Neurocognitive Dysfunction Associated With Sleep Quality and Sleep Apnea in Patients With Mild Cognitive Impairment

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Objectives: *Sleep apnea syndrome (SAS) is considered a risk factor for cognitive decline in the elderly. The specific neurocognitive decline has been suggested as a predictive factor for dementia in patients with mild cognitive impairment (MCI). The authors aim to illustrate the sleep characteristics related to the specific neurocognitive decline in the community-dwelling elderly including patients with MCI.* **Design:** *Cross-sectional.* **Settings:** *Center for sleep and chronobiology in Kangwon National University Hospital.* **Participants:** *Thirty patients with MCI and 30 age- and sex-matched normal elderly subjects were selected.* **Measurements:** *The authors administered seven tests in the Korean version of the Consortium to Establish A Registry of Alzheimer's Disease Neuropsychological battery and conducted nocturnal polysomnography. A p value below 0.05 was considered a statistical significance.* **Results:** *There was no significant difference in sleep parameters between the MCI and normal comparison (NC) groups. Sleep efficiency was positively correlated with Constructional Recall (CR) scores in both NC and MCI groups (r = 0.393 and 0.391, respectively). The amount of slow wave sleep (SWS) was also positively correlated with Boston naming test (BNT) scores in both groups (r = 0.392, 0.470, respectively). Stepwise multiple regression models showed that SWS and the apnea index were significant independent variables associated with the BNT score ($\Delta\beta = 0.43$ and -0.34, respectively; adjusted $R^2 = 0.298$) in the MCI group, and the amount of rapid eye movement sleep was a significant independent variable associated with the CR score ($\Delta\beta = 0.49$; adjusted $R^2 = 0.217$) in the NC group.* **Conclusions:** *Our results show that poor sleep quality and greater severity of SAS were associated with impaired language function reflecting frontal-subcortical pathology in patients with MCI. This suggests that vulnerability to a specific brain damage associated with SAS could increase the risk for dementia.* (Am J Geriatr Psychiatry 2011; 19:374–381)

Key Words: MCI, SAS, poor sleep quality, neurocognitive function

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DOI: 10.1097/JGP.0b013e3181e9b976

Sleep disorders have been considered risk factors for cognitive impairment in the elderly,[1–3] and their prevalence in the elderly who have dementia has been reported to be higher than that in the normal elderly.[4,5] In Alzheimer disease (AD), disruption of sleep-wake rhythm, impaired sleep quality, and sleep apnea syndrome (SAS) are very common. These conditions could be related to the degenerative change in the neurons that are involved in sleep-wake generation and respiration.[6] Cognitive impairment caused by SAS would increase the risk of dementia.[7]

It has been shown that neurocognitive impairments in various domains are attributable to SAS. Its main mechanism is intermittent hypoxemia due to repetitive apneic events and excessive daytime sleepiness (EDS) due to sleep fragmentation.[8] The greater the severity of dementia, the higher the prevalence and degree of SAS.[4,5] The Apolipoprotein E (APOE) ϵ4 genotype, which is known as the risk factor for AD, may also increase the risk for SAS.[9,10] Therefore, its relationship with SAS will be considerable in the elderly with cognitive impairment. Besides, SAS can result in frontal lobe and subcortical damage,[11,12] which is associated with diminished attention spans, memory, and executive functions.[13] The poor performance on delayed recall and executive function tests[12] and the poor performance on delayed recall and category fluency tests[14] have been reported to be the best predictors of a conversion from mild cognitive impairment (MCI) to AD. We assume that subcortical damage could easily affect the cognitive decline in AD, because its correlation was not found in the nondemented elderly but was found in patients with early AD.[15] In other words, the susceptibility to the specific cognitive decline precipitated by SAS would be greater in the elderly who are at risk for dementia and would affect the occurrence of dementia directly or indirectly. EDS, which can be resulted from SAS, usually becomes worse as AD progresses. Several studies have suggested the relationship of EDS with the occurrence of dementia.[2,16]

