Obstructive sleep apnea (OSA) is a common public health problem manifested by sleep-disordered breathing, daytime hypersomnia and poor sleep quality, adverse neurocognitive sequelae, and hypoxia. OSA occurs in about two to four percent of the general population, or an estimated 18 million Americans.1

WHAT IS SLEEP DISORDERED BREATHING?
Obstruction of the upper airway during sleep causes a continuum of breathing disturbances, varying from mild snoring, to partial airway obstruction causing a heightened respiratory effort necessary to preserve airflow and oxygenation, thereby leading to arousal (a respiratory effort related arousal, or RERA), to airflow limitation (hypopnea), and to cessation of airflow (apnea). Some patients have a typical predisposing anatomy of a narrowed oropharynx. Anatomical factors at the level of the nose, nasopharynx, oropharynx, or hypopharynx may all predispose, and most adult patients have multi-level obstructive factors. Common anatomical factors increasing vulnerability toward OSA include nasal septal deviation, polyps, a low-lying palate or redundant soft palatal tissue, a thickened tongue base, or a narrowed hypopharynx.

The mildest form of upper airway obstructive sleep disordered breathing is snoring. Snoring results from narrowing in the nasal passages or oropharynx significant enough to produce turbulent airflow, leading to vibration of the soft palatal tissue. Primary snoring is diagnosed when no other disturbance in sleep or respiration is found during polysomnography. While snoring has been correlated with risk of hypertension, primary snoring is basically otherwise benign except for its disruptive effects on the sleep of bedpartners or roommates.

OSA AND NEUROCOGNITIVE FUNCTION
OSA causes cessation of airflow with resulting hypoxia and microarousals that fragment sleep, leading to nonrestorative sleep and reduced daytime functioning from sleepiness and cognitive impairment. OSA-related sleep fragmentation causes significant morbidity due to impaired daytime functioning, quality of life, and driving safety.2 Daytime dysfunction includes hypersomnia, attentional impairments, executive dysfunction.3-5

OSA-related cognitive impairments are common and broad, including speed of information processing, attention and working memory, executive functioning, learning and memory, alertness and sustained attention, visuospatial learning, motor performance, and constructional abilities, but OSA largely spares global cognitive functioning and language.6-8 Attentional impairments in adult OSA patients are comparable to the effects of alcohol intoxication.7 Vigilance impairments may be enduring or more momentary, with microsleeps mimicking inattention or lapses in concentration; deterioration in driver control over vehicle position and steering has been shown to occur during microsleep episodes in drivers with OSA.8 OSA increases crash risk by two- to three-fold, irrespective of sleepiness or apnea severity.8 Sleep fragmentation, hypoxia, or both may mediate these impairments.6-8

The course of OSA-related deficits following treatment with nasal continuous positive airway pressure (CPAP) is variable, but improvements in vigilance, attention, and reaction time are expected. Treatment with CPAP reduces subsequent crash risk in commercial drivers by 72 percent, toward a level approaching the background rate in the general population.10 However, several structural, functional, and magnetic resonance spectroscopic neuroimaging studies in treated OSA patients have shown signs of persistent hippocampal, dorsolateral prefrontal, cingulate, and posterior parietal neural damage despite nasal CPAP treatment, suggesting that identification and treatment of OSA should be a public health imperative to prevent permanent, irreversible cognitive sequelae of OSA.5-11-12

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