MEMORY, SLEEP AND OBSTRUCTIVE SLEEP APNEA
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Memory is the ability of an organism to store, retain and recall information. Current studies in neuroscience strongly support the notion that a memory is a set of encoded neural connections. Encoding is the registration by the brain of information that has to be stored and then recalled.

Based on the duration of the retention of information, memory is classified into three categories: sensory, short-term and long-term memory. The hippocampus, amygdala, fornix, thalamus and mammillary bodies are involved in specific types of memory. It is widely accepted that the hippocampus, a complex brain structure that is shaped like a seahorse, plays an important role in converting short-term memory into long-term memory.

Recently in China, researchers investigated the effects of corticosterone on the hippocampus. They concluded that corticosterone is necessary for the “dentate gyrus,” an important part of the hippocampus that is thought to play a role in the formation of new memories. The researchers also determined that administration with minimum corticosterone during infancy has a long-term, positive influence on the hippocampus and its function in different developing stages.¹

Data indicate that the hippocampus is involved in contextual memory, which is deranged during rapid eye movement (REM) sleep dreams. This suggests that one reason for the derangement could be a change in the efficacy of synaptic transmission in the hippocampus. During sleep this may result from increased concentrations of the neurotransmitters acetylcholine and dopamine, and of the steroid hormone cortisol, along with decreased concentrations of the neurotransmitters serotonin and norepinephrine.² The changes in functioning of the hippocampal loop underlie differences in how memory information is stored and extracted during REM sleep compared with how this process occurs during wakefulness.³

Recent research suggests that word-pair learning relies on stage 2 sleep spindles and requires little slow wave sleep. Simple motor tasks either may be consolidated in stage N2 sleep or may depend on only small amounts of REM sleep.⁴

MEMORY AND SLEEP APNEA
Many pathological processes including heart failure and obstructive sleep apnea (OSA) are accompanied by memory loss or memory deficit. This is mainly an effect of hypoxia, the inadequate supply of oxygen to the body. Spatial and working memory also may be compromised by injury to the mammillary bodies, two round groups of nuclei on the undersurface of the brain, and the fornix, a c-shaped group of fibers in the brain.⁵

OSA is a common sleep disorder that is characterized by repeated occurrences of hypoxia, hypercapnia (i.e., high carbon dioxide), and transient blood pressure elevation that may damage or alter neural structures. OSA has been shown to compromise emotional and cognitive functions including short-term memory.

Although some memory inadequacies in OSA may result from structural deficits in the hippocampus, mammillary body injury also may be a contributing factor. These structures receive projections from the hippocampus via the fornix and project heavily to the anterior thalamus, a part of the brain through which sensory nerve impulses pass. They have been implicated in other conditions with memory deficiencies such as Korsakoff’s syndrome, a brain disorder involving amnesia and invented memories.

Kumar and colleagues conducted a study that manually traced the brain sections containing both mammillary bodies and calculated their volume. They found that left-side mammillary bodies showed greater volume reduction than right-side bodies. Diminished mammillary-body volume in OSA patients may be associated with memory and spatial orientation deficits found in the syndrome. The mechanisms contributing to the volume loss are unclear, but they may relate to hypoxia and ischemia (i.e., low blood supply). Nutritional deficiencies related to OSA also may play a role in the volume loss.⁶

Brain-morphology studies suggest a significant age effect on total gray matter in control subjects but not in patients with OSA. In multiple sites of the brain in OSA patients, there appears to be a significant unilateral loss of gray matter, which is nerve tissue that contains fibers and nerve cell bodies. These sites include the frontal and parietal cortex, temporal lobe, anterior cingulate, hippocampus and cerebellum. Unilateral loss in well-perfused structures suggests the onset of neural deficits early in the OSA syndrome. The gray matter loss occurs within sites involved in motor regulation of the upper airway as well as in areas contributing to cognitive function.⁷

This also has been confirmed by an independent study in which researchers found more extensive loss of gray matter bilaterally in the parahippocampus in addition to a deficit in the left hippocampus.⁸

Neuropsychological variables are correlated with neither the apnea/hypopnea index (AHI) nor the frequency of sleep arousals,

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but they are correlated with measures of sleep hypoxia in OSA patients with tetraplegia (i.e., quadriplegia, the paralysis of all four limbs or of the entire body below the neck). These deficits may affect rehabilitation in this subset of the population. The neuropsychological functions most affected by nocturnal desaturation are: verbal attention and concentration, immediate and short-term memory, cognitive flexibility, internal scanning and working memory.9

Treating OSA with continuous positive airway pressure (CPAP) therapy may enhance the speed of information processing and vigilance, and may sustain attention and alertness.10

CONCLUSION

Research indicates that sleep plays an important role in memory consolidation and cognitive functioning. Further cognitive testing may be necessary to reveal more subtle memory deficits resulting from sleep-disordered breathing. It is essential that future studies continue to define those deficiencies that are specific to OSA, the relationship between levels of severity and impairment, the role of treatment in reversing these dysfunctions, and the correlation between test results and significant day-to-day social and functional impairment.

REFERENCES