Effects of Sleep Apnea Syndrome on Delayed Memory and Executive Function in the Elderly

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Abstract

OBJECTIVE: To identify differences in cognitive function between elderly patients with sleep apnea syndrome (SAS) and healthy controls.

DESIGN: Cross-sectional.

SETTING: Sleep laboratory at Seoul National University Bundang Hospital**.**

PARTICIPANTS: Sixty-three elderly subjects (26 women; mean age, 68.2 ± 4.8) free of cognitive disorders

MEASUREMENTS: Sleep-laboratory polysomnography findings and cognitive function results determined using the Korean version of the CERAD Neuropsychological Assessment Battery.

RESULTS: When the control group (apnea-hypopnea index, AHI < 15) was compared with the mild-to-moderate SAS group (15 ≤ AHI < 30) and with the severe SAS group (AHI ≥ 30), significant differences in delayed recall ($p < .01$) and errors on trail making test B (TMT B; $p < .01$) were observed, with the severe SAS subjects showing greater impairment on both tests than controls ($p < .05$, for both). Stepwise multiple regression showed that oxygen desaturation index ($\beta = -0.37$, $p < .01$) and educational level ($\beta = 0.24$, $p < .05$) determined delayed recall impairment (adjusted $R^2 = 17.8\%$, $p < .01$). TMT B errors were independently associated with educational level ($\beta = -0.41$, $p < .01$) as well as AHI ($\beta = 0.31$, $p < .01$;

adjusted R^2 = 25.7%, p < .01).

CONCLUSION: Severe SAS is associated with measures of delayed recall and executive function in cognitively healthy older adults. Although further study is needed, this evidence may provide further rationale for the treatment of SAS in older adults. Moreover, the role of SAS as a risk factor of cognitive disorders needs to be determined.

Key words: Sleep apnea syndrome; Elderly; Cognition

Introduction

Sleep apnea syndrome (SAS) results from repetitive nocturnal respiratory pauses, and is characterized by nocturnal hypoxemia and frequent arousals during sleep. Recently SAS has become a focus of public health debate due to its association with serious psychological and functional adverse consequences, such as excessive daytime sleepiness and cognitive impairment.[1,2] Reportedly, SAS is quite prevalent in older adults, occurring in 19-57% of this population.[3-5]

Cognitive impairment in the middle-aged due to SAS has been well-described. However, the association between SAS and cognitive dysfunction in the elderly has yet to be determined. Some studies have reported diminished or impaired global cognition,[6,7] vigilance capability, daytime performance,[8] or attention,[9] but other studies have failed to find any cognitive impairment in SAS.[10,11] These conflicting observations regarding the association between SAS and cognitive impairment in the elderly may be attributable to methodological limitations. First, differences in SAS severity among studies might have reduced the effect size of SAS on cognition. The two large epidemiological studies that reported SAS had minimal impact on cognitive function in the elderly enrolled only patients with moderately severe SAS, that is, mild-to-moderate SAS in the Sleep Heart Health Study (SHHS)[11] and

asymptomatic SAS in the SYNAPSE Study.[10] Second, most of such studies examined subjects using home polysomnography,[6,7,10-12] and apnea-hypopnea indices (AHIs) determined by home and laboratory nocturnal polysomnography (NPSG), particularly in severe SAS, are known to disagree.[13,14] Finally, some previous studies have used instruments for measuring cognition that lacked comprehensiveness, and relied on a single measure of global cognition, such as the Mini-Mental State of Examination (MMSE),[7] or on a few cognitive function tests.[6,11] However, since the cognitive impairment associated with SAS may be subtle and/or patchy, comprehensive and sensitive cognitive assessments are warranted.

To investigate cognitive impairments associated with SAS in the elderly, we compared cognitive functioning between elderly SAS patients and controls using a comprehensive and standardized neuropsychological assessment battery. Because the object of this study was to determine the nature of the relationship between SAS and cognition in cognitively healthy elders, we only included the elderly free of mild cognitive impairment (MCI), dementia, and of other cognitive disorders.

Methods

Subjects

The study subjects were 63 elderly persons (≥ 60 years old); 29 subjects from the sleep clinic at Seoul National University Bundang Hospital and 34 community-dwelling, healthy volunteers who had participated in the Nationwide Survey on Dementia Epidemiology of Korea (NaSDeK).[15] Based on clinical interviews and medical records, we excluded subjects with neurodegenerative disease or major depressive disorder or cognitive disorder. We considered AHI ≥ 15 as diagnostic for SAS,[16] and found that 18 subjects had mild-to-moderate SAS (AHI ≥ 15 and <30; the mild-to-moderate SAS group), and 24 had severe SAS (AHI ≥ 30; the severe SAS group). The 21 subjects with AHI < 15 were enrolled as normal controls. The rates of the clinical sample were 14.3% normal controls, 44.4% mild to moderate SAS, and 75% severe SAS. This study protocol was approved by the Institutional Review Board of Seoul National University Bundang Hospital. All study subjects provided written informed consent.

