REVIEW ARTICLE

Common Sleep Disorders Affecting Older Adults

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Abstract

Sleep disorders in older adults increase with aging, likely due to increased sleep latency, decreased sleep efficiency, and total sleep time. Common sleep issues include chronic insomnia, circadian rhythm sleep–wake disorders, sleep-related movement disorders, and sleep-disordered breathing. Diagnostic tools, such as a comprehensive sleep history and questionnaires, or a sleep log for more specific complaints, are commonly used. Polysomnography is not recommended as a routine test; however, it can be used for abnormal behaviors during sleep or if treatment fails. Sleep disorder management is based on the etiology and may include nonpharmacological and pharmacological alternative treatments. For example, nonpharmacological management for chronic insomnia and some sleep disorders may consist of cognitive behavioral therapy, sleep hygiene education, relaxation therapy, sleep restriction, light therapy, and stimulus control therapy. Because the quality of evidence for pharmacological treatment is poor, the medication choice should be based on shared decision-making between the practitioner and the patient, with limited prescription.

Introduction

Sleep disorders are not part of the normal aging process, but their prevalence may increase with age. Therefore, it is crucial to identify factors associated with sleep disturbances, including substance abuse, chronic medical illnesses, psychiatric conditions, reduced physical activity, and changes in sleep habits that can be expected in older adults. In addition, poor sleep is linked to memory loss and worsening health outcomes in comparison to older adults without sleep complaints.[1] A few studies have also showed an increase in early mortality, nursing home placement, and suicide risk in older adults with sleep disorders.[2]

Diagnosing sleep disorders in the elderly can be challenging, given the physiological changes in this population. For example, older adults have increased sleep latency and decreased sleep efficiency and total sleep time. The age-related changes in sleep structure include alterations in rapid eye movement (REM) and non-REM sleep.[2–4] Non-REM sleep has 3 stages,

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namely, N1, N2, and N3; older adults have less N3 sleep and an increased percentage of the N1 and N2 stages. Also, older adults have decreased REM sleep and earlier sleep onset than younger adults, as well as increased incidences of nighttime awakenings and more daytime napping.[3,4] See Table 1.

Managing sleep disorders in older adults can be difficult due to their multiple comorbidities and medication side effects. However, treating the sleep condition or any common comorbidities associated with sleep difficulties may improve their quality of life and the consequences related to a lack of sleep.[2] Unfortunately, many older adults use over-the-counter medication to self-manage their sleep condition, increasing the risk of dependence, addiction, and falls, as well as cognitive impairment.[1]

Common sleep disorders in older adults are divided into several categories. These include chronic insomnia, circadian rhythm sleep–wake disorders (CRSWDs), sleep-related movement disorders, and sleep-breathing disorders (SBDs).[6] This article will review these sleep conditions in more depth and highlight their nonpharmacological and pharmacological management in the elderly population.

In this review, the authors aimed to update health care professionals on the more recent treatments and recommendations available to help them manage sleep disorders within their older adult population. This review incorporates sleep hygiene recommendations, and behavioral therapies, as well as possible pharmacological management.

Sleep stages	Hallmark changes in sleep stages
Daytime napping	Increased
Nighttime awakenings	Increased
Sleep latency (time to fall asleep)	Increased or no change
Stage N1 (transition between awake and sleep)	Increased
Stage N2 (throughout the sleep)	Increased
Percentage REM sleep	Decreased
Sleep efficiency (time asleep over time in bed)	Decreased
Stage N3 (slow-wave sleep)	Decreased
Total sleep time	Decreased

Table 1: Sleep changes with aging[2,5]

N = stage of non-REM sleep; REM = rapid eye movement.

