Does age worsen EEG slowing and attention deficits in obstructive sleep apnea syndrome?

Annie Mathieu a,b, Stéphanie Mazza a, Dominique Petit a, Anne Décary a,c, Jessica Massicotte-Marquez a,d, Jacques Malo e, Jacques Montplaisir a,c,*

a Centre d'étude du sommeil et des rythmes biologiques, Hôpital du Sacré-Coeur de Montréal, 5400 boul. Gouin Ouest, Montréal, Que., Canada H4J 1C5
b Department of Medical Bioscience, Université de Montréal, Canada
c Department of Psychiatry, Université de Montréal, Canada
d Department of Psychology, Université de Montréal, Canada
e Service de pneumologie, Hôpital du Sacré-Coeur de Montréal, Montreal, Que., Canada

Accepted 16 April 2007
Available online 15 May 2007

Abstract

Objective: The aim of this study was to determine whether EEG slowing is more pronounced in older than younger OSAS patients and to verify whether this cortical slowing is correlated to daytime performance, respiratory perturbation and sleep fragmentation.

Methods: Twelve young OSAS patients (mean age 38.2 ± 2.0 y) and 13 older OSAS patients (mean age 62.2 ± 1.9 y) along with 13 young controls (mean age 35.8 ± 2.0 y) and 14 older controls (mean age 60.2 ± 2.0 y) underwent a polysomnographic evaluation followed by a waking EEG recording. As a global index of cortical slowing, a ratio of slow-to-fast frequencies was calculated in all cortical regions. Daytime performance was assessed using the four choice reaction time test.

Results: Differences in waking EEG and in daytime performance were analyzed by ANOVAs with Group and Age as factors. Waking EEG did not yield a Group by Age interaction. OSAS patients had higher ratios across all regions than controls. Similarly, daytime performance revealed no Group by Age interaction. However, OSAS patients showed more lapses than controls and older subjects were slower than younger subjects.

Conclusions: Our results indicate that age does not interact with OSAS to worsen the severity of cortical slowing, but age can add to the OSAS effect to worsen daytime performance deficits in OSAS patients.

Significance: The daytime performance deficits observed particularly in elderly OSAS patients warrant a careful clinical assessment of these patients to prevent accidents and injuries.

Keywords: Age; OSAS; Waking quantitative EEG; Daytime performance

1. Introduction

In adults, the obstructive sleep apnea syndrome (OSAS) is characterized by repeated episodes of apnea and hypopnea lasting for more than 10 s despite a persistent respiratory effort. Apneas and hypopneas cause intermittent nocturnal hypoxemia, micro- arousal and sleep fragmentation. Excessive daytime sleepiness (EDS) and snoring are typical symptoms of OSAS. Obesity, abnormal hormonal regulation of upper airway musculature and genetic predisposition are frequent risk factors whereas cardiovascular problems (congestive heart failure, arrhythmias), mood and cognitive disturbances and poor quality of life are common devastating consequences (see Pack, 2006 for a review). OSAS is a prevalent sleep problem, affecting 4% of men and 2% of women with EDS as a concomitant criterion (Davis and Stradling, 1996). In aging individuals, prevalence rates go up to 24% (Ancoli-Israel et al., 2001).

Neuropsychological testing has revealed that OSAS patients exhibit a pronounced alteration of the attentional system (Verstraeten et al., 1997; Ferini-Strambi et al., 2003;
Mazza et al., 2005), daytime vigilance (Verstraeten et al., 2000; Mazza et al., 2005) and executive cognitive control capacities (Bédard et al., 1991; Naegele et al., 1995; Ferini-Strambi et al., 2003). To a lesser extent, patients with OSAS show impaired memory (Bédard et al., 1991; Rouleau et al., 2002; Ferini-Strambi et al., 2003), motor coordination (Bédard et al., 1991) and visuo-constructive abilities (Greenberg et al., 1987). In parallel, quantitative EEG (qEEG) studies in OSAS patients demonstrate an EEG slowing during rapid eye movement (REM) sleep in the frontal, central and parietal regions whereas during wakefulness, EEG slowing was observed across all cortical regions (Morisson et al., 1998), and more prominently in the frontal and central regions (Morisson et al., 2001). A compounded effect of sleep fragmentation and intermittent nocturnal hypoxemia could explain the EEG, behavioral and cognitive impairments observed in OSAS patients (see Beebe and Gonzal, 2002; for a review).