Polysomnography

All subjects underwent NPSG using an Embla^{TM} N 7000 recording system (Embla; Reykjavik, Iceland), with standard electrodes and sensors. Every 30-second epoch of the

NPSG was scored based on the criteria of Rechtschaffen and Kales[17]. Apnea was defined as the complete cessation of airflow for at least 10 seconds. Hypopnea was defined as a substantial (> 50%) reduction in airflow for at least 10 seconds or a moderate reduction in airflow for at least 10 seconds, associated with EEG arousal or oxygen desaturation (≥ 4%).[18] The AHI was defined as the total apnea and hypopnea episodes per hour of sleep. Oxygen desaturation index (ODI) was defined as the number of oxygen desaturation (≥ 4%) events per hour of sleep.

Measurement of cognition and behavioral consequences of SAS

To ensure comprehensive neuropsychological assessments, neuropsychologists administered the Korean version of the CERAD Neuropsychological Assessment Battery (CERAD-K-N)[19] to all subjects, with an interval of less than 6 months (40.2 ± 52.4 days; mean ± SD) between the assessments and NPSG sessions. During this intervening period, no SAS patient was treated for SAS using continuous positive airway pressure (CPAP) or any other method. The CERAD-K-N comprises nine neuropsychological tests: the Categorical Fluency Test (CFT), the modified Boston Naming Test (BNT), the MMSE, the Word List Memory Test (WLMT), the Constructional Praxis Test (CPT), the Word List Recall Test (WLRT), the Word List Recognition Test (WLRcT), the Constructional Recall Test (CRT), and the Trail Making Test (TMT) Parts A &-B. Additionally, we assessed mood (Beck

Depression Inventory, BDI), daytime sleepiness (Epworth Sleepiness Scale, ESS) and subjective sleep disturbances (Pittsburgh Sleep Quality Index, PSQI). All tests were administered and scored as previously described.[20] We diagnosed MCI according to the revised diagnostic criteria of MCI proposed by the International Working Group on MCI.[21]

Statistical analysis

SPSS version 17 for Windows (SPSS Inc, Chicago, IL) was used for all statistical analyses. Intergroup differences in parametric clinical variables were analyzed by analysis of variance (ANOVA) with Tukey's post hoc test. Pearson's or Spearman's correlation were used to determine relationships between cognitive function and clinical or polysomnographic variables. Stepwise multiple regression analysis was used to identify the factors that contribute to cognitive function. Dependent variables included neuropsychological tests that were significantly different between normal control and two SAS groups, and candidate independent variables included demographic and clinical characteristics which were significantly correlated with dependent neuropsychological tests. All beta values were standardized. Two-tailed values of $p < .05$ were considered significant.

Results

Table 1 shows demographic and clinical characteristics by group (control, mild to moderate SAS, and severe SAS). The 63 study subjects comprised 37 men (58.7%) and 26 women (41.3%), aged 60 to 85 years (68.2 ± 4.8; mean ± SD). No significant intergroup differences were found for age, gender, education, or body mass index (BMI). Mean AHI, lowest oxygen saturation, percentage sleep time below 90% oxygen saturation, ODI, and mean arousal index were significantly higher in the severe SAS group than in the control and mild-to-moderate SAS groups ($p < .01$ for each). Subjects with severe SAS tended to have higher PSQI scores than in the other groups, but this did not reach statistical significance ($p = .06$). ESS and BDI scores were no different in the three groups.

Table 2 summarizes cognitive performance by diagnostic groups. Significant differences in WLRT scores ($F=6.272$, $p < .01$) and number of TMT Part B errors ($F=5.128$, $p < .01$) were found between the three groups, with severe SAS subjects showing the greatest degree of impairment. AHIs were correlated with WLRT scores ($r = -.35$, $p < .01$) and with number of TMT B errors ($r = .32$, $p = .01$). ODI, another marker of SAS severity and hypoxia, was also correlated with WLRT scores ($r = -.37$, $p < .01$) and number of TMT B errors ($r = .29$, $p = .02$). Educational level was correlated with both WLRT scores and TMT B errors ($r = .25$, $p = .04$; $r = -.42$, $p < .01$, respectively). Stepwise multiple linear regression analysis

was performed to identify factors that contributed significantly to WLRT score and TMT B errors (Table 3). WLRT was affected by ODI (β = -.37, $p < .01$) and educational level (β = .24, $p < .01$; adjusted R^2 = 17.8%, $p < .01$). In addition, TMT B errors were influenced by educational level (β = -.41, $p < .01$) and AHI (β = .31, $p < .01$; adjusted R^2 = 25.7%, $p < .01$).

