

Evidence-Based Recommendations for the Assessment and Management of Sleep Disorders in Older Persons

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Sleep-related disorders are most prevalent in the older adult population. A high prevalence of medical and psychosocial comorbidities and the frequent use of multiple medications, rather than aging per se, are major reasons for this. A major concern, often underappreciated and underaddressed by clinicians, is the strong bidirectional relationship between sleep disorders and serious medical problems in older adults. Hypertension, depression, cardiovascular disease, and cerebrovascular disease are examples of diseases that are more likely to develop in individuals with sleep disorders. Conversely, individuals with any of these diseases are at a higher risk of developing sleep disorders. The goals of this article are to help guide clinicians in their general understanding of sleep problems in older persons, examine specific sleep disorders that occur in older persons, and suggest evidence- and expert-based recommendations for the assessment and treatment of sleep disorders in older persons. No such recommendations are available to help clinicians in their daily patient care practices. The four sections in the beginning of the article are titled, Background and Significance, General Review of Sleep, Recommendations Development, and General Approach to Detecting Sleep Disorders in an Ambulatory Setting. These are fol-

lowed by overviews of specific sleep disorders: Insomnia, Sleep Apnea, Restless Legs Syndrome, Circadian Rhythm Sleep Disorders, Parasomnias, Hypersomnias, and Sleep Disorders in Long-Term Care Settings. Evidence- and expert-based recommendations, developed by a group of sleep and clinical experts, are presented after each sleep disorder. *J Am Geriatr Soc* 57:761–789, 2009.

BACKGROUND AND SIGNIFICANCE

Sleep-related disorders are common in the general adult population, and as the population ages, the prevalence of these disorders increases. A common misconception of clinicians and the public is that this increased prevalence is a normal and expected phenomenon of aging, but this higher prevalence of sleep disruption is often the result of the presence of medical and psychosocial comorbidities in this population. The complicated multifactorial interactions that generate sleep disorders in older individuals pose important challenges to clinicians. Furthermore, many clinicians are unaware of the seriousness and potential morbidity associated with sleep problems in older people, distinct from the morbidity of concurrent disorders. As a result, these issues are often underinvestigated or completely ignored.¹

Because of the high prevalence, complexity, and health implications associated with sleep-related disorders in older individuals, increasing attention is now being focused on this topic. For example, a recent publication has recommended that sleep problems be approached as a “multifactorial geriatric syndrome.”²

Of major clinical concern is the strong bidirectional relationship between sleep disorders and serious medical problems in older persons. Individuals with sleep disorders are more likely to develop hypertension, depression, cardiovascular, and cerebrovascular disease. Conversely, individuals with any of these diseases are at higher than normal risk of developing sleep problems.^{3,4}

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Older individuals consider quality sleep to be an essential part of good health. A Gallup survey of more than 1,000 Americans aged 50 and older (43% of whom were aged 65 and older) found that 80% answered “a great deal” when asked whether sleep was important for healthy aging. In the same survey, and contrary to the myth that older adults need less sleep, 45% believed that they required more sleep than when they were younger, and 25% believed they had a sleep “problem.”⁵

The goal of this article is not to present an exhaustive and comprehensive review of sleep and sleep disorders in older persons. Rather, it presents an overview of sleep disorders and suggests appropriate evidence-based recommendations for assessing and treating sleep disorders in the older adult population. Professionals with expertise in sleep disorders and in the clinical care of older people have developed these recommendations.

As in many areas of clinical research, older persons are often poorly represented (or specifically excluded) in clinical sleep studies. Thus, there are fewer data available from randomized controlled trials for this population than for the general adult population. Nevertheless, given the importance of the subject, and the opportunity for successful intervention, it is prudent and timely to propose recommendations based upon expert consensus of current evidence. Although there have been a number of publications aimed at clinicians concerning sleep and sleep disorders in the adult and older adult populations, there are currently no recommendations for systematically approaching the assessment, treatment, and follow-up of sleep disorders in the older adult population.^{2,6–11}

In developing these recommendations, the authors were cognizant of a number of important themes: (1) the tremendous heterogeneity of the older adult population and thus the critical importance of individualization of assessment and therapy; (2) the limited amount of time clinicians have to spend with each patient, making lengthy assessments for sleep problems unrealistic; (3) the body of knowledge regarding the approach to assessment and treatment of sleep disorders that clinicians need to possess; (4) the role of sleep specialists in this process and the importance of recognizing when and where to refer; and (5) the frequent presence of comorbidities and multiple medication usage in this population of patients, requiring a careful approach and meticulous follow-up.

Sleep-related problems in the acute care hospital setting will not be addressed in this article. Such problems have received less attention in research studies than sleep problems in outpatients, and the state of knowledge concerning these conditions is inadequate to make recommendations with a reasonable level of confidence, so this article will focus on chronic problems with sleep in older persons in the outpatient and long-term care settings.

GENERAL REVIEW OF SLEEP

Major physiological changes occur in the context of aging. One such change that can be problematic for many older adults is the often profound change of the daily sleep–wake cycle.

Sleep is composed of two different physiological states: rapid eye movement sleep (REM) and non-rapid eye move-

ment sleep (NREM). NREM is further divided into four stages. Stage 1 is the lightest stage of sleep. Stage 2 sleep has a higher arousal threshold and is the stage in which most time sleeping is spent. Stages 3 and 4 are collectively referred to as deep sleep, delta sleep, or slow-wave sleep, (based upon their characteristic electroencephalographic profiles) and are associated with a high arousal threshold. Recently, the American Academy of Sleep Medicine has revised this classification slightly, into three NREM stages (Stages N1, N2, and N3, with N3 combining the traditional Stages 3 and 4).¹² Sleep typically occurs in approximately 90-minute cycles of NREM/REM, although more Stage 3/4 sleep occurs in the first half of the night, and more REM sleep takes place in the second half. Awakenings that may be extremely brief or of prolonged duration can interrupt this sleep pattern.

A complex interaction between a time awake–dependent increase in homeostatic sleep drive and a circadian wakefulness drive that typically reaches its maximum in the evening regulate the sleep–wake pattern. Normally, the homeostatic sleep drive and circadian wakefulness drive are both high in the evening, but as homeostatic sleep drive continues to build and circadian wakefulness drive declines, sleep is initiated.

A wide variety of physiological, psychological, and environmental factors can influence this normal sleep–wake process. The most striking change in sleep patterns in older adults is the repeated and frequent interruption of sleep by long periods of wakefulness, possibly the result of an age-dependent intrinsic change in the interaction between the sleep homeostatic and circadian arousing processes that control sleep.¹³ Other age-dependent changes in sleep include decreased total sleep time (TST), reduced sleep efficiency (time asleep as a percentage of time in bed), decreased slow-wave and REM sleep, and increased Stage 1 and 2 sleep. An increased incidence of napping or falling asleep during the day accompany these age-dependent changes in nocturnal sleep.^{14–16} Aging is also associated with a tendency to fall asleep and awaken earlier and to be less tolerant of phase shifts in the sleep–wake schedule such as those associated with jet lag and shiftwork.¹⁷ These changes also suggest age-dependent alterations in regulation of the circadian sleep–wake cycle.

When the sleep of individuals who may be considered to be “optimally aging” is examined, and age-related medical and psychiatric comorbidities are controlled for, it appears that most age-dependent sleep changes occur in early and middle adulthood (aged 19–60).¹³ Further age-dependent sleep changes after age 60 are modest at most, assuming that the individual is in good health.¹³ The presence of medical and psychiatric illnesses is associated with exacerbations of age-dependent sleep disruption.^{13,18–20} Nevertheless, it is important to recognize that such sleep disturbance in the presence of comorbidities is not necessarily simply a symptom of the comorbid condition(s) but may represent an independent problem that may benefit from treatment.^{21,22}

In addition to the effect of age-dependent sleep changes and age-associated comorbidities, common primary sleep disorders such as insomnia, obstructive sleep apnea (OSA), and restless leg syndrome (RLS), can further adversely affect the sleep of older adults. Epidemiological studies

have consistently shown that the prevalence of sleep complaints and sleep disorders grows steadily with advancing age.²³ As many as 57% of older adults complain of significant sleep disruption, 45% have periodic limb movements of sleep (PLMS), 29% suffer from insomnia, 24% have OSA, 19% complain of early morning awakening, and 12% have RLS.^{24–28}

Other factors such as prescription and over-the-counter medications; social drug use; and psychosocial, behavioral, and environmental factors, including poor sleep hygiene (the behaviors and environmental factors that can improve or worsen sleep), can further contribute to sleep problems.^{29,30} Any or several of these factors can adversely affect the sleep of an older adult.³⁰ In older adults, these sleep disturbances and increases in daytime sleepiness can have a significant negative effect not only on quality of life, but also on morbidity and mortality.^{29,31,32}

Although sleep disturbances can have profound health implications, they may also be situation specific. For example, sleep disturbances in a community-dwelling older adult who is aging optimally are likely to differ from those experienced by older adults in an acute hospital setting or in long-term care, particularly in people with dementia who can neither describe their symptoms nor engage as actively in treatment.

In 2006, the Institute of Medicine released a report entitled “Sleep Disorders and Sleep Deprivation: An Unmet Public Health Problem” that recognized the wide range of deleterious health and safety consequences of disturbed and inadequate sleep.³³ The report called for greater awareness among healthcare professionals about the physiology of healthy sleep and sleep disorders across the life span, as well as for the development and implementation of programs to promote the early diagnosis and treatment of sleep disorders.

The last 10 years have seen significant and rapid advances in the ability to diagnose and treat sleep disorders in the general and older adult populations using behavioral and pharmacological approaches. The recommendations presented below for the effective diagnosis and treatment of sleep disorders in this population are offered in the spirit of the Institute of Medicine report and the considerable recent progress made in the effective recognition and treatment of sleep disorders in older adults.

RECOMMENDATIONS DEVELOPMENT PROCESS AND METHODS

Two 2-day conferences involving a multidisciplinary group of sleep experts and clinicians representing major geriatric interest groups and societies were held at the International Longevity Center (ILC, New York) in November 2005 and December 2006. Participants in those conferences uniformly agreed that the time was appropriate to bring together representatives from national sleep organizations, geriatric clinical organizations, and other clinical organizations with an interest in geriatric care to develop and publish evidence and expert consensus-based recommendations for the assessment and management of sleep disorders in older persons. The development of such recommendations seemed especially important given the recent increasing attention in the literature to sleep prob-

lems in older adults and the absence of existing recommendations for this population.

