Absence of major gray matter volume changes in obstructive sleep apnea

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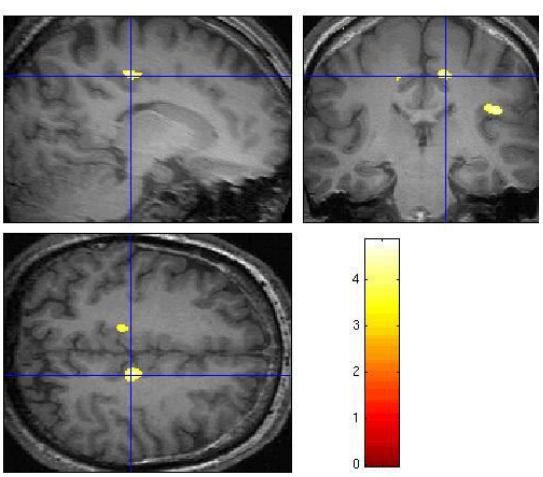
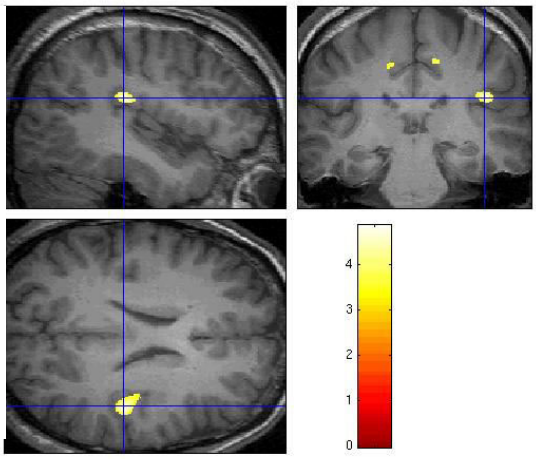
Introduction: Obstructive sleep apnea (OSA) is a sleep disorder associated with intermittent hypoxemia, sleep fragmentation, and impaired neuropsychological function. The current treatment consists of nasal Continuous Positive Airways Pressure (CPAP). Only a few studies have investigated the brain structure of OSA patients. A recent study reported widespread deficits in grey matter volume in OSA patients of varying severity (1). Several of these OSA subjects suffered from co-morbid diseases, such as cardiac failure. The aim of the presented study is to assess changes in brain structure in a homogenous group of newly diagnosed severe OSA patients without co-morbidities, and to investigate any change in brain structure after 6 months of therapy with CPAP.

Methods: We studied 17 male untreated severe OSA patients with AHI > 30 and at least 15% of total sleep time spent at oxygen saturation <90%, and 17 age-matched healthy controls with AHI < 5. Subjects with a history of cerebrovascular or ischaemic heart disease, diabetes mellitus, neurological disorders or substance abuse were not included. OSA patients had a baseline scan after a diagnostic PSG but before commencement of treatment with CPAP. Scans were repeated after 6 months of treatment with CPAP.
A fast spoiled gradient recalled echo at steady state (FSPGR) sequence (TR/TE 8.9/1.9, flip angle 20, matrix size 256x256, FOV 25 x 18.75cm) with contiguous coronal slices of 1.5mm thickness was acquired on a 3T GE scanner. Voxel-based morphometry was performed as previously described (2). A region-of-interest (ROI) analysis assessed the volumes of both temporal lobes and hippocampi, corrected for whole brain volumes (3).



SPM analysis showing gray matter volume decrease in patients compared to controls ($p<0.001$, uncorrected). Significant areas include right insula (left sided panel), bilateral cingulate (right-sided panel).



Results: The ROI analysis showed no difference between patients and controls in the temporal and hippocampal volumes. The VBM analysis, assessed at a threshold of $p<0.001$ (uncorrected), showed grey matter loss in the right insular region, and bilateral cingulate gyri for patients compared to controls (figure). There were no areas of grey-matter volume increase in patients compared to controls. When assessed with correction for multiple comparisons, no differences in grey matter volumes were detected. After 6 months of treatment, pair-wise tests showed no difference in the brain volumes of OSA patients (both VBM and ROI).

Conclusion: In our homogenous population of severe OSA patients without co-morbidities, compared to healthy individuals with documented absence of sleep disturbances, only small areas of gray matter volume deficits were detected, which all did not survive correction for multiple comparisons. This is in contrast to an earlier study, which reported widespread, marked gray matter deficits in OSA patients. The difference is most likely explained by differences in the inclusion criteria (1). Our inclusion criteria were utilized in order to maximize the possibility of finding a true difference between OSA patients and controls, and thus excluded patients with co-morbidities, possibly contributing to the volume change. Our results suggest that grey matter volume deficits are not a prominent feature of OSA, when co-morbid diseases, such as cardiac failure are excluded.

References:

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