There are several similarities between the clinical manifestations of OSAS and those related to normal aging, such as snoring, sleep fragmentation (increased stage 1 and microarousal index, numerous sleep transitions), EDS (with frequent napping), hypertension, cardiovascular diseases, attention dysfunction, executive dysfunction, and memory loss. Studies using qEEG analysis during wakefulness have shown increased slow alpha, theta and delta power with advancing age (Breslau et al., 1989). Similar changes have been reported in patients with OSAS (Morisson et al., 1998, 2001).

Sleep abnormalities and daytime functioning impairment have been widely documented in patients with OSAS, but only a few studies have assessed the effects of age on OSAS characteristics. It is generally agreed that age induces EEG slowing and attention deficits, but does OSAS hasten the process? The aim of the present study was to evaluate the effects of age on waking qEEG and daytime vigilance performance of OSAS patients. We hypothesized that older OSAS patients would have more cortical slowing across all cortical regions and poorer sustained attention compared to younger OSAS patients and controls.

2. Methods

2.1. Subjects

Twelve young OSAS patients (10 men, 2 women; age range 29–50 yrs) and 13 older OSAS patients (13 men; age range 54–74 yrs) were recruited at the Sleep Disorders Center and Pneumology Department of the Sacré-Coeur Hospital in Montreal (Canada). Thirteen young controls (12 men, 1 woman; age range 25–50 yrs) and 14 older controls (14 men; age range 51–72 yrs), matched for education, were recruited from the Montreal area through newspaper advertisements. Both OSAS patients and controls were divided into two age groups with cut-off at 50 years to obtain two sufficiently large subgroups representative of the range of the consulting OSAS patients (between 25 and 75 years of age) with similar symptom severity while keeping the age span and age standard deviation similar in each subgroup (see Table I for subject characteristics).

The inclusion criterion for OSAS patients was an obstructive apnea index (number per hour of sleep) ≥10. Special care was taken to obtain two OSAS patient subgroups (younger and older) with similar symptom severity. On the other hand, controls had to have an apnea–hypopnea index (AHI) lower than five. Exclusion criteria for

<table>
<thead>
<tr>
<th>Table 1 Subject characteristics (mean and standard deviation)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>OSAS</strong></td>
</tr>
<tr>
<td>---</td>
</tr>
<tr>
<td>Age (mean y)</td>
</tr>
<tr>
<td>Education (y)</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
</tr>
<tr>
<td>Neck circumference (cm)</td>
</tr>
<tr>
<td>BDI (score)</td>
</tr>
<tr>
<td>ESS (score)</td>
</tr>
<tr>
<td>AHI (no./h)</td>
</tr>
<tr>
<td>AI (no./h)</td>
</tr>
<tr>
<td>Mean SaO₂ (%)</td>
</tr>
<tr>
<td>SaO₂ &lt; 90% (min)</td>
</tr>
<tr>
<td>Total sleep time (min)</td>
</tr>
<tr>
<td>Sleep efficiency (%)</td>
</tr>
<tr>
<td>Micro-arousals index (no./h)</td>
</tr>
<tr>
<td>Stage 1 (%)</td>
</tr>
<tr>
<td>Stage 2 (%)</td>
</tr>
<tr>
<td>SWS (%)</td>
</tr>
<tr>
<td>REM sleep (%)</td>
</tr>
</tbody>
</table>