A broad national group of 13 such organizations was assembled in 2007. In December of that year, a third conference at the ILC brought together representatives from these organizations, as well as other sleep experts and expert clinicians. Before the meeting, thought leaders in the field were asked to prepare presentations on the major disorders related to sleep disturbances in older adults based upon their prevalence, potential morbidity and mortality, and possibility for response to therapy. The attendees subsequently identified the sleep disorders to be included, and the authors were chosen because of their internationally recognized expertise in each particular sleep disorder. They determined that the recommendations paper should be a multiauthored document that would be submitted to a peer-review journal for publication and that participating organizations would not be asked to provide review before recommendation publication.

In addition to the references cited by the authors of the individual sections, a formal literature search and review was performed for each of the sleep disorders and for the section concerning the specific sleep problems encountered in the long-term care setting. The search focused on randomized controlled trials (RCTs), meta-analyses, and systematic reviews. Nonrandomized clinical trials and controlled clinical trials were also included given the low volume of RCTs in older adults with certain sleep disorders (e.g., parasomnias, hypersomnias). More than 11,600 citations were identified from sources including PubMed, the Cochrane Database of Systematic Reviews, the National Guideline Clearinghouse, and the Centre for Reviews and Dissemination/Health Technology Assessment databases using key word searches for each condition and intervention of interest. Panel members selected and screened approximately 1,700 abstracts for these citations for evidence-based content. Selected full-text, English-language papers were summarized in evidence tables for review by all of the primary authors. The number of evidence-based studies on patients aged 65 and older is limited in some conditions, and consensus can vary as to whether studies of younger subjects can be extrapolated to older subjects.

The primary author proposing a specific recommendation initially assigned the quality and strength of evidence for each recommendation (Table 1). The coalition panel then reviewed all evidence designations, and final designations were decided according to consensus. This assessment methodology has been widely used in previous guidelines.³⁴

DETECTING SLEEP DISORDERS IN AN AMBULATORY SETTING: GENERAL APPROACH

The best method for detecting sleep–wake problems in ambulatory older people is simply to inquire about sleep on a regular basis.

The clinician may do this initially during the patient visit. An alternative is to allow a staff member to administer a brief sleep questionnaire before or during routine vital signs assessments, perhaps before the first visit in all new patients and then at least semiannually in returning patients. The answers to these questions will then be immediately available to the clinician to review or expand upon if

Table 1. Quality of Evidence

Level and Strength of Evidence	Description
Level	
I	Evidence from at least one properly designed randomized controlled trial
II	Evidence from at least one well-designed clinical trial without randomization, from cohort or case-controlled analytical studies, from multiple time-series studies, or from dramatic results in uncontrolled experiments
III	Evidence from respected authorities, based on clinical experience, descriptive studies, or reports of expert committee
Strength of evidence	
A	Good evidence to support the use of a recommendation; clinicians should do this all the time
B	Moderate evidence to support the use of a recommendation; clinicians should do this most of the time
C	Poor evidence to support or reject the use of a recommendation; clinicians may or may not follow the recommendation
D	Moderate evidence against the use of a recommendation; clinicians should not do this
E	Good evidence against the use of a recommendation; clinicians should not do this

necessary. If a bed partner is with the patient, he or she should assist with the answers.

The following 12 questions can serve as the initial assessment regarding sleep.

- (1) What time do you normally go to bed at night? What time do you normally wake up in the morning?
- (2) Do you often have trouble falling asleep at night?
- (3) About how many times do you wake up at night?
- (4) If you do wake up during the night, do you usually have trouble falling back asleep?
- (5) Does your bed partner say (or are you aware) that you frequently snore, gasp for air, or stop breathing?
- (6) Does your bed partner say (or are you aware) that you kick or thrash about while asleep?
- (7) Are you aware that you ever walk, eat, punch, kick, or scream during sleep?
- (8) Are you sleepy or tired during much of the day?
- (9) Do you usually take one or more naps during the day?
- (10) Do you usually doze off without planning to during the day?
- (11) How much sleep do you need to feel alert and function well?
- (12) Are you currently taking any type of medication or other preparation to help you sleep?

If symptoms of a sleep complaint are suggested in this initial screening, further questions may be appropriate to ask when taking a sleep history.³⁵

- (1) Do you have the urge to move your legs or do you experience uncomfortable sensations in your legs during rest or at night?

- (2) Do you have to get up often to urinate during the night?
- (3) How much physical activity or exercise do you get daily?
- (4) Are you exposed to natural outdoor light most days?
- (5) What medications do you take and at what time of day and night?
- (6) Do you suffer any uncomfortable side effects from your medications?
- (7) How much caffeine (e.g., coffee, tea, cola) and alcohol do you consume each day and night?
- (8) Do you often feel sad or anxious?
- (9) Have you suffered any personal losses recently?

The patient's responses should indicate how to proceed with any further history, focused physical examination, or laboratory investigations. Specific questions, examinations, laboratory tests, procedures, and possible referrals are discussed in more detail in the sections concerning the particular sleep disorders. A flow diagram (Figure 1) may be helpful in identifying and treating sleep complaints in older ambulatory individuals.

INSOMNIA

Definition

Insomnia is defined as a complaint of disturbed sleep in the presence of an adequate opportunity and circumstance for sleep. The complaint may consist of difficulty initiating sleep, difficulty maintaining sleep, waking up too early, or nonrestorative or poor-quality sleep. For the diagnosis of an insomnia disorder to be made, the difficulty with sleep must have a negative effect on daily function.³⁶

Insomnia is classified as primary or comorbid. Primary insomnia implies that no other cause of sleep disturbance has been identified. Comorbid insomnia is more common and is most often associated with psychiatric disorders (e.g., depression, anxiety, or substance use), medical disorders (e.g., cardiopulmonary disorders, neurological disorders, or chronic somatic complaints that result in sleep disruption), medications, and other primary sleep disorders (e.g., OSA or restless legs).^{4,24} Comorbid insomnia does not suggest that other condition(s) "cause" insomnia but rather that insomnia and the other condition(s) co-occur and may each warrant clinical attention and treatment.

Prevalence

Although the prevalence of insomnia in the general population has been estimated at 10% to 20%, studies in older adults have found higher frequencies. In a study of more than 9,000 adults aged 65 and older, 42% of participants had difficulty falling asleep and staying asleep, with a higher prevalence found in older adults with poor health and who were taking medications for a variety of medical problems.²⁴ Participants who were depressed were 2.5 times as likely to report insomnia, and those with respiratory symptoms were 40% more likely to do so. The finding that a considerable proportion of sleep complaints in older people may be associated with chronic disease and other health problems is corroborated in other reports.^{19,20,37}

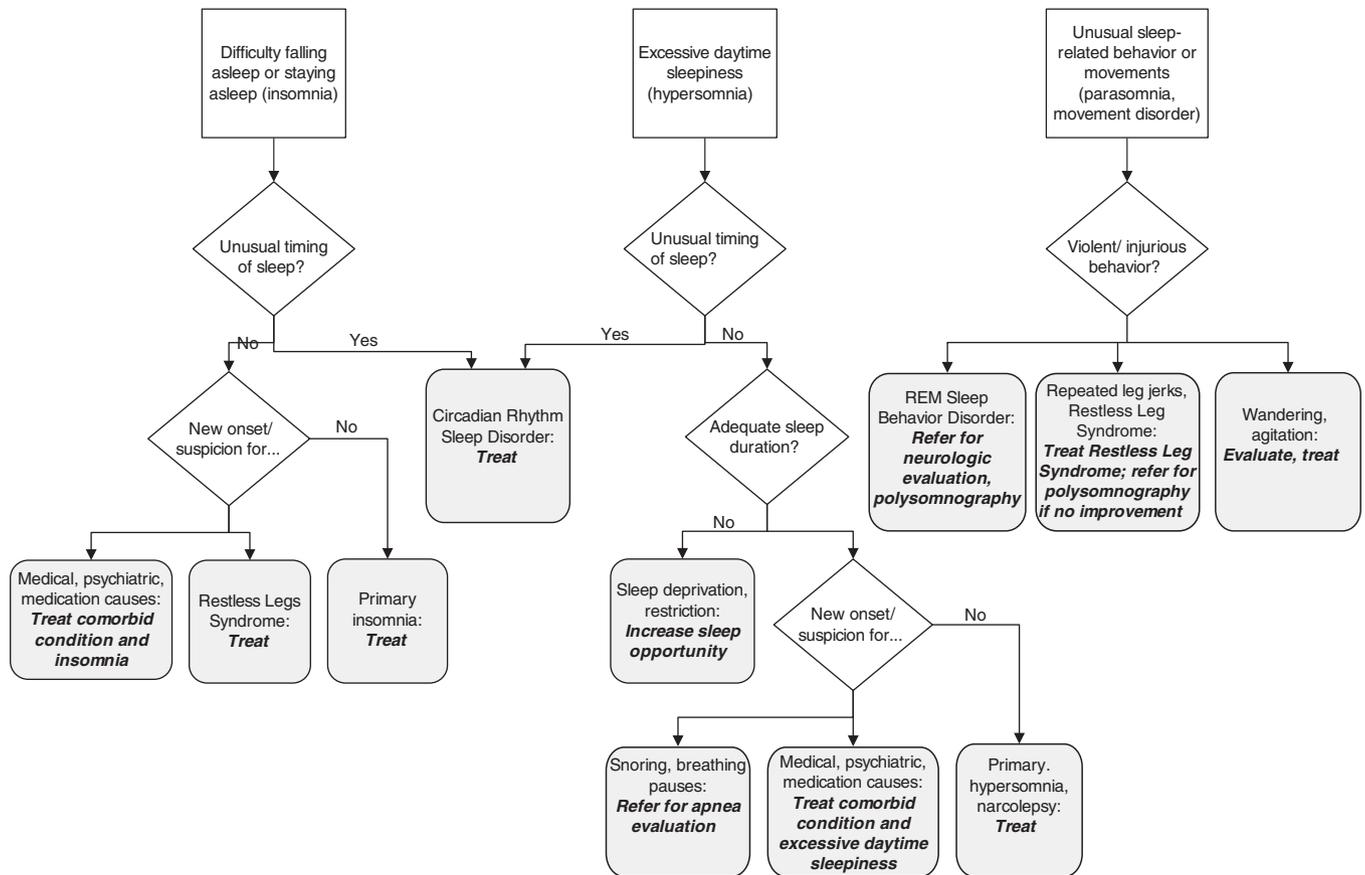


Figure 1. Diagnostic algorithm for sleep disorders in older persons.