Chronic Insomnia

DEFINITION

According to the International Classification of Sleep Disorder diagnostic criteria, insomnia is defined as difficulty initiating or maintaining sleep associated with daytime impairment.[7] The sleep problems must occur at least 3 times per week and be present for at least 3 months.[7]

EPIDEMIOLOGY

Approximately 50% of the elderly complain about difficulty initiating or maintaining sleep.[8] Furthermore, sleep difficulties are more prevalent in females than males of all ages, increasing from 10% in young adults to 30% to 48% in older adults.[2,8] Self-reported sleep difficulties are more common in older Black females, especially in those with associated chronic diseases and depression.[2] Sleep maintenance is the most common type of insomnia (50% to 70%), followed by difficulty initiating sleep (35% to 60%). Roughly 25% of older adults report nonrestorative sleep.[8]

DIAGNOSIS

Insomnia in older adults is often chronic and commonly associated with other medical and psychiatric conditions.[2] The increased sleep disturbance with age is mainly related to higher risk factors for sleep disturbance than the aging process itself.[2,3] A comprehensive sleep history is used to evaluate sleep disorders, including sleep–wake patterns, daytime consequences, presleep conditions, and time spent napping during the daytime. Furthermore, asking about medications, other substances taken for sleep, and specific insomnia complaints with a questionnaire, interview, or a 2-week sleep log is advised.[4,5,9]

In addition, a comprehensive sleep evaluation should try to rule out alternative causes like medical conditions, psychiatric disorders, and substance abuse.[9] The American Academy of Sleep Medicine does not recommend polysomnography or daytime sleep latency testing in the routine evaluation of chronic insomnia.[9] However, polysomnography is endorsed for a sleep-related breathing disorder, narcolepsy, abnormal behaviors during sleep, or if treatment fails.[2,4] Neuroimaging is not recommended as an acceptable means of evaluating insomnia.[9]

NONPHARMACOLOGICAL MANAGEMENT

Multiple nonpharmacological treatment options, including cognitive behavioral therapy for insomnia

(CBT-I), sleep hygiene education, relaxation therapy, imagery, sleep restriction, and stimulus control therapy, should be considered first-line therapies.[6,10] For more information, see Table 2 and Table 3.

PHARMACOLOGICAL MANAGEMENT

Pharmacological management can be used in those who fail to respond to nonpharmacotherapy or still have severe symptoms.[15] The overall quality of evidence for pharmacologic treatment is low. Medication choices should be based on shared decision-making between the practitioner and the patient, and use of prescription medications should be limited to ≤ 5 weeks[15] (Table 4). Other options that can be used if other agents are not effective include atypical antipsychotics, such as quetiapine (25–100 mg/d), tiagabine (4–6 mg/d), gabapentin (100–600 mg/d), and pregabalin (150–300 mg/d).[6]

The Choosing Wisely campaign by the American Board of Internal Medicine Foundation does not recommend using benzodiazepines or other sedative–hypnotics in the elderly population as a first-line treatment for insomnia, agitation, or delirium because it may increase morbidity and mortality.[26] Additionally, several studies have demonstrated tolerance and worsening sleep quality with long-term hypnotic use.[2]

The American Academy of Sleep Medicine also recommends against a few medications prescribed to the older population due to the limited evidence supporting effectiveness; these medications include trazodone, tiagabine, diphenhydramine, melatonin, tryptophan, and valerian.[2,15] Hypnotics, such as benzodiazepines and nonbenzodiazepines (Z-drugs), should not be used for insomnia in the older population due to tolerance and dependency, as well as ataxia, falls, and cognitive impairment.[27] In addition,

Successful practices for a healthy sleep
Be consistent—wake up at the same time during the weekdays, weekends, and vacations.
Sleep 7–8 h every night.
Use the bed only for sleep and sex.
Avoid staying in bed if not asleep after 15–20 min.
Make the bedroom a comfortable and quiet place with a cool temperature.
Limit bright and blue light exposure in the evenings.
Stop using electronic devices 30–60 min before bedtime.
Cut back screen time 2 h before bedtime.
Avoid alcohol or a large dinner before bedtime.
Refrain from consuming caffeine 6–8 h before bedtime.
Exercise regularly during the day.
Decrease fluid intake before bedtime.
Adjust the air conditioning to reduce body temperature by 1°C (1.8°F) 2–3 h before bedtime to help fall asleep within 20 min.
The best sleeping temperature is between 15.6 and 20°C (60 and 68°F).
Eat a low-fat and low-added-sugar supper before bedtime.
If unable to fall asleep, leave the bed and do a quiet activity with low-light levels.
Try to avoid going to bed if you are not sleepy.
Manage stressors by writing them in a journal before bed.
Meditate to handle anxiety.
Try non-blue light creative activities such as puzzles, find-a-word, and coloring books to avoid overthinking.
Hum a favorite song because it calms the brain and vagus nerve. Humming doesn't even need to be out loud to get this benefit.

