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Current Treatments for Sleep Disturbances in Individuals With Dementia

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Abstract

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

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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 activating system located within the midbrain has less to do with the actual sleeping process and more to do with maintaining a state of arousal and awareness of one's environment. Disruptions anywhere along this pathway can cause disruptions in the circadian rhythm and, ultimately, sleep disturbances [4].

Sleep is an active process wherein certain brain structures are activated at certain stages of sleep and deactivated at others [5]. Normal sleep consists of four phases of non–rapid eye movement (non-REM) sleep and one phase of REM sleep, each of which has distinct electroencephalogram characteristics. The brain cycles through these phases approximately every 90 minutes, with four or five cycles per night. Stage 1 is the transition between wakefulness and sleep; individuals in stage 1 sleep are easily awakened and may not even know they had been asleep. Stage 2 sleep involves a loss of conscious awareness and the appearance of characteristic “sleep spindles” and “K complexes” in the electroencephalogram, as well as a decrease in heart and respiratory rates and body temperature. In stages 3 and 4, also known as *deep sleep*, brain waves slow, and arousal is more difficult. If arousal does occur, the individual is groggy and disoriented. REM sleep is the final stage and the stage during which dreaming occurs. During REM sleep, a person's eyes move rapidly, heart and respiratory rates increase, and muscle twitching often occurs. As part of the normal aging process, stages 3 and 4 and REM sleep decrease significantly, which may account for some of the frequent nighttime awakenings, difficulty returning to sleep, and daytime fatigue commonly reported by older adults.

Sleep Disturbances Associated With Dementia Subtypes

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].

In comparison with AD, patients with PD have unique physiologic symptoms that further contribute to sleep disturbances. Hallmarks of PD include muscle rigidity, tremors, akinesia, dystonia, and muscle stiffness that make sleep initiation and sleep maintenance more difficult because of persistent movement and painful joints and muscle spasms. Patients with PD also have increased rates of restless legs syndrome and periodic limb movement disorder that further contribute to fragmented sleep [7•].

Unfortunately, many medications prescribed to alleviate the bothersome symptoms of PD contribute to daytime sleepiness and nighttime awakenings. Levodopa reduces the duration of REM sleep and increases REM sleep latency. Dopamine agonists, including bromocriptine, can cause a sudden and significant attack of daytime sleepiness that can be dangerous if the patient is participating in activities requiring a high level of alertness. Additionally, dopamine agonists and anticholinergics have nighttime stimulating effects and can increase confusion and hallucinations in patients taking these medications [7•].

Primary Sleep Disorders and Medical Morbidity

A recent National Institutes of Health statement on insomnia notes that it is difficult to separate the effects of insomnia from those of comorbid conditions such as depression and pain [13]. As most individuals with dementia are older adults, they are at risk for a variety of age-related

comorbid conditions that can further exacerbate sleep disturbances. Chronic diseases such as ischemic heart disease, diabetes, depression, renal failure, arthritis, and pulmonary disorders and the multiple medications used to treat them are common in the older adult population and increase risk for development of insomnia [14]. Current thought regarding the association between sleep disturbances and heart failure is that it is caused by a multifactorial process related to medication, sleep-disordered breathing, and clinical variables, including remobilization of edematous fluid, nocturia, paroxysmal nocturnal dyspnea, and early-morning episodes of chest pain [15]. Ischemic heart disease, congestive heart failure, and dilated cardiomyopathy have been linked to higher rates of obstructive sleep apnea and restless legs syndrome, further contributing to fragmented sleep and excessive daytime sleepiness [15,16]. Chronic pain from arthritis and other disorders interferes with sleep. In turn, disturbed sleep reduces pain threshold [17]. Narcotic analgesics routinely used to control chronic pain can cause excessive daytime somnolence. Other common medications used to manage chronic disease add to sleep disturbances in older adults and include diuretics for treating heart failure that cause nocturia and bronchodilators for pulmonary disorders that have nighttime stimulating effects.