Consequences of Insomnia

Insomnia in older adults is associated with significant morbidity and mortality. Older adults with difficulty sleeping report poorer quality of life and more symptoms of depression and anxiety.³⁸⁻⁴¹ Napping during the day and sleeping less than 7 hours a night have been associated with a greater risk of falls.⁴² Cognitive decline, difficulty ambulating, difficulty with balance, and difficulty seeing are also associated with poor sleep, even after controlling for medication use.⁴³⁻⁴⁷ The relative risk for greater mortality in older adults has been associated with taking more than 30 minutes to fall asleep and with a sleep efficiency (time asleep as a percentage of time in bed) of less than 80%.⁴⁸

Comorbidities

As mentioned, much of the insomnia seen in older adults is likely to be comorbid with psychiatric illness. It has long been known that depression and insomnia are associated and that the presence of depressed mood may predict insomnia. Many studies have suggested that untreated insomnia is a risk factor for recurrent and new-onset depression.⁴⁹⁻⁵³

Older adults with medical conditions are also more likely to complain of difficulty sleeping. In the 2003 National Sleep Foundation survey of adults aged 65 and older, subjects with more medical conditions, including cardiac and pulmonary disease, reported more sleep complaints and more dissatisfaction with sleep.⁴ Pain associated with osteoarthritis, cancer, or diabetes mellitus; shortness of

breath due to chronic obstructive pulmonary disease or congestive heart failure; nocturia due to an enlarged prostate; and neurological deficits related to cerebrovascular accidents or Parkinson’s disease have all been associated with sleep complaints and insomnia.⁵⁴⁻⁵⁹ Not only do older adults with medical and psychiatric problems have more insomnia, but those with insomnia are also more likely to have medical problems, including heart disease, cancer, high blood pressure, neurological disease, breathing problems, urinary problems, diabetes mellitus, chronic pain, and gastrointestinal problems, even after controlling for depression and anxiety.³

Medications

Many older adults regularly take multiple medications. Medications used to treat various underlying chronic medical and psychiatric conditions also contribute to sleep disruptions, including beta-blockers, bronchodilators, corticosteroids, decongestants, and diuretics, as well as other cardiovascular, neurological, psychiatric, and gastrointestinal medications. Medications used to treat depression, such as selective serotonin reuptake inhibitors (SSRIs) and serotonergic and noradrenergic reuptake inhibitors may also cause or exacerbate insomnia.⁶⁰ In addition to prescription medications, older adults often take over-the-counter preparations that can cause or exacerbate sleep disturbances. Examples include cough and cold medications, especially those containing pseudoephedrine or phenylpropanolamine, caffeine-containing drugs (e.g., combinations of acetaminophen

or aspirin and caffeine), and drugs containing nicotine (e.g., nicotine gum and transdermal patches).

Cigarette smoking and coffee consumption themselves can impair sleep as well.

Assessment of Insomnia

The diagnosis of insomnia in older adults requires that the patient have difficulty falling asleep or staying asleep for at least 1 month and that impairment in daytime functioning result from difficulty sleeping. The differential diagnosis of chronic insomnia is broad, especially in older adults with many medical and psychosocial comorbidities who are also taking multiple medications. Therefore, a thorough clinical history is essential, especially with regard to prescription and nonprescription drugs and remedies and any comorbid conditions or diseases. It is important to establish whether the individual's insomnia is primary or comorbid, although it is not uncommon for older people to have more than one etiological contributing factor responsible for the insomnia.

A focused physical examination, based upon the responses from the clinical history, is also essential. Any laboratory evaluation should follow logically from the results of the history and physical examination.

Treatment of Insomnia

Behavioral Treatment

Behavioral treatments have been shown to be highly effective in the treatment of insomnia in all age groups.^{36,61} Cognitive behavioral therapy for insomnia (CBT-I) has been shown to be most effective. CBT-I combines different behavioral treatments, including sleep hygiene instruction, stimulus control, and sleep restriction, with cognitive restructuring.^{62–64} In CBT-I trials in older adults, insomnia not only resolved, but the effect was also sustained for up to 2 years.⁶²

A number of single-modality behavioral and other nonpharmacological approaches have been used to treat and manage insomnia in all age groups. These include relaxation therapy and imagery, stimulus control, sleep restriction, sleep compression, improved sleep hygiene, sleep education, and cognitive therapies. Exercise and physical activity, massage therapy, chronotherapy, and light therapy are also used. Although any of these may be beneficial for older adults with insomnia, two approaches have met evidence-based criteria for efficacy: sleep restriction–sleep compression therapy and multicomponent cognitive-behavioral therapy.⁶⁵

Sleep Hygiene and Sleep Education

Sleep hygiene and sleep education can be useful when used together with other modalities but are usually not adequate by themselves for the treatment of severe, chronic insomnia. Addressing sleep hygiene entails examining sleep habits, behaviors, and environmental factors that can have an effect on sleep. A practitioner can educate patients about common habits or practices that may interfere with their sleep and implement strategies for avoiding them.⁶⁶ Clinicians must be aware that, as often occurs in this population, older adults may not offer information about sleep practices unless specifically asked about them.

Behaviors and habits that may impair sleep include:⁶⁶

- (1) Frequent daytime napping
- (2) Spending too much time in bed
- (3) Insufficient daytime activities
- (4) Late-evening exercises
- (5) Insufficient bright-light exposure
- (6) Excess caffeine
- (7) Evening alcohol consumption
- (8) Smoking in the evening
- (9) Late, heavy dinner
- (10) Watching television or engaging in other stimulating activities at night
- (11) Anxiety and anticipation of poor sleep
- (12) Clock watching
- (13) Environmental factors, such as the room being too warm, too noisy, or too bright; pets on the bed or in the bedroom; and active or noisy bed partners.

Sleep Restriction–Sleep Compression

Sleep restriction therapy entails limiting time in bed to consolidate actual time sleeping. The patient is counseled to reduce the amount of time in bed to correlate closely with actual time sleeping. The recommended sleep times are based upon sleep logs kept for 2 weeks before sleep restriction therapy is begun. Thus, an individual who reports spending 8.5 hours in bed, but sleeping only 5.5 of those hours, would be counseled to limit his or her time in bed to 5.5 to 6 hours. Time allowed in bed is gradually increased in 15- to 20-minute increments (approximately once every 5 days if improvement is sustained) as sleep efficiency increases, until the individual's optimal sleep time is obtained.⁶⁴

In sleep compression, a variant of sleep restriction, patients are counseled to decrease their time in bed gradually to match total sleep time rather than making an immediate substantial change, as is the case in sleep restriction therapy.⁶⁴ A number of studies support the efficacy of sleep restriction–sleep compression therapy as a treatment for older patients with chronic insomnia.^{67–69} These approaches can also be combined with other modalities.

Stimulus Control

People suffering from chronic insomnia may adopt coping strategies that exacerbate the problem. Watching television or reading in bed, worrying about falling asleep, or using the bedroom for vigorous discussions or arguments are examples of behaviors that can impair sleep by producing associations between the bed and bedroom and those activities; the bedroom should be associated only with sleeping and sex. Stimulus control therapy attempts to eliminate these behaviors in the bedroom and thereby strengthen the association between sleep and the bed and bedroom.

The following are helpful instructions for using stimulus control and practicing good sleep hygiene.^{7,35,66}

- (1) Develop a sleep ritual, such as maintaining a 30-minute relaxation period before bedtime or taking a hot bath 90 minutes before bedtime.
- (2) Make sure the bedroom is restful and comfortable.
- (3) Go to bed only if you feel sleepy.
- (4) Avoid heavy exercise within 2 hours of bedtime.
- (5) Avoid sleep-fragmenting substances, such as caffeine, nicotine, and alcohol.

- (6) Avoid activities in the bedroom that keep you awake. Use the bedroom only for sleep and sex; do not watch television from bed or work in bed.
- (7) Sleep only in your bedroom.
- (8) If you cannot fall asleep, leave the bedroom and return only when sleepy.
- (9) Maintain stable bedtimes and rising times. Arise at the same time each morning, regardless of the amount of sleep obtained that night.
- (10) Avoid daytime napping. If you do nap during the day, limit it to 30 minutes and do not nap, if possible, after 2 p.m.

Relaxation Therapy

The goal of relaxation therapy is to guide individuals to a calm, steady state when they wish to go to sleep. The methods used include progressive muscle relaxation (tensing and then relaxing each muscle group), guided imagery, diaphragmatic breathing, meditation, and biofeedback.⁷⁰

Cognitive Behavioral Therapy for Insomnia

This treatment combines multiple behavioral approaches, usually incorporating sleep restriction, stimulus control, and cognitive therapy, with or without relaxation therapy. Sleep hygiene and sleep education are frequently included. Protocols for older adults may vary somewhat from those used for younger patients, but all approaches aim to correct

the common misperceptions regarding normal aging and sleep by providing information about how much sleep is necessary to maintain health and the physical and psychological consequences of sleep loss. Motivational strategies to increase compliance are also emphasized.³⁶ A number of studies have demonstrated the efficacy of multicomponent CBT in older adults.⁷¹⁻⁷³

Exercise and Complementary and Alternative Treatment Modalities

Some studies report that walking, Tai Chi, acupuncture, and weight training improve sleep for some individuals,⁷⁴⁻⁷⁷ but how these approaches affect sleep is not well understood and is likely to be complex.⁶⁵ Also, difficulties inherent in these studies preclude their recommendation as evidence based. Nevertheless, there are many good reasons to encourage regular physical activity in older individuals, given its positive effect on functional and cognitive status.

Pharmacological Treatment

The Food and Drug Administration (FDA) has approved 10 drugs by for the treatment of insomnia, including benzodiazepines, nonbenzodiazepines, and a melatonin receptor agonist (Table 2). The selection of a drug should depend on matching the characteristics of the particular drug with the patient's complaint. All should be started at the lowest available dose.

Table 2. Food and Drug Administration-Approved Hypnotics for Insomnia

Generic Name	Trade Name	Indication	Geriatric Dose (mg)	Half-Life in Older Persons (Hours)	Comments
Benzodiazepine*					
Flurazepam	Dalmane	Short-term treatment of insomnia	15	126-158	Should not be used in older adults because of very long half-life
Quazepam	Doral		7.5	78	Should not be used in older adults because of very long half-life
Estazolam	ProSom		0.5-1	10-24	Because of long half-life, residual CNS effects are likely
Temazepam	Restoril		7.5-15	3.5-18.4	
Triazolam	Halcion		0.0625-0.25	1.7-5	Poor choice because of very short half-life and high incidence of CNS adverse reactions
Nonbenzodiazepine†					
Eszopiclone	Lunesta	No short-term limitation for use; sleep onset and sleep maintenance insomnia	1-2	9	AEs > 10%: headache, unpleasant taste
Zolpidem ER	Ambien CR		6.25	1.9-7.3	AEs > 10%: dizziness, headache, somnolence
Zolpidem	Ambien		5	2.9-3.7	AEs > 10%: dizziness, headache, somnolence
Zaleplon	Sonata		5	1	AEs: nausea (7%), myalgias (7%)
Melatonin receptor agonist					
Ramelteon	Rozerem	No short-term limitation for use; sleep onset insomnia	8	1-2.6	AEs: headache (7%) Somnolence (5%) Dizziness (5%) Not a Class C-IV scheduled drug

New interpretive guidelines (F329) from the Centers for Medicare and Medicaid Services mandate quarterly review of sedative-hypnotic compounds for residents of long-term care facilities to assess continued need, dose, and possible side effects, including possible decline in functional status or increased incidence of falls.