As the cognitive decline in AD occurs by cerebral neurodegenerative change, the illustration of the causal relationship of sleep disorder with cognitive decline in AD would be difficult to verify. We would rather pay attention to MCI, in which pharmacological interventions could delay progress to full dementia.[17] However, the risk of dementia in patients with MCI seems to be high, for the conversion rate to dementia in patients with MCI has been reported as 12%–15% per year.[18] Therefore, investigation of the relationship between sleep disorder and neurocognitive dysfunction in the elderly with MCI would be meaningful, illustrating a possible risk factor for dementia. Previous studies have limitations in explaining the clinical characteristics of sleep disorders associated with the progression of dementia and do not assess various cognitive domains. We aimed to compare nocturnal sleep characteristics in patients with MCI with those of a matched group of normal older adults and to illustrate which sleep parameters are associated with specific neurocognitive dysfunction in both groups.

METHODS

Subjects

Among the elderly who visited the Public Health Center in Chuncheon City, those older than 60 years were recruited for this study from March 2005 until November 2007. The study protocol was approved by the Institutional Review Board of Kangwon National University Hospital. Written informed consent was obtained from each enrolled patient. The diagnosis of MCI was made using Petersen's criteria,[18] which include the presence of subjective or objective memory impairment without dementia and a score below the 1.5 standard deviation of normative value in at least one neurocognitive test in the Korean version of the Consortium to Establish a Registry for Alzheimer's Disease (CERAD-K) Neuropsychological battery. We applied the following exclusion criteria: 1) the presence of dementia, alcohol dependence, other substance abuse, depressive disorder affecting cognitive function, and other major psychiatric disorders by *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition,* criteria[19]; 2) history of cerebrovascular disease, central nervous system (CNS) disease, or damage affecting cognitive functions; 3) current use of hypnotics or CNS active drugs affecting cognitive function, or present illnesses including chronic renal failure (CRF), chronic obstructive pulmonary disease (COPD), cancer, uncontrolled diabetes, and uncontrolled hypertension; 4) chronic insomnia or restless legs syndrome; and 5) significant impairment of hearing ability, visual

acuity, or language ability, which hindered the completion of neurocognitive tests. Any subjects with abnormalities in their complete blood cell count, liver function test, urine analysis, electrocardiogram (ECG), or chest X-ray were also excluded. Seventeen patients were excluded: four patients with depressive disorder, four with CNS disease, one with uncontrolled diabetes, three with cancer, one with COPD, one with chronic insomnia, two with laboratory abnormalities, and one who refused the nocturnal polysomnography (NPSG). Normal elderly subjects were recruited by age and sex matching to patients with MCI, and we applied the above exclusion criteria. Normal comparison (NC) subjects were defined by the scores of seven neurocognitive tests, which were above −1.0 standard deviation of each mean score.

Thirty patients with MCI and 30 NC subjects were finally selected. For each subject, we administered a 30-item version of the Geriatric Depression Scale (GDS), Epworth Sleepiness Scale (ESS),[20] and Sleep Apnea subscale of the Sleep Disorders Questionnaire.[21] The semistructured interview was performed by a psychiatrist using the CERAD-K Assessment Packet.[22,23]

Neuropsychological Assessment

Seven neurocognitive tests from the CERAD-K Neuropsychological battery were administered: Verbal Fluency (VF), Modified Korean version of the Boston Naming Test (BNT), Word List Memory, Constructional Praxis (CP), Word List Recall, Word List Recognition, and Constructional Recall (CR). The scores of neurocognitive tests were analyzed after adjusting for age, sex, and education.

