Attentional deficits in patients with obstructive sleep apnea syndrome: An event-related potential study

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Abstract

Objective: Patients with obstructive sleep apnea syndrome (OSAS) show cognitive deficits, vigilance alteration and attentional decline. The aim of this study was to use event-related potentials (ERP) to further document the attentional impairments in these patients.

Methods: Twelve OSAS patients and 12 age-matched controls underwent the ERP task which consisted in the presentation of short (50 ms, 50%) and long tones (400 ms, 50%). For these two categories, 90% were standard (1000 Hz) and 10% were deviant tones (750 or 1250 Hz). Subjects had to discriminate short and long tones by a motor response.

Results: OSAS patients had a sustained and delayed P300 in comparison with control subjects following standard tones ($p < 0.05$). A reduction in amplitude was found in OSAS patients for the P3a obtained by the subtraction of standard from deviant tones ($p < 0.05$). No group difference was observed for N1, mismatch negativity and reorienting negativity components.

Conclusions: Apneas and hypopneas produce deficits related to involuntary attentional switch and stimulus classification processing.

Significance: The changes observed in P3a and P300 components further support the hypothesis that attentional deficits play a pivotal role in cognitive deficits noted in OSAS.

Keywords: Sleep apnea; Event-related potentials; Attention; Cognition; P300; P3a

1. Introduction

Obstructive sleep apnea syndrome (OSAS) is a sleep-related breathing disorder, characterized by repetitive pauses (apneas) or reductions in airflow amplitude (hypopneas) attributed to the collapsibility of the upper airway during sleep. The severity of the respiratory events is measured by the frequency of apneas and hypopneas per hour of sleep (apnea hypopnea index – AHI). An apnea index greater than 5 or an AHI greater than 10 are generally considered abnormal (American Academy of Sleep Medicine Task Force, 1999). OSAS is a common sleep problem, affecting approximately 2–14% of adults (for review, see Young et al., 2002). Epidemiological studies show an increasing prevalence with age for the sixth and seventh decades of life (Ancoli-Israel et al., 1991; Young et al., 2002).

The respiratory disturbances found in OSAS include hypoxemia, hypercapnia and increased ventilatory effort against airway occlusion. Apneas and hypopneas are also associated with nocturnal sleep disruption. Patients with OSAS have more EEG arousals during sleep and less stages 3 and 4 non rapid eye movement (NREM) sleep. They also have less REM sleep due to the numerous respiratory events that occur during that sleep stage.

OSAS is associated with diurnal symptoms such as excessive daytime sleepiness and cognitive deficits. Several cognitive functions may be altered in OSAS such as attention, memory, executive cognitive control and motor coordination (Bedard et al., 1991; Ferini-Strambi et al., 2003; Rouleau et al., 2002; Salorio et al., 2002; Verstraeten 1999).
et al., 2004). It has been recently proposed that vigilance and attentional deficits play a pivotal role in all aspects of cognitive deficits noted in OSAS (Mazza et al., 2005; Verstraeten et al., 2004). In fact, these patients showed deficits in almost all attentional processes and these difficulties were reflected not only by the patients’ inability to remain awake in monotonous situations, but also by their problem to treat information even in more stimulating conditions (Mazza et al., 2005).

One method that has been extensively used to assess cognitive and attentional deficits is the measure of event-related potentials (ERP). The most common tasks used in ERP studies is the Oddball paradigm (Picton, 1992). It consists in the presentation of two categories of stimuli (standard and deviant) having different probabilities of occurrence. The participant must generally be attentive to the stimuli and give a manual response when they appear. The appearance of the deviant stimulus generally elicits a P300 wave which is stronger than for the standard stimulus. The amplitude of the P300 component represents the attentional resource allocated in a task (Kramer and Strayer, 1988; Wickens et al., 1983), whereas its latency reflects the stimulus classification speed (Kutas et al., 1977; McCarthy and Donchin, 1981; Polich, 1986).