* All are Class C-IV scheduled drugs, and may also be associated with amnesia and complex sleep-related behaviors such as sleepwalking or sleep eating.

† The nonbenzodiazepines have a fast onset of action (30-45 minutes).

CNS = central nervous system; AE = adverse effect.

The benzodiazepines are psychoactive drugs with varying hypnotic, sedative, anxiolytic, anticonvulsant, muscle relaxant, and amnesic properties. Nonbenzodiazepines, also called benzodiazepine receptor agonists, are comparatively new drugs whose actions are similar to those of the benzodiazepines, although they are structurally unrelated. The one approved melatonin receptor agonist, also comparatively new, has a different mechanism of action. Melatonin receptors, acted upon by endogenous melatonin, are thought to be involved in the maintenance of the circadian rhythm underlying the normal sleep–wake cycle.

The National Institutes of Health (NIH) State-of-Science Conference on Insomnia concluded that the benzodiazepine receptor agonists are efficacious in the short-term management of insomnia and that the frequency and severity of any adverse effects are lower than those found in the older benzodiazepines.³⁶ The NIH document was published before the availability of the melatonin agonist. Nevertheless, although the nonbenzodiazepines may have less of a tendency for dependence and abuse, adverse effects can still become a problem with the newer drugs. No significant effects indicative of potential for abuse or motor and cognitive impairment have been demonstrated for the melatonin receptor agonist.⁷⁸

A meta-analysis that compared hypnotic use with placebo found that sleep quality improved, total sleep time increased, and number of nocturnal awakenings decreased,⁷⁹ but adverse events were also more common with sedative-hypnotics than with placebo, although most adverse events were reported to be reversible and not severe. Older people may be at greater risk for adverse effects because of pharmacokinetic considerations, such as reduced clearance of certain sedative-hypnotics. There is also some evidence of pharmacodynamic differences such as greater sensitivity to peak drug effects. Impairment was shown to be dependent on dose and time since dosing.

Other classes of medications have also been used to treat insomnia in the elderly. The 2005 NIH State-of-the-Science Conference on Insomnia concluded that there is no systematic evidence for the effectiveness of many medica-

tions, including the antihistamines, antidepressants, antipsychotics, and anticonvulsants used off label for the treatment of insomnia, and warned that the risks of use outweighed the benefits.³⁶ Trazodone, a frequently prescribed antidepressant for insomnia in older persons, is sedating, can cause orthostasis, and has no published evidence of sustained efficacy.¹¹

Combination Therapy

Combining behavioral and pharmacological therapy may provide for better outcomes than use of either modality alone. Past studies in adults have shown that combination therapy has been efficacious, with medications providing short-term onset relief and behavioral therapy providing longer-term sustained benefit. Only one randomized controlled clinical trial has evaluated combination therapy in older adults.^{61,62} In this study, combination therapy was not only more efficacious than placebo, it was more efficacious than pharmacological or behavioral therapy alone. The study concluded that, although combination therapy was effective for the short-term management of insomnia in late life, sleep improvements were better sustained over time with behavioral treatment. Although results from the few controlled studies that have been performed on combination therapy are encouraging, there is still enough of a paucity of data to caution against overgeneralization.⁸⁰

Summary

Insomnia in older adults is most often comorbid with medical and psychiatric illness and complicated by the polypharmacy conventionally associated with them. Treatment should include behavioral therapy whenever possible. Successful management of sleep in older adults may result in significant improvement in quality of life and daytime functioning.

For recommendations, see Table 3.

Table 3. Insomnia

Recommendation	Quality of Evidence (Reference)	Strength of Evidence
The healthcare practitioner should periodically screen patients for symptoms of insomnia during health examinations.	III ⁸¹	A
An in-depth sleep history is essential in identifying the cause(s) and consequence(s) of insomnia. In addition, a physical examination is an important element in the evaluation of insomnia patients with medical symptoms.	III ⁸¹	A
CBT-I is an effective treatment for insomnia in older adults.	I ³⁶	A
Nonbenzodiazepines and melatonin receptor agonists are the safest and most efficacious hypnotic drugs currently available.	II-I ^{36,62,83}	B
All Food and Drug Administration–approved drugs for the treatment of insomnia can be associated with clinically significant adverse events.	III ⁷⁹	A
Combining CBT-I and pharmacological therapy can be helpful in some patients.	III ^{61,80}	A
Antihistamines and antidepressants, anticonvulsants, and antipsychotics are associated with more risks than benefits in the treatment of insomnia, particularly in older persons.	II-III ^{36,84,85}	B

See Table 1 for quality and strength of evidence codes.

CBT-I = cognitive behavioral therapy for insomnia.

Future Research

- (1) Does improving insomnia in older adults result in improvement in daytime functioning (including lower risk of falls, less daytime sleepiness, improvement in memory and concentration, improved quality of life)?
- (2) Does improving insomnia in older adults result in improvement in medical comorbidities (including fewer doctor office visits)?
- (3) Does improving insomnia in older adults result in improvement in psychiatric comorbidities (particularly depression and anxiety)?
- (4) Will increasing slow wave sleep in older adults result in improvements in overall quality of sleep as well as improvement in daytime functioning?

SLEEP APNEA

Definition

Sleep apnea is a condition in which people stop breathing while asleep.⁸⁶ Apneas (complete cessation of respiration) and hypopneas (partial decrease in respiration) result in hypoxemia and changes in autonomic nervous system activity, resulting in increases in systemic and pulmonary arterial pressure and changes in cerebral blood flow.^{87,88} An arousal (brief awakening) generally terminates the episode, which results in fragmented sleep. These arousals are believed to be an important contributor to the symptoms of excessive daytime sleepiness (EDS) and the neurocognitive impairment seen in sleep apnea.

Two types of sleep apnea are recognized. In OSA, the primary pathophysiological event is obstruction of the upper airway, manifested by greatly diminished or absent airflow in the presence of an effort to breathe. Central sleep apnea (CSA) is characterized by recurrent episodes of apnea during sleep resulting from temporary loss of ventilatory effort due to central nervous system or cardiac dysfunction.^{89,90} This latter type of apnea is commonly found in patients with congestive heart failure (CHF), particularly in those with Cheyne-Stokes respiration. This guideline will primarily focus on the much more common OSA, defined as sleep apnea associated with EDS.

Prevalence

OSA has been described in all age groups. In the adult population, OSA (defined as 10 or more apneas and hypopneas per hour of sleep) occurs in approximately 15% of men and 5% of women.⁹¹ In older adults, OSA occurs in up to 70% of men and 56% of women.²⁷ The syndrome is much more common in postmenopausal than premenopausal women, but the prevalence increases in both sexes with aging.⁹²

Assessment

Signs and Symptoms

EDS and a history of snoring are by far the most common presenting symptoms in most patients with OSA. Other symptoms of OSA include observed apnea, choking or gasping on awakening, morning headache, and nocturia.⁹³ Although most younger patients with OSA are obese, elderly people with OSA may not necessarily be obese.⁹⁴

Risk Factors

Risk factors for OSA include age, obesity, and anatomic abnormalities affecting the upper airway. In the older population, OSA is also more common in Asians than in Caucasians.⁹⁵ OSA has been associated with heart failure, atrial fibrillation, and stroke, conditions that are more common in the older population.⁹⁶ In women, OSA is often associated with a history of hypothyroidism.⁹⁷

Morbidity and Mortality

Studies show that older adults with OSA are excessively sleepy and that it is likely that OSA contributes to poorer quality of life, greater neurocognitive impairment, and greater risk of nocturia and cardiovascular disease.⁹⁸ Cardiovascular comorbidities particularly associated with OSA include arterial hypertension, heart failure, and stroke.⁸⁷ Often, the hypertension is difficult to control.⁹⁹

Diabetes mellitus is also more common in this population, and there may be an association between apnea and insulin resistance. Depression has also been found as a common comorbidity in women with OSA.⁹⁷ Although mortality is greater in untreated apnea patients younger than 50, the effect of OSA on mortality in the older population is unclear.¹⁰⁰

Management of OSA

OSA is managed using a four-step approach: confirming the diagnosis; determining optimal treatment; general management measures; and ongoing, chronic follow-up.

Confirming the Diagnosis

Taking the History. Because OSA is so common in older people, all older patients should be questioned to determine whether OSA symptoms are present.^{91,101,102} The history should be obtained from the patient and a bed partner or caregiver, if possible, and should include questions covering the cardinal symptoms of OSA, specifically EDS, snoring, and observed apnea. Questions about nocturia, cognitive impairment, and any comorbidities should be included as well. Physicians should consider OSA syndrome in individuals who are overweight or have a history of heart disease, hypothyroidism, or stroke.

The Epworth Sleepiness Scale (ESS), although not validated for use in older persons, is useful for documenting daytime drowsiness.¹⁰³ Nocturia is a surprisingly common finding in OSA patients. This symptom in men is commonly misinterpreted as being caused by prostatic hypertrophy.¹⁰⁴ OSA should be suspected in all patients with hypertension, especially with hypertension that is resistant to treatment.⁹⁹

Physical Examination. The physical should focus on the upper airway, including the nasal and pharyngeal airways, to exclude anatomic obstruction. The skeletal structure of the face must be assessed to exclude the possibility of jaw abnormalities (retrognathia or micrognathia) that may cause OSA in the absence of obesity.¹⁰⁵ Dental structures should be examined if a mandibular advancement device is being considered. Obesity often involves the trunk and neck, and documentation of neck collar size (>17" in men and >16" in women) may be helpful, especially in men.