Laboratory-Based Sleep Measures

All subjects were studied on a single night of laboratory-based polysomnography (PSG). An Embla polysomnographic system (Embla S7000; Medcare system, NY) was used for digital recordings, which included electroencephalograms (C3-A2, C4-A1, O1- A2, and O2-A2 by the international 10–20 system), chin electromyogram (EMG), electrooculogram, ECG, snoring assessment, assessment of respiratory efforts using piezoelectric belts over the chest and abdomen, and assessment of airflow at the nose and mouth using a thermistor. We also recorded bilateral surface EMGs on the legs (with electrodes placed over the anterior tibialis muscles). We monitored oxygen saturation by pulse oximetry. A polysomnographic technologist, who has interrater reliability above 90%, when compared with a psychiatrist certified by the American Board of Sleep Medicine, manually scored all recordings of the sleep stages,[24] limb movements,[25] and respiratory events using standard techniques.[26] The oxygen desaturation index was defined as the mean number of events with decreased oxygen saturation of more than 4% per hour during sleep. SAS was defined by the apnea-hypopnea index (AHI) greater than 5; obstructive sleep apnea-hypopnea syndrome was defined by AHI above 5 and the central apnea index below 5. Periodic limb movements (PLMs) were defined by consecutive limb movements of more than 4 times with a duration of 0.5–5.0 seconds every 5–90 seconds. The diagnosis of PLM in sleep was given when the PLM index was more than 15.[27]

Statistical Analysis

The comparisons of demographic data, neurocognitive test scores, and sleep parameters between the MCI and NC groups were performed using a paired *t* test, and comparisons of gender and diagnoses of sleep disorders were performed using a McNemar test. Adjustments in the ESS and GDS scores, and partial correlation analyses between sleep parameters and neurocognitive measures were performed in each group. Stepwise multiple regression analysis was used to identify independent sleep variables predicting the change in neurocognitive functions in each group. All statistical analyses were done using SPSS 11.5 version, and a two-tailed p value below 0.05 had a statistical significance.

RESULTS

There was no difference in age, sex, education, and body mass index between the MCI and NC groups. The GDS scores in patients with MCI were significantly higher than those of NC subjects, although the ESS and Sleep Apnea subscale scores were not (Table 1).

There were no differences in any sleep measures between the MCI and NC groups in our study (Table 2). In the MCI group, 23 patients were diagnosed with SAS (77%), 21 patients with obstructive sleep apnea-hypopnea syndrome (70%), and 9 patients with PLM in sleep (30%). These findings were not significantly different from those of the NC group (Table 3).

TABLE 1. Demographic and Clinical Characteristics in the Normal Comparison (NC) and Mild Cognitive Impairment (MCI) Groups

	Mean (SD) or Ratio				
	NC Group (N = 30)	MCI Group (N = 30)	*t*	*df*	p
Age, years	67.37 (3.75)	67.97 (4.09)	−1.814	29	0.080
Sex (M:F)[a]	9:21	9:21	—	—	1.000
Education, years	7.90 (5.11)	6.80 (4.67)	0.949	29	0.350
BMI	24.49 (2.75)	24.40 (3.28)	0.121	29	0.905
GDS[b]	7.93 (6.81)	11.59 (5.64)	−2.159	27	0.040
ESS	4.17 (3.36)	4.37 (3.98)	−0.216	29	0.831
SA	21.86 (4.45)	21.47 (4.77)	0.192	28	0.849

Notes: p values were derived from paired *t* test. SD: standard deviation; BMI: body mass index; GDS: Geriatric Depression Scale; ESS Epworth Sleepiness Scale, SA: Sleep Apnea subscale of Sleep Disorders Questionnaire.

[a]p values were derived from McNemar test.

[b]Significant difference between groups ($p < 0.05$).