n.a., non applicable.
 n.s., non significant.
* Main Group effect p < 0.05.
† Main Age effect p < 0.05.
both groups were the presence of medical, neurological or psychiatric disease, sleep disorder (other than OSAS for the patient groups), pulmonary disease or intake of drugs that could affect sleep, EEG or daytime vigilance. Since high blood pressure is a common feature in OSAS, only patients with unstable high blood pressure were excluded (four OSAS patients who were well controlled with anti-hypertensive drugs were included). A Beck Depression Inventory (BDI) (Beck et al., 1998) score ≥ 19 and the DSM-IV-TR (2000) criteria were used to rule out depression. All participants were informed of the research purposes and gave written consent before entering the study, which was approved by the Sacre-Coeur Hospital Ethics Committee.

Upon arrival at the sleep laboratory, all participants were measured anthropometrically (body mass index (BMI) and neck circumference) and completed questionnaires (BDI and Epworth Sleepiness Scale (ESS), Johns, 1991).

2.2. Polysomnography recording

Participants underwent a standard all-night polysomnographic evaluation (PSG). Surface EEG electrodes were positioned according to the international 10–20 system. A Grass polygraph Model 15A54 amplifier system (amplifier gain: 7.5 μV/mm, bandpass: 0.3–100 Hz) was used for recording and all signals were digitized at a sampling rate of 256 Hz using commercial software (Harmonie 6.0, Stellate Systems, Canada).

Sleep was recorded and scored according to the standard method using 20-s epochs (Rechtschaffen and Kales, 1968). Sleep variables studied were total sleep time, sleep efficiency, micro-arousal index, and percentage of stage 1 sleep, stage 2 sleep, slow-wave sleep (SWS: stages 3 and 4 of non-REM sleep) and REM sleep. Thoraco-abdominal plethysmograph and oronasal canula were used to monitor respiration and transcutaneous finger pulse oximeter was used to measure oxygen saturation. An apnea was defined as a total cessation of airflow and a hypopnea as a reduction of airflow of at least 50% lasting 10 s or more (American Academy of Sleep Medicine Task Force, 1999). The sum of apneas and hypopneas, divided by the number of hours of sleep, was referred to as the AHI. The nocturnal hypoxemia variables studied were mean oxygen saturation (SaO₂) and time spent with SaO₂ below 90%.

2.3. Waking EEG

We used qEEG to assess potential cortical dysfunction of OSAS patients; this method has been successfully used to detect cortical dysfunction associated with vascular and neurodegenerative processes (Coben et al., 1990; Petit et al., 1992, 1993; Helkala et al., 1996; Szelies et al., 1994). To avoid the sleep inertia period and to focus on cortical functioning, we recorded waking EEG (10 min/eyes closed) 30 min after morning awakening. Subjects were periodi-
cally asked to open their eyes to prevent drowsiness, or when slow rolling eye movements (sign of sleepiness) were present. EEG power spectra were determined for 4-s epochs using Fast Fourier Transform. A total of 96 s of artifact-free EEG samples were evaluated at a resolution of 0.25 Hz with cosine tapering. Four frequency bands were analyzed: delta (0.75–3.75 Hz), theta (4.00–7.75 Hz), alpha (8.00–12.75 Hz), and beta (13.00–20.25 Hz). As a global index of cortical slowing, a ratio of slow (delta + theta) to fast (alpha + beta) frequencies was calculated [(delta + theta)/(alpha + beta)] for each electrode and averaged to obtain a single value per cortical region: frontal (FP1, FP2, F3, F4, F7, and F8), central (C3, C4), parietal (P3, P4), temporal (T3, T4, T5, and T6) and occipital (O1, O2). This ratio has proven sensitive in previous apnea research (Morisson et al., 1998, 2001).