Differential Diagnosis. OSA needs to be distinguished from sleep deprivation, hypothyroidism, depression, and

the effects associated with using medications with sedating effects. These can all elicit the main symptom: EDS. Prescribed medications and over-the-counter products may also contribute to breathing difficulties during sleep or may produce daytime sleepiness. Inquiring about alcohol use, and obtaining a detailed list of all medications and other products, particularly sedative-hypnotics and opiate analgesics, are important.¹⁰⁶⁻¹¹⁰

Polysomnography. Patients suspected of having OSA based on historical features and physical examination will almost always require objective documentation using polysomnography (PSG) to confirm the presence and severity of the apnea.¹¹¹⁻¹¹⁴ The Centers for Medicare and Medicaid Services (CMS) and most insurance carriers require PSG for reimbursement of continuous positive airway pressure (CPAP) therapy. Comprehensive PSG includes the measurement of variables to document sleep breathing disorders (oxygen saturation in arterial blood, rib cage and abdominal movement, nasal and oral airflow, and snoring sounds), data regarding sleep and stage of sleep (using electroencephalography, electrooculography, and electromyography), and electrocardiogram and leg electromyogram to document the presence of periodic leg movements. The PSG is usually followed by CPAP titration. Although PSG is usually performed in a laboratory setting, CMS may cover home testing in selected patients.¹¹¹

Quantification of OSA. The apnea-hypopnea index (AHI) is the most widely used metric for characterizing the severity of the abnormalities of sleep respiration and is based on the average number of apneas plus hypopneas per hour of sleep in a single night's study. More than five is considered diagnostic for OSA. CMS covers reimbursement for treatment when the AHI is greater than 15 or greater than five with comorbidities (e.g., sleepiness or cardiovascular disease).^{112,113}

Determining Optimal Treatment

Determining Treatment. When OSA in older adults is associated with clinical symptoms, particularly hypertension, cognitive dysfunction, nocturia, high levels of sleep-disordered breathing, or cardiac disease, it should be treated, regardless of the age of the patient. Most patients with OSA, including those with hypoventilation syndromes (e.g., individuals suspected of having obesity hypoventilation, impaired ventilation secondary to neuromuscular diseases, or CSA), those with significant respiratory disease (e.g., chronic obstructive pulmonary disease, severe asthma, or restrictive diseases), and those with significant cardiac disease such as CHF, will probably require referral to or management by sleep specialists. Such patients may require complex treatment.

Treatment. There is no pharmacological treatment for OSA. CPAP is the best approach and first-line of treatment for most patients. CPAP works by stenting open the airway, increasing functional residual capacity of the lungs, possibly increasing pharyngeal dilator activity, and reducing afterload on the heart. Several studies have confirmed that older adults tolerate nightly CPAP use.^{111,112}

The choice of interface-type of headgear (nasal or oronasal mask) for securing the mask to the head and the need for a chinstrap are determined objectively. Response to CPAP is usually assessed as part of comprehensive PSG

during the latter part of the diagnostic study night (split-night study) or during an additional all-night study. The CPAP titration is performed in a split-night study after the patient has been asleep for at least 2 hours and the OSA diagnosis has been confirmed. This involves fitting the patient with an appropriate mask, educating him or her about what is to transpire, and then applying increasing levels of pressure until OSA control is attained. Proper fit and education will help adherence and reduce claustrophobia.

A split-night study may not be appropriate if there is insufficient time during the night to make a diagnosis and also determine optimal pressures. In addition, some patients may require a more-complex device than a standard fixed-pressure CPAP machine. Only after a review of all diagnostic and therapeutic sleep studies can the optimal treatment approach be determined.

Patients without teeth can sometimes present a challenge for CPAP treatment because of bone resorption in the upper and lower jaws. This situation presents difficulties for optimal mask fitting and makes oral appliances unfeasible.

General Management Measures

Although the following general measures have not been evaluated in rigorous randomized clinical trials, they are based on evidence from case series and general physiological findings.

Avoidance of Alcohol, Sedative-Hypnotics, and Opiates. Alcohol and other agents (e.g., opiates, many anesthetic agents, and sedative-hypnotics) can depress upper airway tone and may worsen OSA syndrome.^{107,108,110} Older patients about to undergo surgery should be screened for OSA, at least according to history, because they might receive opiates during the perioperative period.

Weight Loss. A great deal of evidence supports the strong positive correlation between weight and OSA risk. Weight reduction plays an important role in the management of obese OSA patients.¹¹³⁻¹¹⁵ One study of older OSA patients monitored for 18 years found a reduction in the severity of the apnea associated with weight loss.¹¹⁵

Treatment of CHF. Patients with CHF are at risk of developing Cheyne-Stokes respiration, a form of CSA. Cheyne-Stokes respiration can result in severe sleep onset and sleep maintenance insomnia, as well as daytime sleepiness.^{116,117} Older patients with CHF and sleep apnea (particularly CSA) have a 2.7 times greater risk of reduced survival than patients with CHF or apnea alone.¹¹⁸

CHF treatment may improve breathing abnormalities in CSA, but results from a recent randomized clinical trial indicate that CPAP may increase mortality in the first 2 years of treatment. Therefore, CPAP is not currently recommended as a first-line treatment in CHF.¹¹⁹ Small short-term clinical trials have suggested the effectiveness of oxygen and adaptive servoventilation, a ventilatory support mode specifically designed for CHF breathing abnormalities.¹²⁰ No long-term outcome studies are available.

General Surgery in OSA Patients. Older people are more likely than younger people to have general surgery and to require general anesthesia. All older patients, especially those with the risk factors for OSA, must be questioned about the possibility of OSA. If they are at high risk, an objective assessment should be done. If OSA is confirmed during the preoperative assessment, nasal CPAP should be

initiated before hospital admission, and the equipment should be brought to the hospital at admission. The post-surgical period harbors significant risk for such patients, because anesthetic agents and opiates can worsen OSA in unprotected individuals.¹¹⁰

Oral Appliances. These devices, which move the lower jaw up and forward, can be effective, especially in mild to moderate cases.¹¹³ Guidelines for their use are identical to those in younger people, although in older individuals, special attention must be paid to the examination of the jaws and teeth because at least eight healthy teeth in each of the upper and lower jaws are required to anchor the appliances. Patients without adequate dentition cannot be treated with such appliances.

Ongoing, Chronic Follow-Up

OSA is a chronic illness and as such requires long-term management. The main symptoms relate to neurocognitive function and daytime sleepiness. The ESS, although not specifically validated in the older population, is the most commonly used assessment instrument for daytime sleepiness. With CPAP treatment, an improvement in the ESS score of 2 or more points is expected, as well as an overall improvement in subjective sleepiness assessment. When CPAP is no longer effective or sleepiness returns, the patient should be reevaluated.

Cognitively impaired patients may have difficulty mastering the steps involved in putting on their masks and cleaning their CPAP machines and headgear, although one study of patients with mild to moderate Alzheimer’s disease living at home showed that these patients were adherent to CPAP treatment.¹²¹ Help from a family member or caregiver is generally necessary. Some of the newer CPAP sys-

tems can monitor adherence, but the clinical utility of monitoring has not been rigorously determined.

For recommendations, see Table 4.

Future Research

- (1) What kind of alternatives to CPAP treatment in older people (who may have difficulty because of lack of dexterity in using CPAP) can be developed?
- (2) Will the treatment of OSA in older adults result in improvements in nocturia and cognition?
- (3) What are the optimal diagnostic techniques for older people who are in skilled nursing facilities or long term care?

RLS AND PLMS OF SLEEP

Definition

RLS is a sleep disorder characterized by unpleasant leg sensations that disrupt sleep.^{90,126,127} The syndrome is classified as primary or secondary. Primary, or idiopathic, RLS is likely to develop at an earlier age, has no known associated or predisposing factors, and probably has a genetic basis. First- and second-degree relatives of patients with idiopathic RLS have a significantly greater risk of developing RLS than relatives of matched controls.¹²⁸ Secondary RLS can result from a variety of medical conditions that have iron deficiency in common. These include iron-deficiency anemia, end-stage renal disease, and pregnancy.¹²⁹

Prevalence

The prevalence of RLS symptoms is approximately 10% in most population-based surveys.¹³⁰⁻¹³² Although the rate of RLS may be lower in Asian than European populations, the

Table 4. Sleep Apnea

Recommendations	Quality of Evidence (Reference)	Strength of Evidence
OSA is common in older people. All older people should be screened for the possibility of OSA by asking whether the three key features are present: daytime sleepiness, snoring, and observed apnea.	II ^{27,91,98,101,102,122,123}	A
All older patients who are found to be at high risk of OSA should be assessed to document whether the condition is present by referral to a sleep specialist or obtaining a polysomnography.	III ¹¹⁶	A
Older patients whose sleep apnea is associated with congestive heart failure or respiratory disease should be referred to a sleep specialist.	III	A
CPAP is the most reliable treatment at this time for OSA (but not for central sleep apnea). Oral appliances may be appropriate for some patients with adequate dentition.	I ^{111,112,118-120,124}	A
All older patients prescribed CPAP should receive education on the rationale, methods of application, expectations, and follow-up. Bed partners should also be educated. This information improves adherence to treatment.	II ^{112,113,125}	B
Because obstructive apnea is a chronic disease with associated chronic comorbidities, older patients should be followed up frequently, especially during the first 6 months after start of treatment to assess response to treatment, and to monitor adherence.	III ¹¹³	A
Patients with OSA should take their CPAP equipment with them on trips and to the hospital, particularly if they are expecting to undergo a surgical procedure.	III ¹¹⁰	A
A weight loss program should be part of the treatment plan in overweight patients with OSA.	II ^{114,115}	A
The older sedative-hypnotics should not be prescribed for patients with confirmed OSA, and patients should be advised to avoid drinking alcohol within 2 hours of bedtime.	III ^{107,108,110}	A
Patients with OSA require follow-up for their comorbidities, particularly patients with hypertension. Patients treated with CPAP may have a change in their requirements for antihypertensive medications.	III ⁹⁹	A

OSA = obstructive sleep apnea; CPAP = continuous positive airway pressure.

prevalence is similar in African Americans and Caucasians.^{133,134} Because the diagnosis of RLS is based on symptom report, prevalence rates for frequency and severity vary with different criteria. For example, in the Restless Legs Syndrome Prevalence and Impact Study, 7.2% of the survey population reported RLS symptoms, but only 5.0% noted symptoms occurring at least twice per week, and symptoms were moderately or severely distressing in only 2.7%.¹³²

The prevalence of RLS symptoms increases with age for men and women, at least until the seventh or eighth decade. Higher rates of symptoms are consistently reported in women than in men.^{130–132} Some of the age-related risk is due to the fact that, although RLS can develop at any age, it rarely remits. Increasing prevalence of RLS with age may also occur in association with the increasing presence of secondary causes in the aging population, such as iron deficiency and renal failure.