Psychiatric morbidity is also associated with insomnia in older adults with dementia, particularly mood disorders, which can be both a symptom and a predictor of insomnia [18]. Psychotropic medications used to treat conditions such as depression and anxiety can reduce risk for the development of sleep problems secondary to these disorders. However, they are also associated with reduced sleep quality in individuals with untreated obstructive sleep apnea [19] and can have other adverse side effects, including risk for falls and daytime sleepiness in older adults with cognitive impairment.

Pharmacologic Treatments

Pharmacologic treatment is the mainstay of the short-term treatment of sleep disturbances in individuals with dementia. Antidepressants, benzodiazepines, nonbenzodiazepines, and antihistamines are commonly used, although limited empiric evidence exists for their long-term safety and use with cognitively impaired older adults.

Sedative-hypnotic agents, particularly benzodiazepines and nonbenzodiazapines, are the most widely used medications for the short-term treatment of sleep disturbances. Benzodiazepines act to decrease sleep latency and increase total sleep time and decrease the amount of time spent in stage 2 sleep, although they have little effect on the sleep maintenance problems that are most common in older adults [20]. Unfortunately, benzodiazepines are associated with increased incidence of sedation, confusion, anterograde amnesia, daytime sleepiness, and rebound insomnia. The newer generation nonbenzodiazepines, including zolpidem, zaleplon, and eszopiclone, have shorter half-lives and fewer side effects, but data for their use with demented older adults are lacking.

Antidepressants are frequently prescribed as a matter of course for sleep problems in individuals with dementia, as it can be difficult to distinguish whether the sleep disturbances are secondary to depression in these individuals. Selective serotonin reuptake inhibitors are the most common antidepressants prescribed, but trazodone is also widely used, although virtually no evidence-based data support its efficacy with older adults [21]. Although they are effective in decreasing sleep latency, these medications also have undesirable side effects, including somnolence, sedation, dizziness, and weight gain in the older adult population.

Antihistamines, particularly diphenhydramine, are found in many over-the-counter sleep aids and are widely prescribed to older adults [22]. These medications have very high rates of side effects, including sedation, cognitive impairment, increased daytime somnolence, and

anticholinergic responses. Because of side effect risks, they should be avoided as first-line treatment in the older adult population.

As noted previously, the pineal hormone melatonin has circadian and direct sleep-promoting effects. However, the two largest placebo-controlled trials to date that tested the efficacy of melatonin supplementation as a stand-alone treatment for insomnia in patients with AD have yielded nonsignificant results [23,24]. Melatonin agonists are a newer class of hypnotic medications that gained US Food and Drug Administration approval in 2005 and have promising implications for treating sleep disturbances, particularly in older adults. Ramelteon, the first and only medication within this category, acts on the MT1 and MT2 receptors to stimulate the action of melatonin to induce and shorten sleep latency [25]. This medication also has been shown to improve sleep efficiency and increase total sleep time. Of particular benefit to older adults is that ramelteon has not been associated with negative side effects such as cognitive impairment, rebound insomnia, withdrawal effects, or abuse potential that other agents used to treat insomnia produce. Because of the lack of abuse potential, ramelteon is not a scheduled medication and has been approved for long-term treatment of insomnia.

Nonpharmacologic Treatments

Light therapy

Light plays a major role in regulating the phase relationships among core body temperature, melatonin rhythm, and the circadian rest–activity cycle. The American Academy of Sleep Medicine has published practice parameters for the use of bright light to treat sleep and circadian rhythm disorders [26,27••]. However, there is currently no accepted gold standard for when light exposure should occur, how long it should be delivered, which light wavelengths are maximally safe and effective, or which method of light delivery is optimal. Typically, light exposure is timed to coincide with the beginning and end of the human photoperiod. Evening bright light treatment is beneficial for sleep maintenance problems in older adults and for phase-advanced individuals (ie, those who fall asleep in the early evening and awaken too early in the morning). Morning light exposure is most beneficial for phase-delayed individuals (ie, those whose sleep onset and morning rising are pushed to later hours) or those who may be suffering from a seasonal depressive disorder.