2.4. Four choice reaction time test

The four choice reaction time test (FCRTT) is known to be sensitive to attention decrement (Wilkinson and Houghton, 1975; Glenville and Wilkinson, 1979). It consists of a small recording apparatus with four lights arranged in a square and four correspondingly arranged keys. When one of the lights flashes, participants are asked to press the corresponding key as quickly and accurately as possible. The interval between the response and the next stimulus is 120 ms for 10-min task duration. Variables assessed were mean reaction time, number of lapses as defined by a reaction time >1 s and number of errors. The FCRTT trials were administered throughout the day (09:30, 11:30, 13:30, and 15:30), and scores on all trials were averaged. Three practice trials were administered before the PSG in order to stabilize performance according to the standard method (Glenville and Wilkinson, 1979). Subjects were seated in a dimly lit room to perform the FCRTT trials.

2.5. Statistical analyses

STATISTICA 6.0 was used for all statistical analyses. Tests for normality of distribution and equality of variance were performed on all measures. All differences in variables were analyzed by two-way analyses of variance (ANOVARs) with Group (controls and OSAS patients) and Age (younger and older) as factors. Since the FCRTT variables were not normally distributed, log-transformations were used. Post hoc comparisons were conducted using planned comparisons. Relationships between respiratory (AHI and time spent with SaO₂ below 90%), sleep fragmentation (sleep efficiency and micro-arousal index) and obesity variables (BMI and neck circumference) on the one hand, and waking EEG (ratio calculated for each cortical region) and attention variables (MRT and lapses on the FCRTT) on the other hand, were analyzed across the entire OSAS group. For all tests, significance was established at p < 0.05.
3. Results

3.1. Subject characteristics

As presented in Table 1, main Group effects were found for obesity and clinical variables: OSAS patients were overweight ($F_{1,48} = 24.00; p < 0.0001$), had larger neck circumference ($F_{1,45} = 7.04; p < 0.01$) and were sleepier ($F_{1,47} = 21.56; p < 0.0001$) than normal controls. They also showed higher BDI scores ($F_{1,45} = 5.26; p < 0.05$) although none of the patients was clinically depressed based on the DSM-IV-TR (2000) criteria and clinical interview. However, there was no Age effect: older individuals did not differ physically or clinically from younger individuals.

In addition, main Group effects were found for all respiratory variables. Compared to controls, OSAS patients had higher AHI ($44.8 \pm 22.3$ vs $1.5 \pm 1.6; F_{1,48} = 94.67; p < 0.0001$), higher AI ($F_{1,48} = 74.2; p < 0.0001$), lower mean SaO$_2$ ($93.9 \pm 2.8$ vs $96.2 \pm 1.4; F_{1,48} = 13.75; p < 0.0001$) and spent more time with SaO$_2$ below 90% ($27.2 \pm 33.0$ vs $0.6 \pm 2.5; F_{1,48} = 18.43; p < 0.0001$). However, no significant differences were found between younger and older OSAS patients on severity of respiratory variables; both subgroups can be considered mild to moderate OSAS patients.

Finally, main Group effects were also found for sleep architecture: OSAS patients had significantly more stage 1 sleep ($F_{1,48} = 11.73; p < 0.001$) and micro-arousals ($F_{1,48} = 17.82; p < 0.0001$), lower sleep efficiency ($F_{1,48} = 17.82; p < 0.01$) and less REM sleep ($F_{1,48} = 21.56; p < 0.0001$) compared to controls. In addition, there were main Age effects with lower total sleep time ($F_{1,48} = 7.52; p < 0.01$), lower sleep efficiency ($F_{1,47} = 7.70; p < 0.01$) and lower percent of SWS in older subjects of both groups ($F_{1,47} = 26.72; p < 0.0001$) compared to younger individuals. No Group by Age interaction was found for any of the variables measured.