Typical Symptoms and Signs

RLS sensations are usually described as a compelling urge to move the lower extremities, but they may also be reported as a creepy-crawly, burning, itching, or even painful feeling. The resultant sleep disruption may lead to insomnia and daytime sleepiness. Although symptoms most commonly involve the lower extremities, they have also been described in the upper extremities and even the trunk. RLS has a circadian pattern, with the intensity of the symptoms becoming worse at night and improving toward morning. Symptoms also tend to worsen when the individual is at rest. They improve with movement such as walking, rubbing, or stretching. The diagnosis is made according to history without the need for PSG in the majority of cases.^{90,127}

Risk Factors

A familial risk exists for the development of RLS and PLMS (described in more detail below). In an Icelandic cohort of patients with RLS and PLMS, a significant association was found with a common variant on chromosome 6p21.2. The Icelandic investigators reported an association between the variant and PLMS without RLS and no association for RLS without PLMS, suggesting that the variant was a genetic determinant of PLMS.¹³⁵

A variety of medications, including tricyclic antidepressants, SSRIs, lithium, and dopamine antagonists (antipsychotics), have been reported to exacerbate RLS.¹³¹ In addition, several social or lifestyle factors appear to contribute to RLS symptoms. These include higher body mass index (BMI) and caffeine intake, sedentary lifestyle, tobacco use, and lower income.^{131,136}

Pathophysiology

The exact pathophysiology of RLS and PLMS remains unclear, but the spinal cord, peripheral nerves, and central dopamine and narcotic receptors may be involved.^{136,137} The impairment of dopamine transport in the substantia nigra due to low intracellular iron appears to play a critical role in most patients with this disorder.¹³⁷

Assessment

Key questions to include in the history:

- (1) Is there an urge to move the legs, and do uncomfortable or unpleasant sensations in the legs accompany or cause this urge?
- (2) Do the unpleasant sensations or the urge to move begin or worsen during periods of rest or inactivity, such as sitting or lying down?
- (3) Does movement, such as walking or stretching, partially or totally relieve the unpleasant sensations or the urge to move for at least as long as the activity continues?
- (4) Do the unpleasant sensations or urge to move get worse or only occur in the evening or night?

The physical examination is usually unremarkable in primary RLS, although secondary causes such as peripheral neuropathy or radiculopathy may be elicited during an examination. Therefore a thorough neurological examination is important.

There are no specific laboratory tests necessary to establish the diagnosis, although because iron deficiency states are often associated with RLS from secondary causes, obtaining a serum ferritin is recommended. Values less than 50 ng/mL are consistent with a diagnosis of RLS and suggest the need for iron supplementation.

The differential diagnosis for RLS includes peripheral neuropathies, vascular disease (intermittent claudication), neuroleptic-induced akathesias, arthritides, and venous varicosities. A careful history is usually sufficient to distinguish RLS from each of these.¹³⁸

Assessing RLS in the Cognitively Impaired. Cognitively impaired individuals may require a broader approach when considering the diagnosis. The following are considered essential criteria to make the diagnosis of RLS in these patients.¹²⁷

- (1) Signs of leg discomfort, such as rubbing or kneading the legs or groaning while holding the lower extremities
- (2) Excessive motor activity in the lower extremities, such as pacing, fidgeting, repetitive kicking, tossing and turning in bed, slapping the legs on the mattress, cycling movements of the lower limbs, repetitive foot tapping, rubbing the feet together, and the inability to remain seated
- (3) Signs of leg discomfort exclusively present or worse during periods of inactivity
- (4) Signs of leg discomfort diminished with activity
- (5) Criteria 1 and 2 occurring only in the evening or at night or worse at those times than during the day.

Treatment

Pharmacological Approaches

The primary pharmacological therapies are dopaminergic agents. Opioids, benzodiazepines, and anticonvulsants are considered to be second-line agents. If pharmacological therapy is required, evidence supports the use of dopaminergic agents as first-line treatment, especially the newer dopamine receptor agonists such as ropinirole or pramipexole (both FDA approved for RLS).¹³⁹ These agents are associated with less rebound and symptom augmentation than dopamine precursors such as levodopa-

carbidopa. Side effects include nausea, orthostatic hypotension, sleepiness, headache, and compulsive behaviors. In older patients, particular consideration should be given to drug interactions with other medications and the risk of orthostasis.

The beginning dose of ropinirole is 0.25 mg orally 1 to 3 hours before bedtime. The dose can be increased after 2 to 3 days to 0.5 mg and to 1 mg after 7 days. Titration upwards is by weekly 0.5-mg increments to a maximum of 4 mg at week 7 if needed.⁵⁹ Pramipexole 0.125 mg orally should be administered 2 to 3 hours before bedtime. If needed, the dose can be doubled every 4 to 7 days to a maximum of 0.5 mg.¹⁴⁰

Worsening and earlier-onset of symptoms in a patient whose leg discomfort was initially controlled with medication characterizes augmentation of RLS. Typical presentations are symptom onset earlier in the day, worsened intensity of symptoms, and spread of symptoms to other parts of the body, such as from the calves to the thighs. The frequency of augmentation with the newer FDA-approved dopamine agonists is unknown, but it is common in patients who are treated with levodopa-carbidopa.¹⁴¹ There is no standard approach to the treatment of augmentation with the newer dopaminergic agents, but options include taking a dose earlier in the day, splitting the existing doses into early-evening and bedtime doses, and switching to a different class of medication, such as an anticonvulsant.

Nonpharmacological Approaches

Nonpharmacological approaches to the management of RLS include education, moderate exercise, smoking cessation, alcohol avoidance, caffeine reduction or elimination, and discontinuation of offending medications if appropriate.

PLMS AND PERIODIC LIMB MOVEMENT DISORDER

Definition

This movement disorder of sleep, also sometimes called nocturnal myoclonus or periodic leg movements, consists of repeated rhythmical extensions of the big toe and dorsiflexion of the ankle with occasional flexion of the knee and hip. The movements may cause brief awakenings or arousals from sleep, of which the individual may or may not be aware.

Prevalence

As many as 90% of individuals with RLS have PLMS.¹⁴² PLMS associated with arousals is linked to disturbed sleep in older women.¹⁴³ PLMS is more common with aging.²⁵

Typical Symptoms and Signs

PLMS usually occurs predominantly during the first part of the night. Each movement lasts approximately 2 to 4 seconds, with a frequency of approximately every 20 to 40 seconds.⁹⁰

Risk Factors

PLMS is usually associated with other sleep disorders, including sleep-disordered breathing, but the most notable association occurs with RLS, suggesting a similar patho-

physiology. The rate of PLMS correlates with subjective RLS severity.^{140,144} PLMS is also common in patients taking antidepressants.¹⁴⁵ Although the presence of PLMS supports the diagnosis of RLS, limb movements are neither necessary nor sufficient to make the diagnosis of RLS.

Assessment

The revised diagnostic criteria for periodic limb movement disorder (PLMD; below) note that leg jerks occur with many medical conditions and in the presence of many medications. These criteria also “raise the bar” for the “abnormal” number of periodic limb movements in adults, from five to 15 as determined by the PLMS Index (the number of periodic limb movements per hour of total sleep time as determined according to PSG).

Diagnostic criteria for PLMD:

- (1) PSG demonstrates repetitive, highly stereotyped, limb movements.
- (2) The PLMS index exceeds 15 per hour in most adult cases.
- (3) There is clinical sleep disturbance or a complaint of daytime fatigue.
- (4) Another current sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder not better explain the PLMS.

Note: If PLMS is present without clinical sleep disturbance, the PLMS can be reported as a polysomnographic finding, but criteria are not met for a diagnosis of PLMD.

Treatment

There is little evidence to support pharmacological treatment to suppress PLMS or PLMD, even in the face of insomnia or hypersomnia, particularly in older adults. No agent has been FDA-approved to treat PLMS or PLMD.

For recommendations, see Table 5.

Future Research

- (1) What medications are associated with a greater risk of RLS or PLMS in older people?

Table 5. Restless Legs Syndrome (RLS) and Periodic Limb Movement of Sleep

Recommendation	Quality of Evidence (Reference)	Strength of Evidence
Patients with sleep-onset insomnia should be asked about uncomfortable leg sensations.	III ^{90,127,132}	A
Patients with RLS should have their serum ferritin level checked.	III ¹³⁷	B
Dopaminergic agents are the first-line treatment for RLS.	II ¹³⁹	B
Patients who are treated with dopaminergic agents should be warned about the possibility of augmentation.	III ^{90,132}	A
Periodic limb movements rarely need to be treated with medication in the absence of RLS symptoms.	III ^{144,146}	B

See Table 1 for quality and strength of evidence codes.

- (2) Does treatment of RLS affect outcomes such as blood pressure, depression, sleep measures, and health-related quality of life in older individuals?
- (3) What is the natural history of RLS in older individuals?

CIRCADIAN RHYTHM SLEEP DISORDERS IN AGING

Definition

The hallmark of circadian rhythm sleep disorders (CRSD) is the presence of relatively normal sleep that occurs at abnormal times. In the case of advanced sleep phase disorder (ASPD), sleep commences and ends at unusually early times; in the case of irregular sleep-wake disorder (ISWD), sleep is dispersed across the 24-hour day in bouts of irregular length. The combination of age-related changes in sleep and circadian rhythm regulation paired with low levels of light exposure and activity contribute to the development of CRSDs in older people.

Pathophysiology

Optimal sleep quality is achieved when the desired sleep time coincides with the timing of the endogenous circadian rhythm of sleep and wake propensity. CRSD arises from alterations of the central circadian clock or a misalignment between endogenous circadian timing and the external 24-hour social and physical environment. Although the primary pathophysiology of CRSD is a disruption of circadian timing, a combination of physiological, behavioral, and environmental factors often influence the clinical presentation of CRSD. The CRSDs that are most prevalent in older people are ASPD and ISWD.

Significant changes in sleep and circadian regulation occur with aging. Common sleep complaints of older adults include habitually earlier bedtimes and wake times, inability to maintain sleep through the night, undesired early-morning awakening, and frequent daytime sleepiness.^{26,147-149} A change in the circadian timing system of older people or in the interaction between the circadian and homeostatic processes may cause these sleep disturbances in part. Habitual wake time, the rise of hormone secretion, and endogenous temperature nadir of older subjects occurs at an earlier clock hour, suggesting that the earlier wake time may be due to an advance of the circadian clock.¹⁵⁰⁻¹⁵³ There is also evidence that the interaction between a reduction in the homeostatic drive for sleep and a reduction in the strength of the circadian signal promoting sleep may be responsible for the impaired sleep of older individuals in the early morning.¹⁵⁴

Assessment

For a diagnosis of CRSD, an accurate clinical history, sleep diary, actigraphy (a small motion sensor worn continuously, usually on the wrist), or a combination covering at least 7 days should be obtained. Other physiological markers of the circadian phase such as dim-light melatonin onset and nadir core body temperature are adjunctive tools to confirm the phase or amplitude of circadian rhythms but are not widely available clinically. PSG is not routinely indicated, although because of the age-related increase in the prevalence of other sleep disorders, a careful assessment for con-

ditions such as sleep apnea, restless legs, and REM sleep behavior disorder (RBD) should be performed in all patients with CRSD.¹⁵⁵ Furthermore, psychiatric conditions, including depression and anxiety disorders, are frequent comorbidities with CRSD and must be considered in the evaluation and differential diagnosis.