TABLE 2. Comparison of Sleep Parameters Between the Normal Comparison (NC) (N = 30) and Mild Cognitive Impairment (MCI) Groups (N = 30)

	Mean (SD)				
	NC Group	MCI Group	*t*	*df*	p
TST	366.50 (38.68)	358.15 (61.65)	0.646	29	0.523
WASO	62.03 (33.86)	73.57 (43.49)	−1.208	29	0.237
SE (%)	83.93 (8.55)	79.75 (11.44)	1.654	29	0.109
S1 (%)	12.61 (5.96)	14.09 (11.34)	−0.593	29	0.558
S2 (%)	54.50 (10.05)	53.48 (13.53)	0.310	29	0.759
SWS (%)	13.97 (8.45)	14.32 (10.60)	−0.140	29	0.890
REMS (%)	18.92 (6.46)	17.85 (5.81)	0.698	29	0.491
AHI (n/hour)	15.00 (13.56)	13.41 (11.61)	0.682	29	0.501
AI (n/hour)	4.54 (6.57)	4.27 (6.76)	0.175	29	0.862
HI	11.74 (12.86)	9.15 (7.72)	1.473	29	0.152
LOS (%)	84.80 (5.76)	85.97 (5.04)	−0.998	29	0.326
ODI	8.14 (9.97)	6.04 (6.32)	1.153	29	0.258
LMI	18.08 (30.77)	17.90 (28.82)	0.023	29	0.982

Notes: p values were derived from paired *t* test. SD: standard deviation; TST: total sleep time (minutes); WASO: waking time after sleep onset (minutes); SE: sleep efficiency (%); S1: Stage 1 sleep (%); S2: Stage 2 sleep (%); SWS: slow wave sleep (%); REMS: rapid eye movement sleep (%); AHI: apnea-hypopnea index (n/hour); AI: apnea index (n/hour); HI: hypopnea index (n/hour); LOS: lowest oxygen saturation (%); ODI: oxygen desaturation index (n/hour); LMI: limb movement index (n/hour).

TABLE 3. Diagnoses of Sleep Disorders in the Normal Comparison (NC) (N = 30) and Mild Cognitive Impairment (MCI) Groups (N = 30)

	NC Group, N (%)	MCI Group, N (%)	p
SAS	22 (73)	23 (77)	1.000
OSAHS	21 (70)	21 (70)	1.000
PLMS	11(37)	9 (30)	0.791
SAS and PLMS	10 (33)	6 (20)	0.344
OSAHS and PLMS	10 (33)	5 (17)	0.227
Others	7 (23)	4 (13)	0.375

Notes: p values were derived from McNemar test. OSAHS: obstructive sleep apnea-hypopnea syndrome; PLMS: periodic limb movements in sleep.

In the NC group, wake time after sleep onset was negatively correlated with the scores of VF, CP, Word List Recall, and CR ($r = -0.388$, -0.531, -0.395, and -0.381, respectively), and sleep efficiency (SE) was positively correlated with the scores of VF, CP, and CR ($r = 0.407$, 0.434, and 0.393, respectively). Slow wave sleep (SWS) amount was positively correlated with BNT scores ($r = 0.392$), and amounts of rapid eye movement (REM) sleep (REMS) were positively correlated with CR scores ($r = 0.548$) (Table 4).

In the MCI group, SE was positively correlated with CR ($r = 0.391$), and Stage 1 sleep amounts were negatively correlated with BNT scores ($r = -0.487$). SWS was also significantly correlated with the scores of VF and BNT ($r = -0.389$ and 0.470, respectively). The apnea index (AI) was negatively correlated with BNT scores ($r = -0.451$), and lowest oxygen saturation was correlated positively with VF scores($r = 0.418$) (Table 5).

Stepwise multiple regression models showed that SWS and AI were significant independent variables associated with the BNT score ($\Delta\beta = 0.43$, and -0.34, respectively; adjusted $R^2 = 0.298$) in the MCI group, and REMS was an independent variable associated with the CR score ($\Delta\beta = 0.49$; adjusted $R^2 = 0.217$) in the NC group (Table 6).