3.2. Waking qEEG

Table 2 presents results of the slow-to-fast activity ratio in five cortical regions during wakefulness in OSAS patients and controls. Main Group effects were observed with higher ratios for all regions in OSAS patients: frontal ($F_{1,48} = 4.26; p = 0.04$), central ($F_{1,48} = 4.42; p = 0.04$), parietal (0.7 ± 0.1 vs 0.5 ± 0.2; $F_{1,48} = 4.92; p = 0.03$), temporal ($F_{1,48} = 5.66; p = 0.02$) and occipital ($F_{1,48} = 5.70; p = 0.02$). No Age effect or Group by Age interaction was found for the qEEG during wakefulness.

3.3. Sustained attention

A main Group effect was observed for the number of lapses (reaction times > 1 s) ($F_{1,46} = 4.52; p = 0.03$); patients with OSAS having more than controls. OSAS patients also showed slower reaction times although the between-group difference did not reach significance (main Group effect: $F_{1,46} = 3.57; p = 0.06$). Main Age effects were also observed for mean reaction time ($F_{1,46} = 21.90; p < 0.0001$) and lapses ($F_{1,46} = 16.00; p < 0.001$), with older subjects slower than younger subjects. No Group by Age interaction was found for the FCRTT measures.

3.4. Relationships between variables

Relationships between respiratory impairment, sleep fragmentation and obesity, on the one hand, and attention and waking EEG variables on the other hand, were analyzed for the entire OSAS group. Results indicated a relationship between micro-arousal index and EEG ratio across all cortical regions (all $r$ between +0.41 and +0.48; all $p < 0.05$) and a correlation between AHI and ratio for the parietal region ($r = +0.44; p = 0.028$); with trends for frontal, central, temporal and occipital regions (all $r$ between +0.35 and +0.36; $p$ between 0.078 and 0.091).

---

Table 2

Results of the spectral ratio calculated in cortical regions and of FCRTT (mean and standard deviation)

<table>
<thead>
<tr>
<th></th>
<th>OSAS (n = 12)</th>
<th>Controls (n = 13)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ratio</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal</td>
<td>2.0 (1.9)</td>
<td>2.4 (2.1)</td>
<td>1.4 (0.4)</td>
</tr>
<tr>
<td>Central</td>
<td>1.5 (1.0)</td>
<td>1.7 (1.6)</td>
<td>1.1 (0.6)</td>
</tr>
<tr>
<td>Parietal</td>
<td>0.7 (0.3)</td>
<td>0.7 (0.4)</td>
<td>0.5 (0.2)</td>
</tr>
<tr>
<td>Temporal</td>
<td>1.0 (0.7)</td>
<td>1.4 (1.3)</td>
<td>0.8 (0.4)</td>
</tr>
<tr>
<td>Occipital</td>
<td>0.7 (0.6)</td>
<td>1.3 (1.4)</td>
<td>0.5 (0.4)</td>
</tr>
<tr>
<td>FCRTT</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean reaction time</td>
<td>489.2 (84.0)</td>
<td>669.6 (165.0)</td>
<td>474.9 (77.4)</td>
</tr>
<tr>
<td>Lapses</td>
<td>16.9 (16.9)</td>
<td>61.2 (42.0)</td>
<td>12.9 (11.0)</td>
</tr>
</tbody>
</table>

Ratio, cortical slowing calculated on average spectral bands: (delta + theta)/(alpha + beta); FCRTT, four choice reaction time test (results are expressed in their original units).

$^*$ Main Group effect $p < 0.05$.

$^\dagger$ Main Age effect $p < 0.05$. 

---

No relationship was found between obesity and either attention or EEG variables.

4. Discussion

Patients studied were typical OSAS cases. They were overweight, had enlarged neck circumference, disturbed sleep architecture, frequent awakenings, pathological somnolence, and more numerous apneas and hypopneas compared to controls.

Waking EEG was slower in all cortical regions in OSAS patients and, daytime vigilance performance was affected by both condition and age, although our results failed to demonstrate Group by Age interaction for any of the variables measured.

