Sleep apnea linked to cognitive difficulties and deficits in gray matter

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Patients with obstructive sleep apnea (OSA) may blame their daytime difficulties on simple sleepiness, but new research suggests that their brains may be to blame. Specifically, their cognitive challenges may be caused by structural deficits in gray matter, brought on by the intermittent oxygen deprivation that comes with OSA. The good news is that these deficits may be partially or fully reversible with early detection and treatment, according to Italian researchers.

"OSA patients demonstrate several neuropsychological impairments, but current knowledge of the brain structures affected by OSA is limited. This study provides the first evidence that structural brain abnormalities exist in regions susceptible to hypoxemia, and they can change with treatment," said Vincenza Castronovo, Ph.D., clinical psychologist at the Sleep Disorders Center, Vita-Salute San Raffaele University and San Raffaele Scientific Institute in Milan, Italy.

The study was published online ahead of the print edition of the American Thoracic Society's American Journal of Respiratory and Critical Care Medicine.

To determine whether there were quantifiable structural differences in the brains of OSA patients when compared to individuals without OSA, and whether any differences found could be reversed with treatment, researchers compared 17 treatment-naïve individuals with severe OSA and 15 age-matched controls. They gathered baseline measurements of brain anatomy using MRI, as well as individual performance on

cognitive performance tests that assessed short- and long-term memory, executive functions, constructional abilities, vigilance, attention and abstract reasoning. The subjects also completed the Epworth Sleepiness Scale to assess daytime sleepiness and the Beck Depression Inventory to evaluate mood.

The researchers found significant reductions in gray matter (GM) between OSA and non-OSA subjects. Moreover, the specific locations of the deficits indicated that specific brain functions were more strongly affected than others, including executive function (which controls high-order brain functions such as problem-solving) and abstract reasoning.

"We found reduced GM in the OSA group when compared to the non-OSA group in several key regions of the brain," said Dr. Castronovo. "Of particular note were the deficits in the left parahippocampal gyrus and in the left posterior-parietal cortex. We found that these two regions have significant bearing on abstract reasoning and executive function. Deficits in the left posterior-parietal cortex were also shown to be associated with daytime sleepiness."

They also found that on tests, subjects with OSA demonstrated impairments in memory, attention, executive functions and constructional abilities and had higher sleepiness scores.

Remarkably, however, treatment with continuous positive airway pressure (CPAP), seemed to reverse these damages. After three months of treatment, the subjects in the OSA group were evaluated again. There was a GM-volume increase in the left anterior parahippocampal gyrus, which was associated with improved performance on tests of short-term memory and executive function. OSA subjects who had undergone treatment also had gains in the right cornu-ammonis and the enthorinal-cortex bilaterally, both of which were associated with improvements in executive function. Indeed, the researchers observed significant

cognitive improvement in all domains.

While previous studies have published contrary findings, Dr. Castronovo pointed out that with the increasing sensitivity of neuroimaging software, more sensitive analyses and robust results are now possible.

"These results suggest a scenario in which the hippocampus, due to its sensitivity to hypoxia and innervation of small vessels, is the region that is most strongly and quickly affected by hypoxic episodes," said Dr. Castronovo. "The mechanism of the observed brain changes could be either neurogenic or vasogenic," she continued. "We propose it to be vasogenic."

Further research will be necessary to bear out Dr. Castronovo's theory. If it is borne out, it could open new doors for treatment, such as steroid therapy.

"Next, our group will focus on increasing our understanding of brain damage and recovery," said Dr. Castronovo. "We want to evaluate non-hypoxic sleep disordered breathing in sleepy patients to assess the role of hypoxia in structural brain involvement."

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