Table 2: Sleep hygiene education[2,11–13]

Intervention	Description
CBT-I	First-line treatment. Frequently involves 6–10 sessions of therapy focused on cognitive beliefs and behaviors associated with sleep disorders.
Sleep restriction therapy	Limit time in bed until sleep efficiency improves. Determine the average total sleep time (ATST) and add 30 min. Set up a wake-up and bedtime. For example, if the ATST is 6 h and the wake-up time is 6 AM, the bedtime will be midnight. It may be increased by 15 min weekly if feeling tired until sleeping better—no naps during the daytime.
Stimulus control therapy	Use the bed only to sleep. If not asleep, get out of bed and return only if sleepy—no reading or watching TV in bed. Avoid exceeding > 15 min in bed if not asleep.
Relaxation Techniques	Breathing exercises, paced diaphragmatic breathing (belly breathing), mediation, biofeedback, and progressive muscle relaxation. In addition, nose breathing, such as slow breathing with prolonged expiration and tiny breaths, has been shown to help with anxiety by decreasing psychological and physiological arousal.
Brief behavioral therapy for insomnia	A shorter form of CBT-I focused on improving circadian sleep regulation in fewer sessions.
Light therapy	Exposure to sunlight or a lightbox/visor with light intensity from 2000 to 10,000 lux. Use early morning light therapy for the delayed sleep phase and evening light therapy for the advanced sleep phase.

Table 3: Behavioral interventions[2,8,14]

CBT-I = cognitive behavioral therapy for insomnia.

Medication	Dosage (mg)	Mechanism of action	Possible interactions	Side effects	Literature regarding use in the elderly
Antidepressants					
Doxepin	3–6[6]	Highly selective H1 antagonist. Resynchronization of the circadian cycle.[6]	Acetylcholinesterase inhibitors, CNS depressants, alosetron, alpha-1, alpha-2 agonists, aspirin, beta-2 agonists, and haloperidol.[16]	Nephrotoxicity, GI upset, urinary retention, cardiac arrhythmia, orthostatic hypotension, bone marrow depression.[16]	In a study with 76 elderly patients, dosages at 1 mg, 3 mg, and 6 mg were all equally safe, but patients had increased success at 6 mg.[6]
Mirtazapine	7.5–30[6]	Blocks 5-HT_{2A}, 5-HT_{2C}, and $alpha_2$-adrenergic receptors; decreases neurotransmission at 5-HT_2 synapses; increases norepinephrine neurotransmission.[16]	Monoamine-oxidase inhibitors.[16]	Seizures, agranulocytosis, neuroleptic malignant syndrome, somnolence, increased appetite, weight gain, constipation, dizziness, xerostomia.[16]	Some small studies have shown that patients with Alzheimer's benefit from mirtazapine.[6] It may exacerbate hyponatremia or cause SIADH in the elderly. Monitor sodium closely.[17]
Trazodone	25–100[6]	Modulates effect on 5-HTA receptors.[6]	Monoamine-oxidase inhibitors, saquinavir or ritonavir.[16]	Cardiac arrhythmia, priapism, hypotension, serotonin syndrome.[16]	Although evidence is limited, a small randomized, double-blind trial showed subjective improvement in sleep. Another trial with AD patients showed safety at 50 mg dosage.[18]
Benzodiazepine					
Temazepam	7.5–20[19]	A positive allosteric modulator of the GABA-A receptor.[19]	CNS depressants.	GI upset, headaches, dreams, nightmares.[19]	A systematic review of 24 articles found that this medication was optimal for sleep maintenance insomnia in older adults.[19]
Non-benzodiazepines					
Eszopiclone	1–2[6]	Target the α-1 subunit of GABA-A receptor complexes to increase activation.[6]	CNS depressants, CYP3A4 inducers.[20]	Hepatotoxicity, dizziness, headache, excessive somnolence.[16]	In a meta-analysis with older adults, Eszopiclone and low-dose doxepin were superior for total subjective and objective sleep time compared to placebo.[19]
Zaleplon	5–10[41,42]	Target the α-1 subunit of GABA-A receptor complexes to increase activation.[6]	CNS depressants, CYP3A4 inducers.[21]	Hepatotoxicity, dizziness, headache, excessive somnolence.[16]	Not recommended due to increased falls, confusion, increased ED visits, hospitalizations, manual vacuum aspiration, and minimal improvement in sleep latency and duration.[17]
Zolpidem	2.5–5[6] Extended-release: 6.25[21]	Target the α-1 subunit of GABA-A receptor complexes to increase activation.[6]	CNS depressants, CYP3A4 inducers.[21]	Hepatotoxicity, dizziness, headache, excessive somnolence.[16]	In a double-blind trial with 205 older adults, zolpidem decreased sleep latency and increased total sleep time.[21]
Melatonin Receptor Agonists					
Ramelteon	8[6,22]	Activation of melatonin 1 and 2 receptors in the hypothalamus.[6]	CNS depressants, CYP1A2 inhibitors, CYP2C9 inhibitors, and inducers.[22]	Dizziness, somnolence, fatigue, nausea, worsened insomnia.[22]	In a large (n = 800) randomized, double-blind study, ramelteon increased total sleep time and decreased sleep latency in the elderly with ramelteon 4 mg.[23]