4.1. Waking quantitative EEG

EEG spectral analysis showed a slowing of the EEG across all cortical regions in OSAS patients compared to controls. This is most likely caused by nocturnal respiratory perturbation rather than daytime somnolence since EEG changes found in OSAS patients differ from those previously reported in conditions of reduced vigilance. In healthy individuals, drowsiness manifests itself by increased theta activity, more prominently in the central region, and decreased alpha activity in the occipital regions (Hori, 1985; Broughton and Hasan, 1995). Moreover, in the present study, all participants were asked to open their eyes periodically during waking EEG recording to avoid drowsiness, and the electrooculogram was carefully monitored to rule out the presence of slow rolling eye movements, a physiological sign of increased sleepiness. Our findings are consistent with a previous study that showed EEG slowing in all cortical regions in moderate to severe OSAS patients (Morisson et al., 1998). Our results are also in agreement with a morphometry study that showed diffuse grey matter loss in several cortical and subcortical regions, the frontal and parietal lobes and the anterior cingulated gyrus, hippocampus and cerebellum in OSAS patients (Macey et al., 2002). An fMRI study also reported widespread reduced activation in the anterior cingulate, frontal (dorsolateral) and posterior parietal cortices in conjunction with reduced performance on a working memory task in OSAS patients compared to controls (Thomas et al., 2005).

On the other hand, we found no Age effect on the qEEG. This is a controversial issue; previous studies have also noted very little and/or no age-related changes in EEG activity (Dustman et al., 1985; Williamson et al., 1990; Brenner et al., 1995) although others have found a lower dominant occipital frequency (Matejeck, 1980; Torres et al., 1983) and increased theta and delta power with advancing age (Matejeck, 1980; Buysse, 1983; Breslau et al., 1989). One possibility is that the older individuals in our study were too young to exhibit such cortical slowing or that the ratio used was insufficiently sensitive to detect possible changes in that age cohort. We recently demonstrated attention decline, executive dysfunction, memory loss and procedural deficits in older individuals for a similar age cohort (Mathieu et al., 2007), suggesting that cortical slowing could appear later than most age-dependent cognitive declines.

4.2. Four choice reaction time test

Patients with OSAS showed a perturbation of sustained attention reflected by more lapses and a trend toward longer mean reaction times on the FCRTT compared to controls. However, OSAS patients made no more errors than controls. Our findings are in agreement with those reported in a group of mild to moderate OSAS patients (Mathieu et al., 2007) and more severe patients (Bédard et al., 1991). Mean reaction time and number of lapses are more closely related to the ability to stay awake and sustain attention than are errors. An attention deficit in OSAS has been well demonstrated by neuropsychological testing (Verstraeten et al., 2004; Mazza et al., 2005; Mathieu et al., 2007) and by two recent event-related potential studies showing alterations in involuntary attention switching capacities (Gosselin et al., 2006a,b) and in stimulus classification processing (Gosselin et al., 2006b). Thus, OSAS patients exhibit attention deficits across a broad range of attentional processes (Mazza et al., 2005).

Attention decline has also been seen in older compared to younger individuals in the present study. Elderly individuals are known to present sustained and divided attention deficits (see Kallus et al., 2005 for a review) and motor impairments (Crossley and Hiscock, 1992). Thus, one study observed a decreased daytime performance in older compared to younger individuals following sleep deprivation controls. The authors concluded that this was mainly explained by age per se rather than other factors (Buysse et al., 2005). However, despite the presence of both Group and Age effects, no interaction was found. Is there a physiological habituation to sleep fragmentation or to nocturnal hypoxemia with time or a compensation mechanism that prevents a greater interaction between OSAS and age?

