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Journal search and commentary

Article reviewed: Daytime sleepiness and EEG spectral analysis in apneic patients before and after treatment with continuous positive airway pressure ☆

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Objectives

Previous studies indicate that obstructive sleep apnea syndrome (OSAS) is associated with altered cognitive function and daytime sleepiness. Abnormal executive function and psychomotor tasks have been attributed to hypoxemia and attention/memory deficits have been attributed to sleepiness. The authors of the current paper had previously demonstrated that OSAS patients have EEG slowing during wakefulness over all regions studied and during REM-related apnea over the frontal, central and parietal regions. Of note, executive functions are believed to be dependent on the frontal lobe. The current investigation was intended to assess the impact of continuous positive airway pressure (CPAP) on quantitative (spectral) features of the EEG and to determine if any observed alterations are associated with changes in excessive daytime sleepiness.

Study design

Prospective, controlled trial.

Study population

Fourteen patients with obstructive sleep apnea syndrome (OSAS) (age: 45 ± 6.4 years; BMI: 39.1 ± 5.9; Apnea + Hyponea Index: 62.8 ± 25.8; time spent at $<90\%$ S_aO_2: 209.6 ± 178.1 min; minimum S_aO_2: 61.4 ± 13.4 min; mean $\pm$ SD) and 10 control subjects without OSA (age: 44.2 ± 6.1 years, BMI: 26.1 ± 3.8, Apnea + Hyponea Index 0.6 ± 0.6, time spent at $<90\%$ S_aO_2 0.7 ± 1.5 min's, minimum S_aO_2: 87.7 ± 4.6 min). The OSAS patients had a significantly greater BMI, Apnea + Hypopnea Index, lower minimal S_aO_2 and a greater duration of time slept with S_aO_2 $<90\%$ than the control subjects.

Methods

Control subjects underwent an acclimatization nocturnal polysomnogram (PSG) and on the following day, a multiple sleep latency test (MSLT). On the subsequent night a PSG was performed. Data collected during the second night PSG only were included in the analyses. OSAS patients had pre and post CPAP treatment PSGs and MSLTs with an interval of 6–9 months. Adherence to CPAP therapy was assessed by patient report (mean: 6.5 h/night). In addition to recording parameters of breathing during sleep,

☆ Morisson F, Décary A, Petit D, Lavigne G, Malo J, Montplaisir J., (Chest 2001; 119(1): 45–52).

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the EEG was recorded over the frontal, parietal, central, occipital and temporal regions. Bilateral anterior tibialis EMGs were also recorded.

EEG data were collected during wakefulness in normal subjects as well as pre and post CPAP treatment in the OSAS patients. In addition, EEG data were also obtained during Rapid Eye Movement (REM) sleep. In the pre-treatment OSAS patients, REM EEG was obtained during apnea (because with the exception of three patients, insufficient non-apneic REM sleep was available). In the normal subjects and CPAP treated patients, non-apnea REM sleep EEG was analyzed. The authors state that in three OSAS subjects it was possible to analyze REM EEG in the absence of apnea and the results did not differ from intra-apnea REM EEG.

The EEG data were subjected to spectral analysis and the absolute activity for delta (0.75–3.75 Hz), theta (4–7.75 Hz), alpha (8–12.75 Hz) and beta (13–20.95 Hz) frequencies and the ratio of slow /fast frequencies (delta + theta/alpha + beta, DT/AB) were determined across all regions as well as by region, in normal subjects and in OSAS patients before and after initiation of CPAP therapy.

The relationships between Apnea + Hypopnea Index, time spent at $<90\%$ S_aO_2, minimum S_aO_2, Periodic Limb Movements, mean sleep latency and regional as well as overall DT/AB were assessed by Pearson product-moment correlation for the OSAS patients. The significance of differences between the normal subjects, pre and post-CPAP treated OSAS patients were assessed using parametric or non-parametric tests as appropriate.

Results

In comparison to normal control subjects, before initiation of CPAP therapy, OSAS patients had EEG slowing over the frontal and central regions during both wakefulness and REM sleep. However, the degree of EEG slowing was not related to time spent with $S_aO_2 < 90\%$, the Apnea + Hypopnea Index or the mean sleep latency by MSLT. As expected, CPAP therapy was associated with alleviation of most abnormalities of sleep and breathing. PLMS were decreased although still present. Absolute and relative EEG slowing during wakefulness and REM sleep was reversed after 6–9 months of CPAP therapy. Despite this reversal after initiation of CPAP therapy, sleep latency did not normalize although it was significantly improved.

Conclusion

The investigators concluded that treatment with therapeutically effective CPAP was also associated with reversal of EEG slowing during wakefulness and sleep. There were no correlation between changes in EEG frequency during CPAP therapy and daytime sleepiness or severity of OSAS. In this light, the authors speculated that the persistently abnormal sleep latency after an interval of CPAP therapy may be related to persistent obesity (although no data addressing this hypothesis was presented in the paper, nor was evaluating this indicated as an aim of the study).

Comment

Neurocognitive and psychomotor dysfunction have been increasingly reported in conjunction with patients with OSAS. Assessment of the response to CPAP therapy in these regards has yielded variable results, in part due to small study populations or lack of control groups. The mechanism(s) responsible for neurocognitive and psychomotor dysfunction remain ill-defined and elucidation of this issue may provide insight into outcome risk in OSAS and contribute to guidelines relating to which OSAS patients should be counseled to receive treatment. The investigator's previous observation of EEG slowing over the frontal cortical regions, on which Executive functions are dependent, is of great interest as is the current report of EEG normalization following CPAP therapy. While it is somewhat disappointing that clinical correlates of changing EEG characteristics were not found, it must be noted that the relationship between pre/post-treatment EEG features and neurocognitive or psychomotor function was not explored in this paper. Additionally, the degree to which treatment was effectively applied was not adequately assessed in the absence of objective CPAP adherence data. At this time, EEG slowing over frontal and central regions in OSAS patients

and subsequent reversal with treatment remain intriguing physiologic observations consistent with disease impact on the central nervous system, but without known relation to clinical or health outcome. I am confident we will be learning more about this issue in the future.