Few ERP studies have been performed in OSAS patients and most of them used a visual or an auditory Oddball paradigm in order to evaluate the P300 characteristics. However, inconsistent results were reported for P300 amplitude and latency. Most studies noted a prolongation of P300 latency following visual stimuli in OSAS patients (Kotterba et al., 1998; Sangal and Sangal, 1997). An increased P300 latency was also found following auditory stimuli in one study (Rumbach et al., 1991), but not in others (Afifi et al., 2003; Sangal and Sangal, 1997). Two studies found a reduction in P300 amplitude during auditory task in OSAS patients (Rumbach et al., 1991; Walsleben et al., 1989), but other studies did not (Afifi et al., 2003; Sangal and Sangal, 1997). Although P300 obtained in classical Oddball task is known to give a global index of cognitive processes (Picton, 1992), the task may be not sensitive enough to detect attentional dysfunctions in OSAS. Moreover, few studies investigated earlier ERP components, which are crucial in the comprehension of the cognitive deficits observed in OSAS patients. In fact, deficits occurring in early stages of information processing may cause the late attention-related P300 anomalies in OSAS patients.

Based on the Oddball paradigm, Schröger and Wolff (1998) have recently created a new paradigm which elicits ERP components known to reflect automatic stimulus detection, involuntary and volitional attentional processes. In this paradigm, deviant tones are presented in a sequence of standard tones without informing the subject. As in the classical Oddball paradigm, standard and deviant stimuli elicited N1 and P300 waves where N1 is mainly determined by physical features of stimulus (such as loudness, duration or pitch) and thus referred to sensory processes (Naätänen and Picton, 1987). The subtraction of the standard stimulus waveform from the deviant one represents a good measure of the specific brain response to deviant stimulus. In this case, a mismatch negativity (MMN), a P3a component and a reorienting negativity (RON) are observed. MMN is elicited when a deviant tone appeared in a sequence of repeated tones and represents stimulus change detection (Alho et al., 1986; Naätänen et al., 1993). P3a represents an involuntary attention switch to deviant stimulus (Squires et al., 1977). Finally, RON is a late negative component with maximal amplitude in fronto-central regions and it has been hypothesized by Schröger and Wolff (1998) that this component reflects reorientation of attention back to the main task after the processing of an unexpected tone deviance.

The aim of the present study was to measure ERP components in order to clarify cognitive and, more specifically, attentional deficits in OSAS patients. We hypothesized that cognitive functions required to perform the task, i.e., detection and classification of stimuli, orientation of attention towards unexpected events and reorientation of attention back to the main task, are impaired in OSAS patients and will result in reduced amplitudes and prolonged latencies of late ERP components.

2. Methods

2.1. Subjects

The study group consisted of 12 OSAS patients recruited from the Sleep Disorder Center of the Sacré-Cœur Hospital in Montreal, Canada. Only patients with an obstructive sleep apnea index higher than 10 were included in this study.

Exclusion criteria were the presence of any neurological or psychiatric diseases (including depression), a pulmonary disease, the use of drugs known to affect sleep or daytime sleepiness such as antidepressants, hypnotics and benzodiazepines. Since high blood pressure (HBP) is a common feature in OSAS patients, only patients with unstable HBP were excluded (two OSAS patients who were well controlled with anti-hypertensive drugs were included).

Results obtained in patients with OSAS were compared to those of 12 normal controls matched for age, gender and level of education (see Table 1). Normal controls were recruited from newspapers in the Montreal area. They had no history or sleep laboratory evidence of sleep apnea (defined as an AHl greater than 5). Other exclusion criteria were the same as for OSAS patients. All subjects completed the Epworth Sleepiness Scale (ESS) (Johns, 1991) and an auditory examination made by an audiologist. None of the subjects included in the present study showed any auditory deficit in the frequency bands used in the ERP paradigm.

Each participant was informed of the research protocol and gave written informed consent before the beginning of the study. The protocol was approved by a University-Hospital ethics committee.
2.2. Polysomnographic recording

All subjects underwent one night of polysomnographic recording in the sleep laboratory. Polysomnography was performed using four EEG derivations (C3, C4, O1, and O2) referred to linked earlobes. Right and left electrooculogram (EOG) and chin electromyogram (EMG) were also recorded. Sleep stages were scored according to the standard method developed by Rechtschaffen and Kales (1968). Thoracoabdominal plethysmograph and oral/nasal canula were used to monitor respiration and transcutaneous finger pulse oximeter was used to measure oxygen saturation. Apneas were defined as a total cessation of airflow lasting for at least 10 s and hypopneas were scored as a reduction of airflow of at least 50% from baseline, lasting at least 10 s (American Sleep Disorder Association, 1992).