Table 4: Medications for chronic insomnia disorder in older adults (*Continued*)

Table 4: *Continued*

Medication	Dosage (mg)	Mechanism of action	Possible interactions	Side effects	Literature regarding use in the elderly
Tasimelteon	20[22]	Synthetic melatonin receptor agonist at melatonin type 1 and 2 receptors.[22]	CNS depressants, CYP1A2, CYP2C9 inhibitors, and inducers.[22]	Daytime somnolence, fatigue, dizziness, headache, nightmares.[22]	Approved for non–24-h sleep–wake cycle disorders in adults; however, insufficient data regarding safety in the elderly.[22]
Orexin receptor antagonists					
Lemborexant	5–10[44]	Orexin Receptor antagonist at OX1R and OX2R.[24]	CYP3A4 inhibitors and inducers.[24]	Daytime sleepiness, fatigue, dizziness, headaches, nightmares.[24]	In a study of 1006 participants ≥ 55 years old, lemborexant significantly improved sleep onset and maintenance compared with placebo and zolpidem.[24]
Suvorexant	5–20[6]	Orexin receptor antagonist at OX1R and OX2R.[25]	CYP3A4 inhibitors.[25]	Daytime somnolence, sleep paralysis, cataplexy, hypnagogic hallucinations.[25]	Insufficient evidence in the elderly due to its being a newer medication.[25]

AD = Alzheimer's disease; CNS = central nervous system; ED = emergency department; GABA = gamma-aminobutyric acid; GI = gastrointestinal; H1 = histamine H1 receptor; 5-HT = serotonin; OX1R = oxrexin 1 receptor; OX2R = oxrexin 2 receptor; SIADH = syndrom of inappropriate antidirueteic hormone secretion.

physicians should take caution in prescribing medications and check for adverse reactions and interactions with current medications. Polypharmacy is linked to falls, overdose, cognitive impairment, and death, particularly when prescribing ≥ 3 central nervous system (CNS)–active medications.[27]

OVER-THE-COUNTER MEDICATIONS AND COMPLEMENTARY ALTERNATIVE SUPPLEMENTS

Beyond prescribed medications, there have been noted off-label uses of common medicines for insomnia.[17] For example, diphenhydramine, even though it is strongly discouraged by the American Geriatric Society due to its risk of anticholinergic effects, is sometimes taken off-label for insomnia.[17] Another concern is that diphenhydramine, especially when combined with other anticholinergics, can increase the risk of confusion and falls in the elderly.[17]

Melatonin is a customarily used supplement for insomnia. Produced in the pineal gland naturally, synthetic compounds work to mimic the original.[28] Caution should be used with melatonin, as the U.S. Food and Drug Administration (FDA) does not regulate it, so preparations can vary greatly.[28] More studies need to be performed to evaluate its efficacy; however, there have been studies showing increased sleep quality and morning alertness in elderly adults > 55 years old.[28]

Valerian root is another usual complementary treatment for insomnia. Valerian root is effective in subjective, but not objective, parameters for sleep and can cause side effects, such as gastrointestinal upset, allergic reaction, headaches, and restlessness.[29] However, in a meta-analysis of 10 studies including > 1000 patients, valerian root had varying outcomes but was shown to have no adverse reactions and was safe for patients from 7 to 80 years old.[30]