DISCUSSION

Nocturnal sleep characteristics and the severity of SAS between the MCI and NC groups were not found to be significantly different in our study (Table 2). Previous reports have found the greater the severity of cognitive impairment, the greater the severity

TABLE 4. Correlations of Sleep Parameters With the Scores[a] of Neurocognltive Function Tests in the Normal Comparison (NC) Group (N = 30)

	VF	BNT	WLM	CP	WLR1	WLR2	CR
WASO (m)	−0.388[b]	−0.031	0.018	−0.531[c]	−0.395[b]	−0.032	−0.381[b]
SE (%)	0.407[b]	−0.008	−0.129	0.434[b]	0.282	0.085	0.393[b]
S1 (%)	−0.148	0.062	0.064	−0.206	− 0.181	0.014	−0.546[c]
S2 (%)	−0.163	−0.237	−0.028	0.112	0.038	0.213	0.122
SWS (%)	0.012	0.392[b]	0.117	− 0.223	−0.050	−0.011	−0.203
REMS (%)	0.332	−0.202	−0.158	0.288	0.160	−0.288	0.548[c]
AHI	−0.347	− 0.061	0.148	0.049	−0.042	0.025	−0.085
AI	−0.162	−0.232	0.177	0.130	0.089	0.246	0.021
LOS (%)	−0.049	−0.133	0.051	−0.036	−0.105	0.184	0.050

Notes: Data are partial correlation coefficients with controlling for the scores of Epworth Sleepiness Scale and Geriatric Depression Scale. VF: verbal fluency; BNT: Boston Naming Test; WLM: Word List Memory; WLR1: Word List Recall; CP: constructional praxis; WLR2: Word List Recognition; CR: constructional recall.

[a]Scores adjusted for age, sex, and education.

[b]p <0.05.

[c]p <0.01.

TABLE 5. Correlations of Sleep Parameters With the Scores[a] of Neurocognitive Function Tests in Mild Cognitive Impairment (MCI) Group (N = 30)

	VF	BNT	WLM	CP	WLR1	WLR2	CR
WASO (m)	−0.143	−0.238	−0.305	0.108	−0.224	0.100	−0.352
SE (%)	0.093	0.172	0.161	−0.081	0.195	−0.080	0.391[b]
S1 (%)	0.008	− 0.487[c]	−0.149	.214	−0.327	0.238	−0.231
S2 (%)	0.374	−0.025	−0.109	−0.092	−0.072	−0.255	−0.066
SWS (%)	−0.389[b]	0.470[b]	0.198	−0.040	0.299	0.030	0.182
REMS (%)	−0.090	0.092	0.220	−0.190	0.287	0.010	0.283
AHI	−0.323	− 0.263	0.051	0.075	−0.062	0.168	0.136
AI	−0.234	−0.451[b]	0.018	0.044	−0.194	0.251	−0.144
LOS (%)	0.418[b]	−0.165	−0.262	0.225	−0.267	−0.099	−0.051

Notes: Data are partial correlation coefficients with controlling for the scores of Epworth sleepiness scale and Geriatric depression scale.

[a]Scores adjusted for age, sex, and education.

[b]p <0.05.

[c]p <0.01.

TABLE 6. Stepwise Regression Models of Constructional Recall in NC Group (N = 30) and Boston Naming Test in MCI Group (N = 30)

	$\delta\beta$	*t*	p
Constructional Recall[a] (NC group): Adjusted $R^2 = 0.217$, (*df*) $F = (1, 28)$ 9.02 (p = 0.006)			
REMS	0.49	3.00	0.006
Boston Naming Test[a] (MCI group)			
Model 1—Adjusted $R^2 = 0.210$, (*df*) $F = (1, 28)$ 8.70 (p = 0.006)			
SWS	0.49	2.95	0.006
Model 2—Adjusted $R^2 = 0.298$, (*df*) $F = (2, 27)$ 7.16 (p = 0.003)			
SWS	0.43	2.75	0.010
AI	−0.34	−2.13	0.043

