Neurocognitive Decline in a Recreational Pilot With Obstructive Sleep Apnea

Case Report, by Russell C. Tontz, III, MD, MPH

Obstructive sleep apnea (OSA) has been long recognized as having the potential for long-term cardiovascular effects for airmen. However, more focus should be on the detrimental effects OSA may have on the neurocognitive functioning of the airman and the potential consequences to aviation safety if left unrecognized and not controlled.

History

A 42-year-old male Class-3 pilot presents to his aviation medical examiner upon recommendation from his family physician. The pilot works as a construction engineer for a busy commercial construction firm. Over the past month, his colleagues have noticed that he is less decisive in his decision-making and has displayed more forgetfulness. The applicant admits that he is drinking more coffee to stay awake throughout the day and that he is not as “sharp” as he usually is at work. He owns a Piper Warrior and has flown it 200 hours since obtaining his Class-3 certificate five years ago. However, he has not flown in the past six months due to increased work demands.

The pilot’s only other reported health condition is that he is overweight. He denies any history of smoking or illicit drug use and reported minimal alcohol use. His physical exam was benign—other than a BMI (body mass index) of 32 and a neck circumference of 17 inches. An office MMSE (Mini Mental Screening Examination) was within normal limits. An Epworth sleepiness scale given in the office was 15.

Upon further questioning regarding his sleep behavior, the applicant admits that his wife has mentioned that he has always snored quite loudly. He says that he wakes up at night for unknown reasons and feels tired in the morning, although he tries to get eight hours of sleep every night. His family physician, who has known him since a child, notices an obvious difference in his personality and, knowing he is a private pilot, asks that he be evaluated by his AME.

Aeromedical Concerns

This individual needs to be evaluated for obstructive sleep apnea because he has risk factors such as elevated BMI, neck circumference, and elevated Epworth Sleepiness scale scores.

He admits to having poor sleep and resultant daytime somnolence, which is a clear risk to flight safety. His forgetfulness may be the result of his OSA and the resultant chronic intermittent hypoxia.

The FAA will consider each case on an individual basis, in consultation with a specialist, after an overnight polysomnography (PSG) is obtained. The American Academy of Sleep Medicine recommends that portable monitoring can be used as an alternative to the gold standard PSG, when the patient has a high pre-test probability of moderate-to-severe OSA. The documentation of treatment and treatment response is also necessary for the FAA to medically certify.

In this case, the PSG showed severe sleep apnea, and the sleep specialist recommended not only CPAP but also neurocognitive testing, based on the airman’s complaints of forgetfulness. The neurocognitive test showed verbal and spatial deficits below average for the airmen’s age and level of education. No baseline tests were available to compare the current findings with, but the results were consistent with research that suggests links between OSA and neurocognitive changes.

Outcome

The Regional Flight Surgeon disqualified the airman for any class of medical certificate and requested that he promptly surrender his unexpired medical certificate. The case was reviewed by the Aerospace Medical Certification Division (AMCD), and they recommended that the airmen be treated with continuous positive airway pressure, based on the recommendations from the sleep specialist. Repeat PSG and neurocognitive testing were to be done in six months. The repeated tests showed much improvement, as did the airman’s symptoms. The AMCD determined that his condition was compatible with aviation safety and that he was eligible for a third-class airman medical certification under the provisions of an Authorization for Special Issuance of a Medical Certificate, as this applicant has a medical condition that is disqualifying under Title 14 of the Code of Federal Regulations (14 CFR) part 67.

Discussion

The most common medical cause of excessive daytime sleepiness is obstructive sleep apnea. OSA is a common sleep disorder, affecting at least 2 to 4% of middle-aged individuals. OSA is characterized by the repetitive complete or partial collapse of the upper airway during sleep. Collapse of the upper airway causes cessation (obstructive apnea) or a significant reduction (obstructive hypopnea) of airflow. In career fields like aviation, the risk of excessive daytime sleepiness not only affects the individual but also affects the safety of the general public.