Last, patients with mild insomnia and discomfort due to pain may benefit from over-the-counter acetaminophen before sleep.[2]

Circadian Rhythm Sleep–Wake Disorders

DEFINITION

A disparity between the endogenous circadian rhythm and the external environment (the social or physical activity) may lead to CRSWDs.[6,31]

EPIDEMIOLOGY

Factors influencing circadian rhythm include age, less exposure to light, food intake, and reduced activity.[6] About 20% of the elderly experience early morning awakening.[2,6,31]

DIAGNOSIS

Extrinsic CRSWDs include work shift disorder (due to reduced total sleep hours because of a change in work schedule) and jet lag disorder (when the endogenous circadian rhythm does not match the time zone).[6]

However, intrinsic CRSWDs are caused by alterations to the endogenous circadian rhythm system, which include delayed sleep–wake phase disorder, sleep occurring at least 2 hours after an expected social time, advanced sleep–wake phase disorder, irregular sleep–wake rhythm disorder, irregular sleep and awake episodes, and non–24-hour sleep–wake phase disorder.[6,31] Advanced sleep–wake phase disorder is most common in the elderly. Sleep onset is involuntary, occurring at least 2 hours before the expected time, and the awakening is spontaneous in the early morning.[6] If older adults try to sleep late, they still wake up early.

Comorbid conditions, like obstructive sleep apnea (OSA) and periodic limb movement disorder (PLMD), should be ruled out, as well as depression and bipolar disorder, because they can cause early morning awakening.[6,31]

NONPHARMACOLOGICAL MANAGEMENT

Nonpharmacological treatment includes light therapy and behavior therapy.[6] Patients are advised to stay in the dark during the early morning and remain outdoors or in intense light in the early evening to delay the circadian rhythm.[6,31] Sleep time should be advanced by 3 hours every 2 days until the desired time is achieved.[6]

PHARMACOLOGICAL MANAGEMENT

The pharmacological approach targets the melatonin receptors via a melatonin receptor agonist. Administration of supraphysiologic doses of melatonin agonists during specific time interval has been shown to phase shift the circadian clock and potentially promote sleep.[6,31] Although ramelteon (8 mg/d 30 minutes before going to bed and without food), and tasimelteon (20 mg/d just before going to bed also without food), can be used in the United States currently, only tasimelteon is approved for treating a CRSWD, namely, non–24 hour sleep–wake disorder.[2,6,31] In addition, ramelteon is the only nonbenzodiazepine receptor agonist approved by the FDA to treat sleep-onset insomnia.[6,31]

Dietary melatonin has not been evaluated or approved by the FDA to prevent or treat any disease or condition.[6,31] Its evidence includes limited efficacy and safety data, secondary to limited FDA regulation and substantial variability in potency, purity, and safety between available dietary supplements.[6,31]

Sleep-Related Movement Disorders

Sleep-related movement disorders include restless leg syndrome (RLS), PLMD, and REM sleep behavior disorder (RSBD).[6]

RESTLESS LEG SYNDROME

Definition

RLS is a neurological sensorimotor disorder that manifests as an uncomfortable, restless sensation that urges the leg(s) to move and usually happens at rest or at night.[6,32]

Epidemiology

Prevalence of RLS generally increases with age at a rate of 10% to 35% in patients < 65 years old. RLS is more common in females and is related to pregnancy, iron deficiency, uremia, neuropathies, and cardiovascular disease.[33]

Diagnosis

The diagnosis is clinical, including an overwhelming sensation of crawling, tingling, or pain that cause the limb to move to relieve the urge and the discomfort. It can be unilateral or bilateral.[33,34] The more the disease progresses, the more likely it will involve the arms and sometimes the phantom limb, causing problems initiating sleep. Symptoms are worse during rest or at night and tend to subside during the daytime.[33] Common comorbidities with RLS include depression, anxiety, attention deficit hyperactivity disorder, and kidney dialysis.[6,32]