2.3. Stimuli and procedures

All subjects were tested between 8:00 and 9:30 AM using an auditory paradigm. We presented a dichotic sequence of short (50 ms) and long tones (400 ms). Each type of stimuli appeared in 50% of the trials. For these two categories, 90% of the stimuli were 1000 Hz tone (standard), 5% were 750 Hz tone (deviant) and 5% were 1250 Hz (deviant). The inter-stimulus interval was 2 s and the tone intensity was 70 dB SPL. A total of 480 stimuli were presented. Subjects had to distinguish short from long tones by pressing two different buttons with their index fingers. The task had a duration of 20 min and was divided in two blocks. Constant EEG and visual monitoring was done in order to ensure that subjects were not transiently falling asleep during the task.

2.4. ERP recordings

The EEG was recorded from 23 electrodes (Fz, F3, F4, F7, F8, FCz, FC3, FC4, Cz, C3, C4, T7, T8, TP7, TP8, Pz, P3, P4, P7, P8, Oz, O1 and O2) placed according to the guideline for standard electrode position (Electrode Position Committee, 1991). All electrodes were referred to the nose, with a forehead ground and the impedance was kept below 5 kΩ. Horizontal and vertical EOG were recorded. The filter band-pass was 0.01–40 Hz. The EEG was digitized at 256 Hz with a 100 ms prestimulus baseline. Stimulus presentation and EEG recording were done with Neuroscan system (Neurosoft, Inc. Sterling, USA).

2.5. Data analysis

Reaction times (RT) are reported relative to the point in time where the short tone had or would have had (in the case of long tones) its offset (i.e., 50 ms after the stimulus onset). Only successful trials were submitted to analysis and trials on which either the EEG or EOG artifacts exceeded ±75 μV were rejected automatically. EOG artifact correction was made using Neuroscan software. The EEG was averaged time-locked to the stimulus for each type of stimuli: standard stimuli (short or long tones of 1000 Hz) and deviant stimuli (short or long tones of 750 and 1250 Hz). Additional filters were applied on average ERP (0.01–20 Hz, 12 dB/octave). Amplitude was measured relative to the mean of the prestimulus baseline. Latency was defined as the maximum positive or negative amplitude within the latency window on pre-defined electrodes for each ERP component.

2.6. ERP component definitions

Latency and amplitude of the N1 component were measured on the maximal negative peak within the latency window of 100–200 ms and were analyzed on three electrodes (Fz, FCz and Cz). The P300 component was defined as the largest positive peak within 300–700 ms and was measured on Fz, FCz, Cz, Pz, and Oz for standard stimuli. P300 was not analyzed for deviant stimuli, since it may be confounded with the P3a due to their common latency range. In our subjects, this component spreads on a large amount of time and peak analysis did not reflect the P300 dynamic. The mean amplitude of this component was thus measured in two different latency windows: 300–500 ms and 500–700 ms. MMN, P3a and RON were obtained by subtracting the standard from the deviant averaged waveforms.

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Table 1

<table>
<thead>
<tr>
<th>Variables</th>
<th>OSAS patients</th>
<th>Control subjects</th>
<th>t-value (df = 22)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of subjects</td>
<td>12 (9 m ; 3w)</td>
<td>12 (11 m ; 1w)</td>
<td>na</td>
<td>na</td>
</tr>
<tr>
<td>Age (years)</td>
<td>47.9 ± 13.7</td>
<td>44.4 ± 9.5</td>
<td>0.62</td>
<td>ns</td>
</tr>
<tr>
<td>Education (years)</td>
<td>12.5 ± 3.3</td>
<td>14.1 ± 2.8</td>
<td>-1.4</td>
<td>ns</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>31.0 ± 5.8</td>
<td>23.1 ± 2.3</td>
<td>4.2</td>
<td>***</td>
</tr>
<tr>
<td>Epworth Sleepiness Scale score</td>
<td>15.1 ± 6.7</td>
<td>5.6 ± 2.9</td>
<td>4.5</td>
<td>**</td>
</tr>
<tr>
<td>Mean O₂ saturation (%)</td>
<td>92.5 ± 4.5</td>
<td>96.8 ± 0.8</td>
<td>-3.3</td>
<td>*</td>
</tr>
<tr>
<td>Time spent with O₂ &lt; 90% (min)</td>
<td>96.5 ± 120.3</td>
<td>0.0 ± 0.0</td>
<td>2.8</td>
<td>***</td>
</tr>
<tr>
<td>Apnea/hypopnea index</td>
<td>51.2 ± 23.9</td>
<td>1.2 ± 1.4</td>
<td>7.2</td>
<td>***</td>
</tr>
<tr>
<td>Obstructive apnea index</td>
<td>31.3 ± 17.0</td>
<td>0.6 ± 1.0</td>
<td>6.2</td>
<td>***</td>
</tr>
</tbody>
</table>

na, non applicable; ns, non significant; BMI, Body mass index.