Advanced Sleep Phase Disorder

Clinical Presentation

The defining characteristic of ASPD (also known as advanced sleep phase syndrome) is sleep-wake times that are earlier than desired or earlier than conventional. Sleep onset times may be as early as 6:00 p.m. to 9:00 p.m., even if the patient attempts to delay sleep onset. These are coupled with wake times between 2:00 a.m. and 5:00 a.m.

Excessive sleepiness during waking hours and sleep maintenance insomnia may occur in conjunction with abnormal sleep timing. Sleep is otherwise normal when individuals are permitted to sleep on their own particular sleep-wake schedule.

Diagnostic criteria require verification of the advanced sleep-wake phase through the use of at least 1 week of actigraphy or sleep log. Other sleep disorders, medical or psychological conditions (such as depression), medication factors, or substance use disorders that may be causing the symptoms need to be excluded.¹⁵⁵ As expected, an earlier onset of high melatonin levels and core body temperature minimum are seen, and these can confirm the diagnosis but are not required in the routine assessment.¹⁵⁶ Not all individuals with an advanced sleep phase have ASPD, and many older people are not particularly bothered by their sleep phase and have no consequent functional impairment. Such individuals can be considered "morning types" or "larks" rather than ASPD patients.

Prevalence

In middle-aged to older adults, the prevalence of ASPD is estimated at 1% to 7%.^{157,158} ASPD is much less common in the general adult population, with only a few reported cases of non-age-related ASPD.¹⁵⁹⁻¹⁶¹

Pathophysiology

The pathogenesis of ASPD is thought to involve a combination of behavioral and genetic factors. For example, early sleep times and ophthalmological conditions such as cataracts may decrease light exposure at a time that would delay the sleep phase (i.e., evening hours), thereby perpetuating the advanced sleep phase. Intrinsic factors, such as a shortened endogenous circadian period (<24 hours) or alterations in the relationship between circadian timing and sleep homeostatic regulation may play a role in the development of ASPD.^{159,162} Furthermore, familial forms of ASPD have been reported in which the phenotype segregates in an autosomal-dominant inheritance pattern,^{159,161,163} and mutations in the circadian clock *hPer2* and *CK1 delta* genes have been identified.^{164,165} Thus, less exposure or weak responses to entrainment agents such as light and physical activity, together with intrinsic changes in circadian and sleep regulation and genetic predisposition, may all contribute to the development of ASPD in older individuals.^{166,167}

Treatment

A combination of good sleep hygiene practices and methods to delay the timing of sleep and wake times is often recommended for the treatment of ASPD. Chronotherapy has been used successfully in ASPD. In this approach, sleep times are advanced every 2 days until the desired sleep–wake time has been achieved, although the need for rigorous adherence, the length of the treatment, and the necessity for close follow-up limit its overall clinical practicality. Therefore, use of evening light within the phase delay portion of the light phase response curve (PRC) is one approach used to treat. In addition to light and good sleep hygiene, other behavioral adjustments are also central to the effective treatment of the disorder.

Light. Successful phase delay with the use of evening light therapy has been reported in several studies. Light therapy in these patients may additionally improve sleep efficiency and total sleep time.^{168,169} Bright-light therapy used in the delay portion of the light PRC (in the evening between 7:00 p.m. and 9:00 p.m.) can help normalize or delay circadian rhythms in patients with ASPD. Bright-light therapy generally consists of broad-spectrum light of 2,500 to 10,000 lux for 1 to 2 hours duration. Unfortunately, light at lower intensities may not delay sleep phase effectively. In addition, older subjects appear to have poorer response to the generally superior phase-shifting properties of short wavelength (blue) light than their younger counterparts, raising the question of the usefulness of this spectrum of light in the treatment of older subjects with ASPD.^{170,171}

Older adults may have difficulty tolerating bright light, and adherence to and efficacy of light therapy may decline over time. Close follow-up is advised. The clinician should use the timing, intensity, and duration of light exposure (7:00 p.m. to 9:00 p.m.) as a general guideline to initiate therapy. If the initial therapy fails, a referral to a specialist to adjust the timing or duration of light therapy is recommended.

Side Effects. Light boxes filter ultraviolet rays, so they are considered to be safe, although side effects have been reported, including hypomania, mild headache, nausea and vomiting, and self-limited visual problems.¹⁷² A specialist should evaluate patients with ophthalmological disease before beginning light therapy, to determine whether this approach is appropriate. Additional caution is advised in subjects with preexisting mania, retinal photosensitivity, and migraine.

Dosing and Duration of Treatment. Although the exact length of treatment and dosing levels have yet to be clearly established, light therapy represents a potentially important instrument in the manipulation of circadian phase. The American Academy of Sleep Medicine has confirmed the potential usefulness of light therapy for CRSDs such as ASPD.¹⁷³

Melatonin. Theoretically, melatonin delivered in the morning should result in a delay in sleep phase based on the melatonin PRC,¹⁵² but data supporting the efficacy of melatonin in ASPD are lacking. Additionally, melatonin may produce soporific effects, which may result in residual morning sleepiness.

Irregular Sleep–Wake Disorder

Clinical Presentation

The lack of a clearly identifiable circadian pattern of consolidated sleep and wake times characterizes irregular sleep–wake disorder (ISWD), also known as irregular sleep–wake rhythm. Although the total amount of sleep obtained over a 24-hour period is within the normal range, the time asleep is broken into at least three different periods of variable length. Erratic napping typically takes place during the day, whereas nighttime sleep is severely fragmented and shortened. Symptoms of chronic insomnia or daytime sleepiness may appear as a consequence. To confirm the diagnosis, the exclusion of other disorders that may better explain the patient's irregular sleep, as well as at least 1 week of actigraphy or the use of a sleep log demonstrating three or more sleep bouts within the 24-hour day, is required.¹⁵⁵

Prevalence

ISWD is most commonly encountered in patients with dementia, particularly in those who are institutionalized, although other disorders of the central nervous system (e.g., traumatic brain injury and mental retardation) can lead to an irregular sleep–wake pattern.^{153,174,175}

Pathophysiology

It is likely that the development and maintenance of an irregular sleep–wake rhythm result from dysfunctional central processes responsible for circadian rhythm generation and less exposure to external synchronizing agents such as light and social activities.¹⁷⁶ The pathogenesis of the disease may be related to a loss of neurons or other deleterious changes within the suprachiasmatic nucleus (SCN).¹⁷⁷ A few studies have demonstrated a decrease in the number of neurons within the SCN in patients with Alzheimer's disease.^{178,179} Also, residents of long-term care facilities often lack exposure to adequate light and do not participate in regular daytime activities. This may contribute further to a decrease in the amplitude of circadian rhythms. Lower daytime light levels are associated with more nighttime awakenings, even after controlling for degree of dementia.¹⁸⁰

Treatment

The primary goal of treatment of ISWD is to consolidate the sleep–wake cycle. To this end, measures aimed at restoring or enhancing exposure to the various zeitgebers ("time-givers," environmental cues that provide an estimate of time of day) are critical. Patients should be exposed to bright light during the day while avoiding it in the evening.^{181,182} Daytime physical and social activities should be strongly encouraged.^{177,183–185} A multicomponent approach using a variety of behavioral treatment options is recommended.

Light. The overall approach to light therapy for the treatment of the irregular sleep–wake type is to increase the duration and intensity of light exposure throughout the daytime and avoid exposure to bright light in the evening. Bright-light exposure delivered for 2 hours in the morning at 3,000 to 5,000 lux (a unit of light or illumination) over the course of 4 weeks has been found to decrease daytime napping and increase nighttime sleep in subjects with dementia.¹⁸⁶ Light may help consolidate nighttime sleep,

Table 6. Circadian Rhythm Sleep Disorders (CRSD)

Recommendation	Quality of Evidence (Reference)	Strength of Evidence
All older people with symptoms of insomnia and excessive daytime sleepiness should be screened for the possibility of a CRSD.	III ⁹⁰	A
Diagnosis is made primarily according to history. In addition, a sleep diary or actigraphy should be performed for at least 7 consecutive days to confirm the circadian sleep-wake pattern.	III ⁹⁰	A
Circadian phase markers (e.g., core body temperature, melatonin) are useful to confirm the diagnosis, but there is insufficient evidence to recommend their routine use in diagnosis and they are not available clinically.	III ⁹⁰	C
Polysomnography is indicated if other primary sleep disorders are suspected but is not indicated for diagnosis.	III ⁹⁰	B

See Table 1 for quality and strength of evidence codes.

decrease agitated behavior, and increase the amplitude of circadian rhythms.^{181,182,186}

Melatonin. Studies evaluating the use of melatonin in ISWD have yielded inconsistent results. One trial involving patients with Alzheimer's disease found no statistically significant differences in actigraphy-derived sleep measures between control subjects and individuals taking 2.5 mg of melatonin, although a trend toward improvement was seen with a 10-mg dose.¹⁸⁷ A review of current evidence has found inconclusive evidence for the efficacy of melatonin treatment in circadian and sleep disorders,^{188,189} although melatonin may be effective in patients with known melatonin deficiency.¹⁹⁰

Other Therapeutic Approaches. Structured physical and social activity may help provide the temporal cues needed to improve the regularity of the sleep-wake schedule. A reduction in nighttime light and noise and improvement in incontinence care can encourage a favorable sleep environment that will minimize awakenings in nursing home residents.¹⁹¹ Furthermore, elderly subjects with disrupted sleep-wake patterns consistent with ISWD slept less during the day and increased participation in social and physical activities and social conversation when they followed a routine of less time in bed during the day, structured bedtime routine at night, 30 minutes or more of sunlight exposure a day, and more physical activity.¹⁹² A multidimensional, nonpharmacological approach that includes more sunlight exposure and social activity during the day, less time in bed during the day, and less nighttime noise may be particularly effective.

Recommendations

For recommendations for evaluation of CRSD, see Table 6.

For recommendations for management of ASPD, see Table 7.