Nonpharmacological management

Nonpharmacological treatment with lifestyle changes and dietary modifications is usually helpful in controlling RLS.[33,34] Medications such as antipsychotics, antiemetics, antidepressants, beta-blockers, anticonvulsants, and lithium are known to exacerbate RLS and should be avoided.[2,6,33] Dysfunction of the dopaminergic system and iron deficiency is thought to be involved in its pathogenesis.[33]

The American Academy of Neurology recommends ferrous fumarate supplementation with vitamin C in patients with RLS and serum ferritin of < 75 mcg/L and IV iron replacement if severe symptoms are present.[6,33] Workup for the causes of iron deficiency should be initiated, and commonly associated diseases like neuropathies, cardiovascular disease,

hypertension, depression, and diabetes should be ruled out.[6,10]

Pharmacological management

Pharmacological treatment: non–ergot-based dopamine agonists (ropinirole, pramipexole, and rotigotine) are the first-line treatments of primary RLS and have high levels of evidence for efficacy and safety.[6,10] Second-line treatments include the dopamine precursors (levodopa) and ergot-based dopamine agonists (eg, cabergoline).[6,10]

PERIODIC LIMB MOVEMENT DISORDER

Definition

PLMD is defined as periodic episodes of sleep-related phenomena that are characterized by involuntary repetitive, stereotypical movements of the limbs.[2,35]

Epidemiology

The prevalence of PLMD is up to 80% in patients with RLS and up to 4% to 11% in the older adult population. The prevalence of both conditions increases as the patient ages.[35,36]

Diagnosis

Repeated jerky leg movement mimics the Babinski sign (extension of the big toe, dorsiflexion of toes and ankle, and often flexion of the knee and hip).[35,36] In addition, PLMD is associated with a high cardiovascular and cerebrovascular risk as they represent the motor manifestation of an increased sympathetic overactivity, which causes nocturnal elevated blood pressure and heart rate. PLMD also can be seen without comorbid sleep or medical disorders. Patients often have a non-rested sleep and daytime fatigue.[35]

PLMD is commonly associated with other sleep disorders and other medical conditions, including congestive heart failure, diabetes, hepatic and renal disease, migraine, alcohol dependence, and several neurologic and psychiatric disorders.[35,36] Some medications can trigger PLMD, including antidepressants and lithium.[35]

Nonpharmacological management

Nonpharmacological treatment aims to avoid certain medications that may aggravate PLMD, including antidepressants like mirtazapine, venlafaxine, sertraline, fluoxetine, and amitriptyline.[6,35] Caffeine-containing products should also be avoided. Treating underlying diseases like iron deficiency anemia in cases associated with RLS is also helpful.[35]

Pharmacological management

Dopaminergic medications used to treat RLS, such as pramipexole, ropinirole, rotigotine, and other drugs like gabapentin and pregabalin, will reduce the periodic limb movements in patients with PLMD.[36] Based on a few small studies, clonazepam 1 mg at bedtime, melatonin 3 mg 30 minutes before bedtime, and valproate 150–600 mg at bedtime may improve sleep quality.[35,36]

REM SLEEP BEHAVIOR DISORDER

Definition

In RSBD, the loss of muscle atonia usually happens during normal REM sleep. It commonly results from dysfunction of the brain stem circuits responsible for maintaining normal REM sleep atonia and suppressing behaviors during REM sleep.[6] Such abnormal behaviors happening during REM sleep may cause injury or sleep disruption.

Epidemiology

The prevalence of RSBD is relatively low, with a rate of ~0.5% in the general population and up to 5% to 7% in the elderly population. More than 70% of patients with RSBD will develop a neurodegenerative disease within 12 years of their diagnosis.[6,34]

Diagnosis

The clinical diagnosis includes extreme motor behaviors like talking, kicking, biting, and punching in response to violent dreams. It often happens with intense emotional stress or during withdrawal from sedatives or alcohol, but it can also occur in healthy individuals. In addition, the eyes remain closed, and the forceful movement stays in the surrounding environment.[6,34] It is essential to ask patients to rate their sleep quality, helping physicians to triage and diagnose untreated RSBD earlier. Polysomnography showing a loss of normal skeletal muscle atonia during REM sleep is required for diagnosis.[37]

Idiopathic RSBD is linked to neurodegenerative disorders (eg, Parkinson's disease, Lewy body dementia, multiple system atrophy). It is often an

early manifestation of neurodegeneration occurring in the brain stem before spreading to other CNS regions.[6,34]