* p < 0.01.
** p < 0.001.
*** p < 0.0001.

Latencies and amplitudes of the highest negative peak were measured in the 100–250 ms latency window for MMN on three electrodes (Fz, FCz and Cz). Inversion of polarity in temporal electrodes served for the identification of MMN. P3a was defined as the highest positive peak in the 300–500 ms latency window also on Fz, FCz and Cz, whereas RON was the highest negative peak in the 400–600 ms latency window on the same three electrodes.

2.7. Statistical analyses

Demographic, clinical characteristics and accuracy in classifying short and long tones in OSAS patients and control subjects were compared using Student t-tests. Group differences in RT were analyzed with three-way analyses of variance (ANOVAs) with one independent measure (Control/OSAS: GROUP) and two repeated measures (Standard/Deviant: STIMULUS; Short/Long: DURATION). We used two-way ANOVAs (GROUP by ELECTRODE) to analyze latencies and amplitudes for the N1, MMN, P3a and RON components. P300 latency was also analyzed with two-way ANOVAs (GROUP by ELECTRODE), whereas P300 mean amplitude was analyzed with three-way ANOVAs with one factor (GROUP) and two repeated measures (ELECTRODE; LATENCY WINDOW). A Greenhouse-Geisser correction for sphericity was applied to all repeated measures. When ANOVAs showed significant main effects, Tukey HSD tests were used for post hoc comparisons. Pearson correlation coefficients were used to measure the relationships between demographic data, RT and ERP characteristics. Since there were multiple analyses, correlations were considered significant at p < 0.01. All results are reported as mean ± standard deviation.

3. Results

3.1. Subject characteristics

Demographic and clinical characteristics are presented in Table 1. No between-group difference was found for age or education level. However, a significant difference was found for body mass index (BMI) where OSAS patients showed a higher BMI than control subjects. Significant group differences were also observed on all respiratory variables. OSAS patients showed higher scores than control subjects on the ESS which represents higher daytime drowsiness in OSAS patients than in control subjects. All control subjects had an ESS score lower than 10, which is generally considered as normal (Johns, 1991), whereas nine OSAS patients had a score higher than 10. No significant correlation was found between age and clinical characteristics.

3.2. Behavioral results

RTs could not be analyzed in two subjects (one control and one OSAS subjects) due to technical problems. Fig. 1 shows RT in OSAS and control groups for short and long tones and for standard and deviant stimuli. A STIMULUS effect was found for RT (F(1,20) = 82.79, p < 0.0001) characterized by shorter RT for standard than for deviant stimuli. A DURATION effect was also observed (F(1,20) = 29.80, p < 0.0001) where subjects had longer RT for long tones in comparison with short ones. No between-group difference was found for the number of errors, the number of omissions and RT.

3.3. Event-related potentials

Fig. 2 shows average ERP waveforms for deviant stimuli in all individual subjects and Fig. 3 presents the grand-average waveforms for standard (a) and deviant (b) stimuli in OSAS and control groups separately. The deviant minus standard waveform is also presented (c). A clear N1 was observed for each subject for frontal, fronto-central and central electrodes following standard and deviant stimuli. A large P300 was also elicited in both groups for standard stimuli. Standard from deviant stimulus subtraction highlighted a MMN and a RON in frontal and central regions and a P3a which can be observed in all electrodes, but more
prominent in anterior regions. No correlation was found between demographic data (including BMI), respiratory variables, RT and ERP characteristics, except a significant relationship between the behavioural distraction effect for long tones and the sleep efficacy in patients with OSAS ($r = -0.74, p < 0.01$) where higher sleep efficiency is related to a lower distraction effect.