4.3. Explorations of relationship between cortical slowing and sleep/somnolence

Two relationships were found: (1) between EEG slowing ratio in all regions and micro-arousal index and (2) between EEG ratio of the parietal region and AHI. The diffuse cortical slowing is probably a manifestation of some neuronal events triggered by sleep fragmentation (indexed by micro-arousal) rather than a reflection of daytime sleepiness per se. Two studies have shown that EEG slowing in wakefulness is not associated with either objective sleepiness (measured by the MSLT: Morisson et al., 1998) or subjective sleepiness (measured by the ESS: Sforza et al., 2002). On the other hand, a recent ERP study found decreased cortex reactivity to attention-involving tasks...
accompanied by higher micro-arousal indices and more sleep transitions in OSAS patients compared to controls (Gosselin et al., 2006b). Finally, Bédard and coworkers (1991) reported that decreased alertness (measured by the FCRTT), but not sleepiness, was correlated with nocturnal sleep disturbance. Nevertheless, although our OSAS patients were not greatly hypoxemic, we cannot exclude the possibility that recurrent nocturnal hypoxemia may have played a role in this relationship. Morisson and colleagues (1998) reported a modest positive correlation between slowing ratio in all regions pooled and time spent with oxygen saturation below 90% in OSAS patients. In Morisson’s study, patients as a whole spent more time with oxygen saturation below 90% than our patients, but with a larger inter-subject variation. Since our OSAS patients showed much less variability on this parameter, a relationship with an EEG variable was probably more difficult to demonstrate. The fact remains that micro-arousal and nocturnal recurrent hypoxemia are concomitant events (Beebe and Gonzal, 2002).

The relationship between cortical slowing ratio particularly in the parietal region, and AHI can be interpreted as an expression of the attention decline brought on by the severity of the OSAS. The superior parietal region (Brodmann areas 5 and 7) is known to be implicated in attentional visual processing (spatial processing and selection) and orientation whereas the inferior parietal region (Brodmann areas 21 and 23) is more involved in visual pattern recognition speed. As mentioned earlier, in OSAS, grey matter loss has been measured in the parietal and anterior cingulate cortices (Macey et al., 2002) with reduced activation in anterior cingulate (Thomas et al., 2005). These two brain regions have been identified as related to attentional processes (see Crottaz-Herbette, 2001; for review).

Finally, our results suggest that OSAS worsens brain functioning compared to controls, consistent with previous studies (Morisson et al., 1998; Ayalon et al., 2006). Although no Group by Age interaction was measured, we cannot exclude the possibility that aging could negatively affect the patient’s condition. Structural changes (a general reduction in brain volume and neuron size) and functional changes (decreased oxygen use and cerebral blood flow with the greatest reduction in the frontal lobe and less consistency for temporal and parietal regions) accompany normal aging (see West, 1996 for a review) and could potentially play a greater role as OSAS progresses. In fact, our vigilance results showed both Group and Age effects, with older OSAS patients performing the worst.

5. Limitations and conclusion

Our study directly measured the relative impact of age on OSAS, by comparing younger and older OSAS patients. However, we did not take into account disease duration, which is difficult to determine precisely. Furthermore, we did not distinguish between the relative contributions of nocturnal hypoxemia and sleep fragmentation to cortical slowing observed in OSAS patients. Our patients were not severely hypoxemic. The study may not have had enough statistical power to detect the unexpectedly subtle changes due to the interaction between the OSAS and aging. The study’s strength is that younger and older OSAS patients were well matched, allowing the confounding effects of BMI, EDS and depression to be eliminated.

Acknowledgements

This research was supported by the Canadian Institutes of Health Research (grant to J.M.) and ANTaDIR (studentship to S.M.). The authors are grateful to Jean Paquet, PhD, for statistical advice, Sylvie Rompré, for assistance in EEG scoring and analysis, Nadia Gosselin for a manuscript review, Mireille Charron for patient recruitment assistance and, Benoit Adam for assistance throughout the entire protocol.

References

Davis RJ, Stradling JR. The epidemiology of sleep apnea. Thorax 1996;51:865–70.