Numerous sleep disorders are classified within the broad category of sleep-disordered breathing. All of these syndromes are characterized by the cessation or partial cessation of airflow, causing multiple arousals from sleep. Obstructive sleep apnea syndrome is the most common type of sleep apnea and presents, together with central sleep apnea, the most severe sleep-disordered breathing condition (caused by an obstruction of the upper airway). The severity is usually determined by the respiratory disturbance index, which is the sum of the number of apneas and hypopneas per hour of sleep. An apnea is a cessation of airflow, and a hypopnea is a 30% to 50% reduction in airflow during at least 10 seconds. Since its introduction in 1981, positive airway pressure has been the most efficacious therapy and is often the first option for OSA patients.
Chronic, untreated OSA is an independent risk factor for hypertension, diabetes, stroke, and cardiac rhythm disturbances, along with the higher risk of occupational injury and lost work productivity. The cardio-metabolic abnormalities in OSA are believed to be the consequence of intermittent hypoxia and sleep fragmentation, which promote atherosclerosis through increased sympathetic drive, oxidative stress, and inflammation of the vasculature.

Besides the known cardiovascular complications of uncontrolled OSA, neurocognitive impairment may also be present. This neurocognitive impairment seen in OSA is due to the adverse effects of sleep fragmentation and/or intermittent hypoxia, and it remains unclear whether cognitive deficits return to normal after OSA treatment. Patients with severe OSA may experience hundreds of respiratory disturbances per night, resulting in extreme sleep fragmentation.

The clinical pathology of the intermittent hypoxemia associated with sleep apnea syndrome and the apnea of prematurity suggests that there may be long-term adverse consequences of chronic cyclical hypoxia. Besides the known risk for hypertension, coronary and cerebral vascular disease, some researchers suggest that persistent bouts of hypoxia may result in residual neurocognitive effects of OSA such as verbal and spatial executive deficits that may occur in individuals with severe OSA, despite treatment and normalized sleep.

A meta review by Bucks et al. supports the presence of relationships between attention/vigilance dysfunction and sleep fragmentation, and between hypoxemia and global cognitive function. A significant decrease in overnight verbal memory consolidation is noted in patients with OSA, compared to healthy subjects. Furthermore, treatment of OSA with continuous positive airway pressure appears to improve executive dysfunction, delayed long-term verbal and visual memory, attention/vigilance, and global cognitive functioning.

Canessa et al. demonstrated not only neuropsychologic impairments in memory, attention, executive functions, and constructional abilities, but also associated decreases of gray-matter volume in specific cerebral regions. After three months of treatment, they observed a significant improvement in all cognitive domains, as well as an increase of gray-matter volume in specific hippocampal and frontal brain regions. In the study by Canessa et al., true structural brain changes related to OSA and its reversibility with proper treatment may be a motivating factor for compliance with treatment goals. These changes are significantly correlated with improvement in specific neuropsychologic tests (executive functioning and short-term memory), underlining the importance of early diagnosis and treatment of sleep apnea.

Conversely, Verstraeten suggests that this increased cerebral response is more consistent with the adaptive compensatory changes following total sleep deprivation and not necessarily structural prefrontal brain damage. Although nocturnal hypoxemia may eventually result in neuronal damage or injury in severe OSA that remains untreated for a long period of time, there is no solid scientific basis today to suggest the presence of irreversible brain lesions in OSA patients as a group.

Most would agree that early detection and treatment of OSA is important to prevent or stop the cardiovascular consequences of this disorder. Further research to better determine the underlying mechanisms needs to be promoted. The risk of neurocognitive decline should be emphasized to all OSA patients so as to increase their motivation for compliance to improve their quality of life.

References


About the Author

Maj Russell C Tontz, III MD, MPH, is a board certified Family Medicine and Aerospace Medicine physician who currently is a Resident of Aerospace Medicine at the USAF School of Aerospace Medicine. At the writing of this case report, he was on rotation at the FAA’s Civil Aerospace Medical Institute.