Discussion

In a sample of elderly persons free of MCI and dementia, recruited from a sleep clinic and in the community, we found that subjects with severe SAS had deficits in delayed recall (i.e., WLRT) and executive function (i.e., TMT B error) as compared to healthy controls. ODI and educational level were found to be significant predictors of delayed recall, and AHI and educational level were found to predict executive function. These findings suggest that hypoxia is associated with impaired cognitive function in older patients with severe SAS. Hypoxia may negatively impact neuronal integrity in several brain regions, and the prefrontal cortex and hippocampus have the greatest vulnerability to the effects of intermittent hypoxia.[22,23] Both of these regions play pivotal roles in memory and executive function.[22,24] Findley et al. reported that hypoxic older people had more cognitive deficits than non-hypoxic older people, and the degree of this cognitive impairment was correlated with the severity of hypoxia and not with the degree of sleep disruption.[25] Also, Aloia et al[26] found that the degree of sleep-disordered breathing, especially degree of oxygen desaturation, was associated with degrees of delayed verbal recall and reduced constructional abilities in a study of SAS patients older than 55.

We found no differences between healthy controls and SAS subjects for cognitive tests that measure attention and vigilance. Attention and vigilance are mainly affected by

daytime sleepiness,[27,28] and, in the present study, daytime sleepiness was not greater in the SAS patients. Reportedly, the relationship between SAS and daytime sleepiness is weaker in elderly subjects than in middle-aged subjects.[29] Thus, elderly subjects with SAS might maintain attention and vigilance, except for the small proportion that experience excessive daytime sleepiness.

No difference between mild to moderate SAS patients and healthy controls was found in any domain of the CERAD-K-N, and this result is comparable to that of the SHHS study, in which most SAS subjects had mild to moderate SAS.[11] The SYNAPSE study, another well-designed large-scale study, reported that undiagnosed SAS had a limited impact on cognitive function in community-dwelling, healthy subjects, and that only severe SAS subjects tended toward lower cognitive scores on the delayed recall and Stroop tests.[10] However, the SYNAPSE study and the present study differ with respect to both subject recruitment and SAS severities. In the SYNAPSE study, all subjects lived in their communities and had no SAS-related symptoms, but we included subjects visiting a sleep clinic and volunteers from the community. In addition, in the SYNAPSE study subjects with an AHI of > 15 had much lower ODIs than our subjects (14.0 vs. 30.0), which may explain different findings of these two studies about the relationship between SAS and cognition in the elderly.

This study has several limitations that should be considered. First, the CERAD battery might not be sensitive to minor differences in cognitive status among older adults without dementia because the researchers that originally developed the CERAD battery did so to evaluate cognitive function in elderly with cognitive impairments including dementia. Although the CERAD might not be the most sensitive battery for detecting the subtle impacts of medical conditions on cognition, it is robust to ceiling effects in normal elders, particularly in those with wide educational backgrounds, as in Korean elders.[19] In addition, the CERAD battery has been used successfully to determine the impacts of medical/psychiatric conditions on cognition of community-dwelling elders.[30] Second, we invited SAS patients to visit our sleep clinic to participate in the cognitive function test, and thus, we cannot rule out the possibility that patients with cognitive dysfunction might have been more willing to take the test. Such a selection bias could partly explain the cognitive impairment observed in severe SAS patients, because we recruited a much higher proportion of severe SAS patients from the sleep clinic. Third, the lack of corrections for multiple comparisons during the analyses of cognitive function test findings and a cross-sectional study design with small cohort size should be also considered limitations of this study. Future studies with larger sample sizes and more thorough statistical analysis are needed to corroborate our findings.

In conclusion, cognitively healthy elders with severe SAS were found to show impairments in delayed recall memory and executive function as compared with normal controls, and this was associated with hypoxia. However, we found no association between sleepiness and cognitive function in older adults with SAS. The cognitive impairment seen in older adults with severe SAS may provide further indication for treatment of SAS in this population. Further study is needed to determine whether SAS is a risk factor for the development of other cognitive disorders in older adults.

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Conflict of interest statement

The authors have no conflict of interest to declare in relation to this work.