The average conversion time from idiopathic RSBD (iRSBD) to an underlying neurodegenerative disease that accumulates alpha-synuclein in the nerve cells, also called synucleinopathy, is 4–9 years from iRSBD diagnosis and 11–16 years from symptom onset.[38]

Approximately 33% to 46% of patients with Parkinson's disease have RSBD.[39] Risk factors for iRSBD include prior head injury, occupational farming, pesticide exposure, low education level, smoking, ischemic heart disease, and inhaled corticosteroid use.[38] Patients with Parkinson's disease and RSBD have been shown to have poorer cognitive performance, as they are more likely to be diagnosed with mild cognitive impairment and subjective cognitive decline.[39] In longitudinal studies, Parkinson's disease is associated with worsening sleep maintenance throughout the illness.[40] In addition, patients with Parkinson's disease with insomnia often also have more severe depression, worse balance problems, and gait deficiencies.[40] Therefore, there is an increased risk of conversion to neurodegenerative disease in patients ≥ 50 years old with mild cognitive impairment signs and sleep dysregulation.[38] For any patient with these risk factors, a healthy lifestyle focusing on smoking cessation and alcohol abstinence is recommended. In addition, treatment of comorbidities, such as hypertension, diabetes, anxiety, and depression can also be helpful.[38]

Unfortunately, there is currently insufficient evidence to support the pharmacological treatment of these patients, and more research is needed.[38]

Nonpharmacological management

Nonpharmacological treatment focuses on environmental modification to prevent harm and injuries.[6,37] Bed alarms, sitters, treating underlying causes, and discontinuing medications that exacerbate RSBD, like antidepressants, are the mainstay modalities.[6,34] Offending medications include selective serotonin reuptake inhibitors, serotonin–norepinephrine reuptake inhibitors, tricyclic antidepressants, monoamine-oxidase inhibitors, beta-blockers (atenolol, bisoprolol), tramadol, and cholinesterase inhibitors.[6,34]

Pharmacological management

Low-dose clonazepam (0.5–1 mg/d) is the preferred treatment for RSBD, given 30 minutes before bedtime, but with cautious use in patients with sleep apnea.[37] Zopiclone is similar to clonazepam in treating RSBD symptoms, with a much shorter half-life.[34,37] High-dose melatonin (3–15 mg) at bedtime (the median effective dose is 6 mg) is an alternative to clonazepam that is well tolerated and has fewer side effects compared to clonazepam.[6,34]

Dopaminergic agonists like pramipexole (0.1–0.5 mg) and acetylcholinesterase inhibitors (rivastigmine) may be considered as second-line agents in treatment-refractory patients.[6,34]

Sleep-Breathing Disorders

DEFINITION

SBDs comprise a group of conditions that include OSA, primary or secondary central sleep apnea (CSA), nonobstructive hypoventilation, hypoxemia disorders secondary to pulmonary parenchymal, and vascular.[4] In addition, neuromuscular or chest wall disorders, periodic high-altitude breathing, and Cheyne–Stokes respiration can be causes of SBDs.[6]

EPIDEMIOLOGY

SBD prevalence is up to 34% in 30- to 70-year-old men, 17% in 30- to 70-year-old females, and 23.9% in 65- to 100-year-old patients overall.[6]

DIAGNOSIS

In OSA, the most common SBD, sleep arousal and arterial hypoxia happen due to intermittent partial or complete closure of upper airways, resulting in respiratory cessation for 20–30 seconds after a brief gasping. Apnea is a complete cessation of breathing, while hypopnea is a reduction in the airflow by 30% with ≥ 4% oxygen desaturation.[6,10]

The apnea–hypopnea index (AHI) is the total number of apnea and hypopnea episodes per hour of sleep. An AHI of > 5 is required to diagnose an SBD.[6,10] CSA presents as cessation of breathing ≥ 5 times during sleep due to loss of ventilatory drive. Patients usually have wide neck circumference, high body mass index, low soft palate, large tongue, uvula, and tonsils.[10]

OSA is caused by periodic narrowing or obstruction of the pharyngeal airway during sleep due to anatomical (structural) or neuromuscular (nonstructural) elements. Pharyngeal structures such as

enlarged adenoids, enlarged tonsils, relaxed tongue, abnormal lower jaw position, and fatty tissue surrounding the pharynx can adversely affect airway diameter and contribute to airway blockage or collapse.[41]