[a]Scores adjusted for age, sex, and education.

of SAS and nocturnal sleep disruption.[4,28,29] In contrast to our study, those studies were mostly done on patients with severe dementia in nursing homes,[4,28] and the assessment of sleep was done with self-report questionnaires.[29] Bonanni et al.[30] show no difference in the severity of SAS between patients with mild dementia and normal elderly subjects, suggesting that nocturnal sleep disruption would not be overt in patients with MCI. Furthermore, sleep can be affected by medical illnesses or various

medications, which are highly concurrent, especially in patients with dementia.[31] Although subjects with these confounding factors affecting nocturnal sleep were strictly excluded, 10 patients with severe SAS with an AHI above 30 were included in our study. Because seven of them belonged to the NC group, the difference in SAS severity might not have been found between the two groups.

The diagnostic distribution of sleep disorders in the MCI group did not differ from that of the normal elderly group in our study (Table 3). Epidemiological studies have consistently shown that SDB increases with age in the general population.[4,5] The proportion of SAS according to the criteria of AHI above 5 was 77% in the MCI group, which was similar to 81% in the study by Ancoli-Israel et al.,[5] applying the same criteria of AHI in the community-dwelling elderly.

Previous studies reported cognitive functions in various domains were affected in obstructive sleep apnea (OSA) patients.[4–8,32] Although it has been reported that the language function was relatively spared in OSA patients, its basis is not clear.[32] In our study, based on NPSG data, the greater severity of SAS was associated with decreased language functions in patients with MCI (Table 5). The BNT score in patients with MCI had a negative correlation with AI ($r = -0.419$, $p = 0.03$), and the VF score tended to be positively correlated with lowest oxygen saturation ($r = 0.384$, $p = 0.053$) after controlling SWS and the scores of ESS and GDS by partial correlation analysis. Stepwise multiple regression analysis also showed that AI was a significant variable predicting the BNT score along with SWS (Table 6).

In previous studies on the community-dwelling elderly, only the APOE-e4-positive group revealed a significant relationship of AHI with cognitive dysfunction.[10] Similar to our result, this suggests the vulnerability to further cognitive impairment was related to SAS in the elderly with cognitive dysfunction. Language function could be associated with the frontal-subcortical area[33] in addition to multiple regions of the cerebral cortex.[34] Previously, decreased executive function and subcortical damage were proposed as predictive factors for converting MCI into dementia.[35] Recently, Artero et al.[14] also reported, on the basis of a 2-year follow-up study, that the best predictor of dementia was a decreased language function in the elderly with memory impairment. A future neuroimaging study could verify whether SAS is associated with a specific brain region affecting language function.

Our study based on NPSG data showed that SE was related to visual memory function in both the normal elderly and MCI groups. However, the sleep measures reflecting sleep quality were not associated with the visuospatial and delayed verbal memory function in patients with MCI but in normal subjects (Tables 4 and 5). It has been reported that poor sleep quality in elderly subjects mainly affects attention, reaction time, and memory function.[36] However, there has been no study on the relationship of neurocognitive function to objective sleep parameters in the community-dwelling elderly.

It has been known that visuospatial ability and delayed verbal recall could be robustly impaired in early stages of dementia, when compared with other cognitive domains, and measures of these domains were useful discriminators between patients with early AD and nondemented elderly subjects.[37] Because the impairment in such domains might be more prominent than those of other domains in patients with MCI such as patients with early dementia, a possible association of sleep quality with these cognitive functions may have been masked in the patients with MCI of our study. That is, the existence of the specific neurocognitive impairment associated with poor sleep quality in the MCI group could be different from the normal elderly group.