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Table 1. Demographic and Clinical Characteristics of Subjects

	Normal control (n=21)	Mild to moderate SAS (n=18)	Severe SAS (n=24)	F	P
Age, yr	68.7 ± 5.5	66.7 ± 3.6	69.0 ± 4.7	1.41	.25
Male, n (%)	11 (52)	11 (61)	15 (63)	.26	.77
Education, yr	12.4 ± 5.3	14.2 ± 4.2	12.0 ± 5.0	1.10	.34
Body mass index , kg/m^2	24.2 ± 2.8	24.4 ± 2.3	25.8 ± 4.0	1.66	.20
BDI score	9.4 ± 5.5	8.8 ± 8.9	12.2 ± 9.2	1.03	.36
PSQI score	6.2 ± 4.0	5.0 ± 3.1	8.0 ± 4.6	2.96	.06
ESS score	7.1 ± 3.8	8.3 ± 3.6	7.3 ± 4.1	.50	.61
AHI, n/h[*†‡]	10.1 ± 10.6	21.7 ± 3.7	47.8 ± 13.0	76.70	< .01
ODI, n/h[†‡]	7.7 ± 10.0	16.0 ± 5.6	40.5 ± 15.1	50.70	< .01
Nadir SpO_2, %[†‡]	87.1 ± 2.8	83.2 ± 5.8	77.3 ± 7.5	16.05	< .01
Time SpO_2 < 90%, %[†‡]	0.3 ± 0.7	1.9 ± 2.6	10.8 ± 15.3	7.71	< .01
Total sleep time, min	360.7 ± 58.5	381.6 ± 54.9	364.1 ± 44.4	.87	.42
Wake after sleep onset, min	86.7 ± 52.1	79.0 ± 42.9	77.3 ± 35.5	.28	.76
Sleep efficiency, %	78.3 ± 11.3	80.1 ± 10.9	78.4 ± 10.7	.16	.85
Total arousal index n/h[†‡]	9.3 ± 14.1	21.2 ± 14.2	31.5 ± 18.0	11.09	< .01

Data are presented as mean ± SD.

Normal control, AHI<15; Mild to moderate SAS, 15 ≤ AHI < 30; severe SAS, AHI ≥ 30.

SAS, sleep apnea syndrome; AHI, apnea-hypopnea index; BDI, Beck depression inventory; PSQI, Pittsburg sleep quality index; ESS, Epworth sleepiness scale; ODI, Oxygen desaturation index.

[*] $p < .05$, normal controls vs. mild/moderate SAS

[†] $p < .05$, normal controls vs. severe SAS

[‡] $p < .05$, mild to moderate vs. severe SAS

Table 2. Cognitive Function Test Scores in the three study groups

	Normal control (n=21)	Mild to moderate SAS (n=18)	Severe SAS (n=24)	F	P
Verbal Fluency	17.0 ± 4.3	18.0 ±5.0	16.3 ± 4.1	.80	.46
BNT	12.6 ± 1.5	12.9 ± 1.4	11.8 ± 2.5	2.26	.11
MMSE	27.0 ± 1.9	27.6 ± 1.8	26.0 ± 2.9	2.33	.11
WLMT	19.4 ± 3.1	19.8 ± 2.9	17.7 ± 3.8	2.63	.08
WLRT†‡	7.2 ± 1.5	7.4 ± 1.4	6.1 ± 1.2	6.27	<.01.
WLRcT	9.3 ± 0.8	9.7 ± 0.5	9.4 ± 1.0	1.68	.19
CPT	10.2 ± 1.2	10.3 ± .8	9.5 ± 1.7	2.65	.79
CRT	8.0 ± 2.3	8.0 ± 2.9	6.7 ± 2.8	1.89	.16
TMT A error	0.2 ± 0.5	0.4 ± 0.7	0.3 ± 0.5	.62	.54
TMT A time	51.6 ± 16.4	46.5 ± 24.9	64.6 ± 31.4	2.91	.06
TMT B error†	0.8 ± 0.9	1.1 ± 1.7	2.1 ± 1.6	5.13	<.01
TMT B time	147.6 ± 57.4	133.8 ± 81.2	169.8 ± 71.6	1.38	.26

Data are presented as mean ± SD.

Normal control, AHI<15; Mild to moderate SAS, 15 ≤ AHI < 30; severe SAS, AHI ≥ 30.

SAS, Sleep apnea syndrome; MMSE, Mini-Mental State Examination; BNT, Boston naming test; WLMT, word list memory-immediate test; WLRT, word list-delayed free recall test; WLRcT, word list-recognition test; CPT, constructional performance; CRT, Constructional recall test; TMT, trail making test;

†$p < .05$, normal controls vs. severe SAS

‡$p < .05$, mild to moderate vs. severe SAS

Table 3. Stepwise Multiple Linear Regression Model of WLRT score and TMT B error

		Adjusted R^2	β	*p*
WLRT	ODI	.12	-.37	<.01
	ODI, Education level	.17		
	(ODI)	.12	-.36	<.01
	(Education level)	.06	.24	.04
TMT B error	Education level	.16	-.42	<.01
	Education level, AHI	.25		
	(Education level)	.16	-.41	<.01
	(AHI)	.09	.31	<.01

Only statistically significant variables are presented in the table.

WLRT, word list-delayed free recall test; TMT, trail making test; AHI, Apnea-hypopnea index; ODI, Oxygen desaturation index.