Over the past decade, interest has increased in the effects of light therapy on the treatment of sleep disorders in older adults with dementia, both alone and combined with other therapies [28]. With the exception of a few multicomponent behavioral interventions described subsequently, all the controlled studies to date examining the effects of light therapy on sleep in dementia have been conducted with nursing home residents. Lyketsos et al. [29] reported improvements in total sleep time at night after 4 weeks of morning light exposure delivered using a full-spectrum light box for 1 h/d (dose > 2500 lux). However, in a series of larger studies, Ancoli-Israel et al. [30-32] and Dowling and colleagues [33,34] found that neither 1 nor 2 hours of morning or evening bright light improved nighttime sleep quality, although improvements were noted in the timing and stability of rest–activity rhythms, particularly in individuals who had the most disturbed sleep–wake patterns at the start of treatment.

Furthermore, using a standing light box is problematic because patients must sit still for the treatment and be supervised so they do not fall asleep or wander away. To address this, investigators have looked at whether wall- or ceiling-mounted illumination systems that expose nursing home residents to light without restricting personal movement or activity can improve sleep. In a recent controlled trial, residents were exposed to higher levels of ceiling-mounted light for 4-hour intervals in the morning or evening, compared with all day for 11 hours and also with standard interior lighting [35]. Bright light exposure in the morning or all day produced significant improvements in total nighttime sleep. Treatment effects were related to

dementia severity, season of the year (which affects daytime light length), and sedating medication use. In a small but intriguing pilot study, Fontana Gasio and colleagues [36] also found that nursing home residents exposed to low-intensity, dawn-to-dusk stimulation had significantly shortened sleep latency and increased sleep duration post-test compared with residents exposed to a dim red light placebo.

Finally, several investigators have examined whether combined light therapy and melatonin supplementation enhances treatment sleep effects. Dowling et al. [37] reported that nursing home residents who received greater than 2500 lux of morning light (via light box) plus 5 mg of melatonin at night for 10 weeks had significantly greater daytime activity and improvement in the day–night sleep ratio compared with individuals who received bright light alone. Riemersma-van der Lek and colleagues [38••] also found that combination melatonin (2.5 mg) and light (whole day, ceiling mounted) reduced resident agitation and improved several sleep parameters, including sleep latency, total sleep time, duration of uninterrupted sleep bouts, and sleep efficiency. Although some adverse side effects were reported for melatonin (dysphoric mood) and light therapy (irritability, dizziness, headache), and treatment effects were relatively modest, the authors concluded that whole-day bright light is safe for use in long-term care settings.

Physical activity

Regular exercise builds muscle mass, improves strength, reduces falls, and improves mood in older adults and younger people. Exercise also has been linked to phase shifting of circadian rhythms and promotion of more restful sleep in older adults [39,40]. Although regular physical activity may also enhance the sleep of individuals with dementia, no controlled trials looking at the isolated effects of exercise on sleep in dementia have been published thus far. Studies have shown that care-givers can be trained to function as exercise “coaches” for individuals with dementia, and older adults with a wide range of cognitive impairment enjoy participating in structured exercise programs [41]. Studies also have suggested that participation in nonstrenuous daytime activities can have a beneficial effect on sleep in nursing home residents with dementia. For example, Richards et al. [42] showed that residents with severely disturbed sleep (estimated baseline sleep percentage < 50%) randomized to receive 1 to 2 hours of individualized social activities in 15- to 30-minute sessions for 21 consecutive days had significantly reduced daytime sleep, sleep latency, and number of nighttime awakenings compared with controls.