Our study showed that decreased SWS is related to impaired language function in both the normal elderly and MCI groups (Tables 4 and 5). Stepwise regression models also show that SWS was a significant predicting variable of the BNT score (Table 6). Language dysfunction is associated with frontal lobe pathology, and the basal forebrain cholinergic system is known to play key roles in sleep regulation and neurocognitive functions including working memory and learning.[38] If there is a lesion in the area, which is closely related to AD pathology,[39] it would result in SWS decrement.[40] It suggests that the decrement of SWS sensitivity reflects the frontal lobe pathology in patients with MCI.

There was no relationship to REMS with any neurocognitive function in patients with MCI, but its relationship to CR was shown in normal elderly subjects (Tables 4 and 5). Stepwise multiple regression analysis in the NC group also show that REMS was the only significant variable predicting the CR score

among wake time after sleep onset, SE, Stage 1 sleep, and REMS (Table 6). REMS disruption usually occurs in severe dementia but is not robust in mild dementia.[41] In addition, the converting rate of MCI to dementia has been reported as 12%–15% per year, meaning a relatively rapid cognitive impairment.[18] This suggests the effect of decreased REMS on cognitive dysfunction would have been relatively smaller than the degree of cognitive impairment itself in patients with MCI.

Our study suggests that the impairment of language function and visual memory might be associated with frontal lobe dysfunction. The frontal lobe activity was relatively diminished during sleep and mostly diminished during SWS and REM sleep in previous neuroimaging studies.[42,43] The frontal lobe is also known to be very sensitive to sleep fragmentation or sleep disruption including decreased SWS and REMS. Accordingly, these cognitive dysfunctions possibly reflect frontal lobe damage.

In patients with MCI, GDS scores were significantly higher than those of the normal elderly in our study (Table 1). Although the concurrence of depression in the early stage of AD is relatively common, the temporal relationship between depression and dementia is not conclusive.[44] Devanand et al.[45] showed that more than 90% of the nondemented elderly with depression were diagnosed as having dementia at the follow-up in a large population study, suggesting that depression is a precursor of dementia. In our study, the significant correlations of the GDS scores with the scores of neurocognitive tests were not found in the MCI group or the normal elderly group ($p > 0.05$, $df = 26$, partial correlation analysis controlling ESS score).

Our study has some limitations. First, our result of comparing sleep parameters between the NC and MCI groups is not conclusive, because effect size (Cohen's d value <0.5) was not relatively large. There remains the possibility of Type I error, using many parameters in our correlation analyses without statistical adjustment. A further increment in the number of subjects might strengthen our findings. Second, frontal lobe function tests were not performed, although SAS and poor sleep quality are closely associated with frontal lobe dysfunction. Third, the neurocognitive function was not assessed longitudinally. Fourth, the variables of sleep architecture could have been influenced by the first- night effect, because single-night PSG for each subject was conducted because of the cost for two-night PSGs in our study. Nevertheless, this is the first study to examine the relationship of sleep characteristics with neurocognitive function in patients with MCI and to illustrate specific cognitive dysfunctions related to SAS as one of the possible contributing factors in the occurrence of dementia.

In our study, there was no difference in nocturnal sleep characteristics and the severity of SAS between patients with MCI and normal elderly subjects. However, the SAS was associated with language dysfunction in patients with MCI. Particularly, the occurrence of language dysfunction reflecting frontal-subcortical pathology in patients with MCI suggests that the vulnerability to specific brain damage related to SAS increase the risk for dementia. Decreased SWS is also associated with the impaired language function in both groups. A further study on the relationship of SAS to the frontal lobe function in patients with MCI is necessary.

Technical support for the statistical analysis of our study was given by Dr. Hui S. Cho, Associate Professor of the Department of Preventive Medicine, Kangwon National University College of Medicine, Chunchon, Kangwon-do, Republic of Korea.

This research was supported by the grant (R04–2004-000-10131–0) from the Korea Research Foundation (Seoul, Korea).

A portion of this work was presented at the 20th Anniversary Meeting of the Associated Professional Sleep Societies in Salt Lake city, UT, June 21, 2006.

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