A neuromuscular response stimulates the dilator muscles in the pharynx to prevent airway obstruction. The genioglossus muscle is the biggest dilator of the upper airway. Its anatomy includes slow-twitch (type I) and fast-twitch (type II) muscle fibers. Type I fibers produce energy aerobically and are essential to sustain prolonged duration endurance activity. The type II fibers, however, are involved in short, powerful activity.[41]

In patients with OSA, the genioglossus muscle has been found to have a more notable proportion of fast-twitch muscle fibers and a reduced proportion of slow-twitch muscle fibers, causing a loss of muscle tone as a person enters sleep.[41]

Untreated OSA can be a leading cause of cardiovascular diseases, including coronary artery disease, stroke, and atrial fibrillation.[10] CSA is commonly seen in congestive heart failure. Its presence increases mortality in patients with heart failure. It is also seen in healthy individuals with central neural lesions and at high altitudes.[42]

OSA must be addressed and treated due to its association with underlying vascular disease, particularly in the older adult population. Myocardial infarction and stroke occur at higher rates in patients with OSA and both are leading causes of death in the elderly.[20,43] In addition, OSA increases the risk of cardiovascular disease through intermittent hypoxia and hypercapnia, inflammation, and activation of the sympathetic nervous system.[20,43] A decade-long cohort study found that hypoxia with SpO2 < 90% in patients with OSA was the single most significant factor in cardiovascular events.[43]

Furthermore, anxiety and falling asleep are usually associated with an untreated SBD, not primarily because of a high body mass index but, rather, due to the ability or not to breathe easily through the nose during the day and night (tongue up in a resting position on the roof of the mouth). Consequently, evaluating a weak tongue or tongue tie in patients with suspected SBD is fundamental.[44]

Most health care professionals use the "stick your tongue out" test. However, the better test is "can you open your mouth wide and hold your tongue flat against the roof of your mouth for several seconds."[44] The worsened ability of the tongue to perform specific movements, also known as praxia lingual, is associated with an increased risk of SBD and appears to have an early onset in life.[44]

NONPHARMACOLOGICAL MANAGEMENT

In mild-to-moderate SBD, AHI 5–15, positional therapy (sleep on the lateral side instead of in the supine position), an oral appliance, and weight loss are recommended.[4,10] Smoking cessation, avoiding alcohol and caffeine, and managing comorbidities are strongly recommended. In moderate-to-severe SBD, AHI > 15, a positive airway pressure device is the gold standard therapy; continuous positive airway pressure is preferred.[6,10] Bilevel positive airway pressure should be used if continuous positive airway pressure is not tolerated or the positive airway pressure pressure needed is < 20 cmH2O. Weight loss and positional therapy should be used. Sedatives and hypnotics should be avoided in patients with SBD.[6,10]

When the SBD originates from a weak tongue, a device called eXciteOSA can be used.

The eXciteOSA is a rechargeable mouthpiece, recently approved by the FDA, which fits on the tongue and delivers neuromuscular electrical stimulation to the genioglossus muscle to increase muscle endurance to promote airway patency.[41]

PHARMACOLOGICAL MANAGEMENT

Stimulants may be used if daytime sleepiness persists even with positive airway pressure.[6,42] Optimizing heart failure therapy may improve CSA. Nocturnal oxygen supplementation and theophylline have been shown to decrease the severity of CSA.[42]

Conclusion

Sleep disorders in the elderly are a common but underdiagnosed condition.[3] Most health care professionals do not know what to recommend for patients with sleep disorders and usually avoid discussing this, which is why SBDs often go untreated for ~3–8 years before diagnosis.[45] Therefore, the effects on sleep from age-related physiological changes and possible adverse effects of medications, medical conditions, and mental health disorders should be considered in older adults.[3]

In this article, the authors discussed differential diagnosis, clinical diagnosis, how to provide

comprehensive sleep hygiene education, and the different nonpharmacological and possible options for pharmacological treatment. In addition, comprehensive assessment and appropriate management of sleep disorders, including associated comorbidities, may prevent cognitive decline and improve the quality of care and health care outcomes in the older adult population.

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