Behavioral and multicomponent interventions

Many behavioral treatments for insomnia, including stimulus control, sleep restriction, progressive muscle relaxation, biofeedback, sleep hygiene education, paradoxical intention, and multicomponent cognitive-behavioral therapy, are known to be effective with older adults [43]. Because of the risk for sedating medication side effects, behavioral strategies are also commonly recommended as first-line treatment for sleep-disturbed individuals with dementia. Standard recommendations include maintaining regular bedtimes and rising times, limiting daytime napping, and restricting time in bed. Dietary recommendations include establishing consistent meal times; avoiding alcohol, nicotine, and caffeine; and emptying the bladder before bedtime. The sleep environment should not be overly hot or cold, and an effort should be made to reduce excess ambient light and noise [44].

Most of the randomized trials that have included behavioral recommendations to improve sleep in patients with dementia have been conducted in nursing homes and been multicomponent interventions focused on reducing day in-bed time, increasing social and physical activity, and altering the environment to make it more conducive to nighttime sleep. Naylor et al. [45] found that short-term (2 weeks) exposure to twice-daily structured group social activity combined

with low-intensity physical activity (total time, 90 minutes in both the morning and afternoon) increased slow-wave sleep in older adult residents. Alessi and colleagues [46] and Martin et al. [47] reported that 5 days of exposure to a multicomponent intervention that included resident exposure to outdoor bright light (≥ 30 min/d with light > 20,000 lux), keeping residents out of bed during the day, daily participation in a low-level physical activity program (10−15 minutes, three times daily), establishment of a consistent bedtime routine, and reduction of nighttime noise and light in residents' rooms led to an advance in rest/activity rhythms and modest reductions in the duration of nighttime wakefulness. However, Ouslander et al. [48] found no significant improvements in any sleep variable using a similar multicomponent intervention that was implemented in eight nursing homes using trained research staff.

Several factors may account for these mixed results. The multicomponent interventions were all relatively brief and differed in their treatment protocols. Nursing home residents tend to have high rates of medical comorbidity, moderate to severe cognitive impairment, and mixed dementia diagnoses, which can complicate treatment response. It also can be challenging to implement behavioral changes in institutional settings with variable staffing structures and care policies. It may be expected that behavioral interventions for sleep would be more effective with community-dwelling dementia patients, who usually have fewer medical complications, are less cognitively impaired, and have caregivers who can better control the home environment and sleep–wake routines. McCurry et al. [49] reported that patients in an 8-week multicomponent intervention that included sleep hygiene education, daily walking (30 minutes), and increased light exposure (1 hour using a light box) had a 32% reduction in time spent awake and nighttime awakening compared with controls, and treatment effects were maintained at 6-month follow-up. Although these results are promising, behavioral interventions that require changing established bed/rising routines and keeping a person with dementia awake during the day can be challenging for family caregivers, who may be worried about adding to their already-considerable caregiving burden and skeptical that behavioral interventions alone will help [50]. A larger, National Institutes of Health–funded, controlled trial is under way to examine the relative efficacy of walking, increased light exposure, and a combination intervention for improving sleep in community-dwelling individuals with AD. The outcomes of this trial will provide needed additional information about the long-term feasibility of behavioral treatments to improve sleep in this population, as well as data regarding the patient and caregiver characteristics that are associated with positive treatment outcomes.

Conclusions

Sleep disturbances in older adults with dementia are common. Their etiology is complex, involving multiple factors, such as neurodegenerative changes in the brain, the patient's environment, medical or psychiatric morbidity, and medications used to treat chronic illnesses and dementia-related behavioral symptoms. An accurate diagnosis of the neurologic disorder and comprehensive review of current medications are important for understanding possible causes of patient sleep changes and for developing a plan of care to improve nighttime sleep and daytime wakefulness and reduce caregiver burden.

Because of the multifaceted nature of sleep disturbances and fragility of older adult patients with dementia, nonpharmacologic options should always be considered as first-line treatment. Pharmacologic options should be used judiciously, with potential side effects seriously considered before prescribing hypnotic and psychotropic agents. In light of the aging of the US population and subsequent probable increase in the prevalence of dementia, continued research to develop safe, effective medications and solid, evidence-based guidelines for sleep problems in this population is needed.

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