but these measures were normal before the start of treatment. A limitation of the study was lack of a control group and small sample size.

In contrast, Dostálová et al. studied the neurocognitive and neuropsychiatric effects of CPAP in OSA patients [36]. They conducted a cross-sectional, prospective observational study of 126 patients with OSA using the Montreal Cognitive Assessment (MoCA) for the evaluation of cognitive impairment, the Beck Depression Inventory, and the State-Trait Anxiety Inventory, together with the Epworth Sleepiness Scale for the evaluation of neuropsychiatric symptoms and a

person's general level of daytime sleepiness. Short-term (3 months) CPAP treatment of patients with OSA alleviated daytime sleepiness, as well as depressive and anxiety symptoms; however, there was no significant improvement in cognitive performance with use of CPAP. Study limitations are small sample size, no comparison group, and lack of cognitive impairment as measured by the MoCA on the pre-test.

In a large retrospective study, Dunietz et al. evaluated the associations between CPAP therapy, adherence and incident diagnoses of Alzheimer's disease (AD), MCI, and dementia not-otherwise-specified (DNOS) in older adults [37••]. They utilized Medicare claims data of 53,321 beneficiaries, aged 65 +, with an OSA diagnosis. CPAP treatment and adherence were independently associated with lower odds of incident AD diagnoses in older adults. Results suggest that treatment of OSA may reduce risk of subsequent dementia. Strengths of the study included a large, representative sample, longitudinal design that allowed for new dementia diagnosis claims in an OSA cohort, adjustment for other comorbid conditions known to affect dementia risk, and use of fee-for-service data that maximized the likelihood of capturing all relevant claims. Study limitations include a relatively short follow-up interval (3 years), and the study did not take into account the duration of OSA, length of treatment, or degree of adherence to CPAP therapy.

There were two recent studies which evaluated the long-term effects of CPAP use and MCI. Wang et al. studied the effect of CPAP adherence on cognition in older adults with mild obstructive sleep apnea and MCI [38]. Comparing two groups: (a) CPAP adherent group with MCI with an average CPAP use of  $\geq 4$  hours per night and (b) a CPAP nonadherent group with MCI with an average CPAP use of  $< 4$  hours per night. Those in the MCI with CPAP adherence group compared to the MCI with nonadherence to CPAP group demonstrated a significant improvement in psychomotor/cognitive processing speed, measured by the Digit Symbol Coding Test. Study limitations were lack of a randomized control group and possible confounding variables that were not accounted for.

Another study on the long-term effects was by Richards et al. who conducted a quasi-experimental pilot study to examine whether 1-year use of CPAP in adults with OSA and MCI would slow cognitive decline [39]. They compared a CPAP adherent and MCI group to a non-adherent CPAP and MCI group. Memory was assessed using the Hopkins Verbal Learning Test-Revised, and psychomotor/cognitive processing speed was assessed using the Digit Symbol subtest from the Wechsler Adult Intelligence Scale Substitution Test. They found that controlling for baseline differences, 1 year of CPAP adherence in patients with MCI compared to patients with MCI with non-adherence significantly improved cognition and may slow the trajectory of cognitive decline. Improvements were greater in psychomotor/cognitive processing speed, and the effect size was small to moderate for memory and attention. Although both studies are promising on the benefits of CPAP in

patients with MCI, further studies with large, adequately powered studies are needed to confirm these findings.

Not all studies demonstrate a benefit of CPAP on cognitive performance. Skiba et al. assessed the relationship between CPAP use in patients with MCI and in patients with OSA [40]. They conducted a retrospective review comparing 96 patients with MCI and OSA that were CPAP compliant, CPAP noncompliant, and no CPAP use. Patients were followed for 2.8 years, and in contrast to Wang et al. and Richards et al., CPAP use in patients with MCI and OSA was not associated with delay in progression to dementia or cognitive decline. This study was not a randomized trial, which may have introduced bias into overall compliance with medical treatment. Schwartz et al. conducted a systemic review and meta-analysis on the effects of CPAP compared to mandibular advancement device (MAD) in improving the quality of life (sleepiness, cognitive, and functional outcomes) in patients with OSA [41]. There were no significant differences between MAD and CPAP in quality of life, cognitive, and functional outcomes. The study results may have been limited by low treatment compliance.

In summary, there is evidence that CPAP therapy may improve cognitive function or slow the progression to MCI and AD, but large longitudinal studies demonstrating this are lacking. Studies that evaluate cognitive function and the use of CPAP are limited by study design, small sample size, and lack of randomization, and most do not attempt to explain the etiology of the cognitive findings by imaging, biomarkers, or polysomnographic data. Studies are needed on the effects of other OSA treatments and cognitive function.

## Conclusion

OSA is a common clinical condition with a significant impact across cognitive domains. Its greatest impact appears to be on attention, vigilance, and information processing speed. Furthermore, the presence of OSA seems to have a significant impact on development and progression of MCI. Impact of OSA treatment, particularly with CPAP, appears to mitigate and slow the rate of cognitive decline and may reduce the risk of dementia. Larger properly controlled studies, of a prospective nature, are required to further elucidate the degree of treatment effect.

## Compliance with Ethical Standards

**Conflict of Interest** The authors have nothing to disclose.

**Human and Animal Rights and Informed Consent** This article does not contain any studies with human or animal subjects performed by any of the authors



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