Current Treatments for Sleep Disturbances in Individuals With Dementia

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Sleep disturbances are widespread among older adults. Degenerative neurologic disorders that cause dementia, such as Alzheimer’s disease and Parkinson’s disease, exacerbate age-related changes in sleep, as do many common comorbid medical and psychiatric conditions. Medications used to treat chronic illness and insomnia have many side effects that can further disrupt sleep and place patients at risk for injury. This article reviews the neurophysiology of sleep in normal aging and sleep changes associated with common dementia subtypes and comorbid conditions. Current pharmacologic and nonpharmacologic evidence-based treatment options are discussed, including the use of light therapy, increased physical and social activity, and multicomponent cognitive-behavioral interventions for improving sleep in institutionalized and community-dwelling adults with dementia.

Introduction

Current estimates indicate that 35 million Americans over the age of 65 years are living in the United States. This number is expected to double by the year 2030. Along with advanced age comes a myriad of chronic illnesses, many of which eventually cause dementia. The decreased functional status, changes in cognition and mood, and behavioral disruptions, including sleep disturbances, that are frequently seen in people with dementia place significant stress on the family and caregivers. The resulting increased burden is associated with increased rates of institutionalization and increases in overall health care costs [1].

The causes of sleep disturbances in individuals with dementia are multifaceted, including the following: 1) physiologic changes related to the dementing illness and normal, “nonpathologic” aging; 2) primary sleep disorders such as sleep apnea and restless legs syndrome; 3) medical and psychiatric morbidity; 4) medication side effects; 5) environmental and behavioral factors, including poor “sleep hygiene”; and 5) some combination of the above [2]. Although dementia’s progression is largely irreversible, several measures that can improve sleep in individuals with dementia may ease caregiver burden and reduce the risk for premature institutionalization. In this article, we describe the neuropathology of sleep, the sleep changes associated with the most common dementia subtypes, and evidence-based treatment options.

Neurophysiology of Sleep

Sleep is a complex phenomenon that is rooted in neurologic function. The central sleep and circadian regulation centers are located deep within the brain and include the anterior hypothalamus, reticular activating system, suprachiasmatic nucleus (SCN), and pineal gland. Sleep is generally understood to be governed by an interaction of circadian and homeostatic processes. The homeostatic process of sleep refers to “sleep drive”—that is, the fact that sleep tendency increases as one gets further away from the last sleep period and decreases the longer that sleep time is accumulated. The circadian timing system underlies the temporal organization of most neurobehavioral and physiologic processes, including body temperature, melatonin production, and the 24-hour sleep–wake cycle [3•]. The SCN is a group of neurons located at the base of the hypothalamus, just above the optic chiasm, where the optic nerves meet and cross. The SCN is highly sensitive to light. Light entering the retina travels along the optic nerves to the SCN, which triggers the pineal gland to stop producing the neurohormone melatonin. Melatonin is an essential component in sleep, thermoregulation, and blood pressure; its production is highest during the night, when light stimuli are minimal or absent. The reticular
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Alzheimer's disease

Alzheimer's disease (AD) is the most common form of dementia in the United States. Current estimates indicate that 5.1 million Americans are living with AD. Most of these individuals are over the age of 65 years, and the prevalence rate increases with advancing age. As a result of the aging baby boomer population, by the year 2050, it is projected that 60% of those over the age of 85 years—11 to 16 million individuals—will have AD [1,6].

Cross-sectional studies suggest that approximately 25% to 35% of individuals with AD have problems sleeping [7•]. Sleep disturbances in AD are believed to be a result of a progressive deterioration and decrease in the number of neurons in the SCN, which cause fluctuations in neurohormones that are critical in the homeostatic maintenance of the circadian rhythm [8]. Common symptoms include nighttime sleep fragmentation, increased sleep latency, decreased slow-wave sleep, and increased daytime napping. “Sundowning,” another common phenomenon occurring during the middle to late stages of AD, is marked by an increase in confusion, wandering, and agitation that often (although not always) occurs in the late afternoon into the evening, with improvements seen during the daylight hours. Although the nature of sundowning has been debated over the years, it is believed to be related to a disturbance in circadian rhythm that causes significant delays in peak body temperature and alterations in endogenous melatonin secretion [8].

Medications used to lessen the negative behavioral symptoms of AD and to slow disease progression are often associated with side effects that negatively affect sleep and wakefulness. Acetylcholinesterase inhibitors such as donepezil slow cognitive decline in some patients with AD but can cause nighttime stimulation and have been associated with reports of dream disturbances [7•]. Atypical antipsychotic medications such as olanzapine and risperidone increase daytime fatigue and somnolence [9•]. Use of these medications should be individualized based on patient status, behavioral symptom severity, and patient sensitivity to side effects.

Parkinson's disease and Lewy body dementia

Parkinson's disease (PD) is caused by progressive degeneration of the substantia nigra, which normally produces the neurotransmitter dopamine. The reduction in the manufacturing of dopamine causes “misfiring” of nerve impulses within the brain and results in the characteristic motor abnormalities seen in the disease. The onset of dementia in PD patients typically occurs 10 or more years after the initial onset of motor signs. PD is part of a complex of neurodegenerative disorders called the synucleinopathies, which also include diffuse Lewy body disease (DLBD). DLBD shares many pathologic characteristics with PD and AD, including the presence of Lewy bodies and senile plaques, but is clinically distinguished by a more rapid onset and progression of dementia, fluctuating cognition with variations in attention and alertness, recurrent visual hallucinations, and parkinsonian motor signs [10].

Sleep disturbances are highly prevalent among patients with PD and DLBD [7•,11]. Common problems include prolonged sleep latency, increased nighttime sleep fragmentation, nightmares, and increases in early-morning awakenings. Daytime sleepiness and sudden-onset sleep attacks during waking hours are also common and a significant threat to patient safety and quality of life. REM sleep behavior disorder is a condition in which individuals physically act out dreams during REM sleep, particularly in the second half of the night [12]. This occurs because of a disruption in the normal sleep paralysis mechanism that inhibits this action. Body movements can be violent and can even cause harm to the patient or bed partner but often are not remembered after awakening in the morning. REM sleep behavior disorder is most common in older men, and most individuals diagnosed with it ultimately go on to develop symptoms of DLBD or PD [12].