### 3.3.1. Components observed following standard stimuli

Patients with OSAS showed a large and sustained P300 wave which starts at 300 ms and continues until 700 ms (see Fig. 3a). A different dynamic was observed in control subjects, where the P300 wave was sharper and returned to the baseline level earlier than what was observed in patients with OSAS. A significant GROUP by WINDOW by ELECTRODE interaction was observed for P300 ampli-

![Fig. 2. Average ERP waveforms on FCz for deviant stimuli for all individual subjects.](image)

![Fig. 3. Grand-average ERP waveforms in OSAS patients (dark line) and control subjects (grey line) for standard stimuli (a), deviant stimuli (b) and deviant minus standard subtraction (c).](image)
tude \((F(4,88) = 5.37, p < 0.01, \varepsilon = 0.51)\). Planned comparisons showed significant GROUP by WINDOW interactions for \(Fz\), FCz, Cz and \(Pz\) \((p < 0.01\) in all cases) and patients with OSAS had a higher P300 amplitude than control subjects in the 500–700 ms window (significant group difference for \(Pz\), \(p = 0.03\), and trends for \(Fz\) and \(Cz\), all \(p\)-values \(< 0.09\), but not in the 300–500 ms window. A GROUP difference was also observed for P300 latency \((F(1,22) = 6.07, p < 0.05)\); control subjects had a higher P300 maximal peak amplitude than patients with OSAS. No between-group difference was observed for N1 amplitude and latency.

3.3.2. Components resulting from the deviant minus standard subtraction

3.3.2.1. MMN. MMN showed a similar dynamic in OSAS and in control subjects (see Fig. 3c) and this observation was confirmed by statistical analysis where no significant group difference was found for MMN amplitude or latency.

3.3.2.2. P3a. A significant group difference was observed for P3a amplitude where the control group showed higher amplitude than did the OSAS group \((F(1,22) = 4.47, p < 0.05)\). In addition to the reduced amplitude, patients with OSAS tended to have a prolonged P3a latency in comparison with control subjects \((F(1,22) = 4.03, p = 0.057)\) as we can observed in Fig. 3c.

3.3.2.3. RON. No group difference was observed for RON amplitude. Since nearly no RON component was observed in OSAS patients, peak analysis was difficult to perform in this group. We therefore decided to make window analyses where mean amplitude was computed in specific latency ranges (early window: 420–499 ms; late window: 500–579 ms). Again, no group difference in mean amplitude was found for early and late RON windows. No group difference was found for RON latency.

4. Discussion

The aim of the present study was to measure ERP components reflecting automatic stimulus detection, involuntary and volitional attentional processes in order to clarify cognitive deficits in OSAS patients. The major finding is that abnormalities in P300 and P3a components were found in patients with OSAS, whereas no anomalies were observed for earlier ERP components.

4.1. Event-related potentials

Following standard stimulus presentation, a wide and prolonged P300 was observed in patients with OSAS in comparison with control subjects. A delayed latency of P300 has already been reported in previous studies (Kotterba et al., 1998; Sangal and Sangal, 1997; Rumbach et al., 1991). In these studies, P300 was measured only on deviant stimuli and thus, represents the stimulus classification speed when an infrequent stimulus occurred. However, in the present study, the prolonged P300 latency found for standard stimuli in OSAS reflects a delay in the processing of habitual and repetitive target stimuli. The increased P300 amplitude found in OSAS patients in the 500–700 ms latency window is explained by the fact that P300 rapidly returned to the baseline level after the maximal peak amplitude, whereas, in OSAS patients, the P300 component remained at a high amplitude for an extended period of time. Overall, these results suggest that patients with OSAS allowed sufficient attentional resources to treat target stimuli, but these attentional resources are delayed and persisted longer than in control subjects.

When an unexpected tone deviance occurs, OSAS patients showed a reduction in the amplitude of the P3a component. This component reflects the specific orientation of attention toward the deviant stimulus and its reduction in OSAS most likely results from decreased attentional resources allocated to analyze the unexpected tones. The change in P3a amplitude and the trend observed in the prolongation of the P3a latency may be explained by an alteration in P3a generators. Several studies using intracranial recordings, magnetoencephalography, analysis of scalp current density and functional neuroimaging, investigated the P3a sources. Strong prefrontal generators have been observed (Bledowski et al., 2004), but the hippocampus, cingulate, auditory and parietal cortices seem to contribute to the P3a production (Alho et al., 1986; Halgren et al., 1995, 1998; Yago et al., 2003). Brain imaging studies performed in OSAS patients showed a functional disturbance of the lateral prefrontal cortex (Thomas et al., 2005), and a loss of gray matter in various brain regions including the frontal regions (Macey et al., 2002). In parallel, neuropsychological studies showed cognitive deficits in OSAS patients that are probably due to frontal lobe dysfunction (see Beebe and Gozal, 2002, for review). Therefore, our results are in agreement with those of neuropsychological and of brain imaging studies and suggest that patients with OSAS have deficits that may be related to a frontal lobe dysfunction.

4.2. The role of hypoxemia, sleep fragmentation and daytime sleepiness

There are major controversies regarding the role of recurrent nocturnal hypoxemia, sleep fragmentation and vigilance fluctuation in cognitive deficits of OSAS patients. Most researchers currently believe that both transient nocturnal hypoxemia and sleep disruption contribute to cognitive deficits seen in OSAS patients (Beebe and Gozal, 2002). Prefrontal cortex seems to be particularly sensitive to sleep deprivation (Drummond and Brown, 2001; Durmer and Dingess, 2005) and possibly to recurrent hypoxemias. According to a recent model suggested by Beebe and Gozal (2002) and previous neuropsychological studies (Bedard et al., 1991; Naegele et al., 1995, 1998), an alteration in...
the prefrontal cortex functioning may be responsible for almost all cognitive deficits observed in OSAS. In the present study, patients presented a high number of respiratory events without being severely hypoxemic and they showed ERP abnormalities. P300 and P3a are known to be affected by sleep deprivation (Morris et al., 1992; Salmi et al., 2005) and especially in the frontal region (Gosselin et al., 2005), while MMN seems to be resistant to sleep deprivation (Salmi et al., 2005). Therefore, our results are congruent with those observed following total sleep deprivation, but more studies will be needed to further assess the specific role of hypoxemia on impaired ERP components.

Previous studies found attenuated P300 amplitude in the frontal regions during sleep onset, whereas no amplitude reduction was showed in the parietal region (Bastuji et al., 1995; Cote et al., 2002). In the present study, no reduction in P300 amplitude was found in patients with OSAS, no sleep period was observed on the EEG and no group difference was found in the number of omissions which can reflect transient sleep periods. Moreover, only trials with correct response were considered for ERP analyses. Therefore, the reduction of P3a amplitude in the frontal regions cannot be attributed to transient sleep onset in our OSAS patients. However, we cannot exclude that sleepiness might have played a role in the ERP abnormalities in the OSAS group.

4.3. Pre-attentional versus attentional deficits

Voluntary attentional switching is considered as a late information processing stage and it is influenced by early sensory or automatic processing. Auditory discrimination tasks require that tone characteristics, such as intensity, duration and frequency, be correctly perceived and actively maintained in short-term memory in order to further detect acoustic changes. In the present study, all subjects had a normal auditory examination. Moreover, early and automatic detection of deviance in auditory characteristics, which occurs approximately between 100 and 200 ms following the stimulus presentation, was preserved in OSAS patients, since normal amplitudes and latencies were observed for N1 and MMN. Although these components are known to have a few frontal generators, most of them are found in the auditory temporal cortex (see Alho, 1995 and Escera et al., 2000 for review). Anomalies observed in OSAS patients began at approximately 300 ms, when attentional functions associated to prefrontal cortex are highly involved in the processing of auditory events. We can therefore conclude that only attentional processes are affected in OSAS, whereas automatic detection processing is adequate.

No statistically significant group difference was found for the RON, which may reflect the reorientation of attention to the principal task as suggested by Schröger and Wolff (1998), even though this component was nearly absent in OSAS patients. High inter-subject variability in the RON amplitude and latency may be responsible for the lack of a significant group difference.

4.4. Behavioral performance and information processing speed

When an unexpected, novel or salient sound is introduced in a sequence of repetitive tones, this sound takes high priority in brain information processing. Attention to the ongoing task is suspended in order to investigate the significance of this deviant sound. Behaviourally, a prolongation of RT is then expected following this sound. According to these premises, an attentional switching effect was observed in controls as well as in OSAS patients, characterized by longer RT for deviant than for standard stimuli. However, the RT and the number of correct responses did not allow us to distinguish the two groups. This absence of difference in RT may in part be explained by the design of the task. In fact, most subjects responded after the offset of the 400 ms tone, rather than after the offset of the short tone. In the present study, this strategy increases RT in both OSAS and control subjects. In patients with OSAS, important attentional resources were allocated to stimulus classification, which was reflected by P300 amplitude, and this may explain the normal RT observed in these patients during the task.

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