For recommendations for the management of ISWD, see Table 8.

Future Research

- (1) Research is needed to define the timing, duration, and optimal light wavelength of bright-light therapy for older adults with ASPD.
- (2) Multicenter placebo-controlled randomized studies are necessary to determine the efficacy, safety, and tolerability of long-term therapy with bright light in older adults.
- (3) Placebo-controlled randomized clinical trials of the efficacy and safety of melatonin receptor agonists are required in the treatment of ISWD in patients with dementia.
- (4) Basic research to understand the pathophysiology of circadian rhythm sleep disorders, including the role of genetics, is also a priority.

PARASOMNIA: REM BEHAVIOR DISORDERS (RBD)

Definitions

Parasomnias are undesirable nondeliberate physical or emotional events that occur during sleep. They most often appear during entry into sleep or during arousals and may arise from specific sleep states, such as NREM or REM states. Parasomnias may include abnormal movements, behaviors, emotions, perceptions, dream enactment, and autonomic activity that occur during sleep or are associated

Table 7. Advanced Sleep Phase Disorder (ASPD)

Recommendations	Quality of Evidence (Reference)	Strength of Evidence
Chronotherapy (sleep-wake scheduling) is achieved by advancing sleep and wake times until the desired sleep and wake times are achieved. This approach, although possibly useful, is often clinically impractical.	III ¹⁶⁰	C
Scheduled bright light in the evening delays circadian rhythms and improves sleep in patients with ASPD.	II ^{168,193}	B
Melatonin should not be used in older persons with ASPD.	III ¹⁹³	B
Overall, there is little scientific evidence to support the efficacy of behavioral interventions, but because of the lack of alternative approaches and because the risks and relative costs are low, behavioral interventions are recommended.	III	B

See Table 1 for quality and strength of evidence codes.

Table 8. Irregular Sleep–Wake Disorder (ISWD)

Recommendation	Quality of Evidence (Reference)	Strength of Evidence
For institutionalized patients with ISWD, in whom dementia is common, increasing exposure to bright light during the day can improve sleep–wake consolidation and circadian rest–activity rhythm.	II ^{193,194}	A
Melatonin is not indicated for the treatment of ISWD in older adults with dementia.	I ¹⁸⁷	B
Multimodal approaches that combine bright-light exposure during the day, decreasing light exposure at night, physical activity, social activity, structured bedtimes and wake times, and noise reduction can decrease nighttime awakenings, total wake time, and daytime sleepiness.	I–II ^{182,195,196}	A

See Table 1 for quality and strength of evidence codes.

with arousal from sleep. Manifestations of parasomnias include enuresis, sleepwalking, night terrors, dream anxiety attacks, nocturnal complex seizures, and REM behavior disorder. The non-REM sleep parasomnias are more common in children, whereas RBD is more common in older adults.

REM Sleep Behavior Disorder

Symptoms and Signs

RBD is one of the most dramatic and potentially injurious of the parasomnias. Patients report complex, often violent motor behaviors associated with dream enactment; approximately 10% of patients do not have dream recall.¹⁹⁷ The potential for self- and bed-partner injury is high, especially during severe episodes. The majority of cases occur with advancing age, typically manifesting in the sixth or seventh decade.¹⁹⁷

Pathophysiology

RBD is based on an underlying pathophysiology in which there is a lack of the normal atonia associated with REM sleep because of a dysfunction of motor neuron inhibition. The dream enactment is associated with loss of muscle atonia during REM sleep. PSG demonstrates intermittent loss of REM sleep-associated muscle atonia, with the patient manifesting complex, often violent motor activity associated with dream mentation.¹⁹⁸

Assessment

Diagnosis of RBD is made according to the history and PSG evidence of greater electromyographic activity during REM sleep (lack of atonia). The sleep study may also capture the actual episodes of limb jerking and other complex, vigorous, and violent behaviors. If there is evidence of abnormal neurological activity, a full neurological workup, including brain magnetic resonance imaging, may be needed.^{198–200}

RBD has been seen in association with various brainstem abnormalities, extrapyramidal neurological disorders, and medical conditions (e.g., Parkinson's disease, progressive supranuclear palsy, Shy-Drager syndrome, multiple-system atrophy, brainstem stroke, brainstem tumor, demyelinating disease, and medication toxicity or withdrawal). It may also be idiopathic. The differential diagnosis of RBD includes non-REM parasomnia, sleep apnea, periodic movements of sleep, nocturnal seizures, and nocturnal rhythmic movements. Medications such as tricyclic antidepressants, monoamine oxidase inhibitors, and SSRIs have been shown to induce or exacerbate RBD, and RBD has also been

described during alcohol and barbiturate withdrawal and with caffeine use.^{199–204}

Treatment

Management of RBD involves pharmacological treatment and interventions that address environmental safety. The most effective drug therapy is clonazepam at a dose of 0.5 to 1 mg at bedtime. Patients who report sleep-onset insomnia or morning drowsiness as a result of the medication may take clonazepam earlier (1–2 hours before bedtime). Clonazepam is effective in 90% of cases. There is little evidence of abuse and only infrequent reports of tolerance in older patients. Beneficial effects are observed within the first week of clonazepam treatment, resulting in control of vigorous, violent sleep behaviors, although mild to moderate limb movement, sleep-talking, and other complex behaviors may persist. Discontinuation of treatment usually results in recurrence of symptoms.¹⁹⁸

Other medications that may be efficacious are levodopa, dopamine agonists, and melatonin. Although there are studies reporting the efficacy of melatonin, it is a nutritional supplement that is not approved by the FDA and, in terms of pharmacological preparation, is poorly regulated.^{205–207} It should probably not be used in older patients.

Environmental safety is an important concern with management of RBD. Patients should be advised to remove potentially dangerous objects from the house, to pad hard and sharp surfaces around the bed, to cover windows with heavy draperies, and even to place the mattress on the floor to avoid falling out of bed, if necessary. The combination of drug therapy and implementation of safety precautions offers safe and effective management of RBD.

For recommendations, see Table 9.

Future Research

- (1) Multicenter trials are needed of the safety and efficacy of pharmacological therapies in older adults with parasomnias, such as RBD. Trials should be conducted for clonazepam, melatonin, and dopamine agonists.
- (2) Research is necessary to elucidate the relationship between RBD and Parkinson's disease.

HYPERSOMNIAS

Definition

The hypersomnias of central origin are a group of disorders characterized by a primary complaint of excessive sleepi-

Table 9. Parasomnias

Recommendation	Quality of Evidence (Reference)	Strength of Evidence
All older adults with a history of vigorous motor behaviors during sleep should be asked about prior episodes or the potential for injurious behavior associated with dream mentation.	III ^{90,198–200}	A
Polysomnography is indicated for the diagnosis of RBD.	III ^{90,198–200}	A
In a patient with RBD and an abnormal physical examination, further neurological evaluation is recommended.	III ^{201–204}	B
Clonazepam has demonstrated efficacy and is indicated for the treatment of RBD.	III ^{198,208}	B
Other medications, such as dopamine agonists may be indicated for the treatment of RBD.	II ²⁰⁷	B

See Table 1 for quality and strength of evidence codes.
RBD = rapid eye movement sleep behavior disorder.

ness not caused by disturbed nocturnal sleep or misaligned rhythms. Excessive sleepiness is the inability to maintain wakefulness and alertness during the major waking episode of the day. This may result in unintentional episodes of falling asleep at inappropriate times or places or in planned naps related to irresistible sleepiness. Patients occasionally use the word “sleepy” to describe times when they are “fatigued” or “tired.” However, the term “excessive sleepiness” is better reserved for cases characterized by a greater sleep propensity and falling asleep at inappropriate times.

Disorders that cause disturbed nocturnal sleep with subsequent sleepiness during the waking period may coexist with a hypersomnia. The nocturnal sleep disorder must be controlled before assigning a diagnosis of hypersomnia. One example would be the patient with hypersomnia and OSA; the sleep apnea must be adequately treated before considering an independent diagnosis of a hypersomnia.

A number of disorders are included under the category of hypersomnias. The more common ones seen in older adults include narcolepsy with cataplexy, narcolepsy without cataplexy, narcolepsy due to a medical condition (secondary narcolepsy), idiopathic hypersomnia with long sleep time (sleep time > 10 hours), idiopathic hypersomnia without long sleep time (total sleep time of 6–10 hours), hypersomnia due to a medical condition, and hypersomnia due to a drug or substance.

Prevalence

The prevalence and demographic characteristics of a number of the disorders in this category are unknown. Narcolepsy with cataplexy has an overall prevalence of 0.05%, with a slight preponderance in men.^{90,209,210} Although all ages can be affected, the prevalence of narcolepsy with cataplexy and the other hypersomnias specifically in older adults is unknown, although the medical conditions, comorbidities, and medication usage typically associated with hypersomnia are prevalent in older adults.

Symptoms and Signs

All of the disorders in this category have several symptoms and signs in common. Each presents with excessive sleepiness during waking hours. Other symptoms are more specific to individual disorders.

Idiopathic Hypersomnia

Idiopathic hypersomnia with long and short sleep times are defined according to a 3-month history of EDS (excessive

daytime sleepiness) with a sleep time longer than 10 hours and between 6 and 10 hours, respectively.^{90,211–214} The daytime sleepiness may be shorter than 3 months in the case of hypersomnia due to a drug or substance.⁹⁰ Some patients may experience symptoms associated with excessive sleepiness such as memory lapses, concentration problems, automatic behavior (an episode that occurs during a period of sleepiness and that is not remembered subsequently by the individual), ptosis, and hallucinations.^{215–222}

Narcolepsy (with or without Cataplexy)

To establish a diagnosis of narcolepsy with cataplexy, EDS and a definite history of cataplexy is required.⁹⁰ Cataplexy is an episode of muscle weakness usually manifesting as weakness in the legs or arms, buckling at the knees, or dropping items from the hands in association with emotion (e.g., laughter or anger). This symptom is absent in narcolepsy without cataplexy.⁹⁰ Other features of narcolepsy include automatic behaviors, hypnagogic hallucinations, sleep paralysis, and disturbed nocturnal sleep. A hypnagogic hallucination is a hallucination, most often visual, that occurs at sleep onset, and sleep paralysis is an episode of immobility that occurs at sleep onset or upon awakening.

Hypersomnia Associated with Comorbidities

Patients with hypersomnia due to a medical condition have a complaint of excessive sleep present almost daily for at least 3 months that is secondary to a significant medical or neurological condition.^{90,223–225} Medical conditions include Parkinson's disease, posttraumatic brain injury, Niemann-Pick disease type C, myotonic dystrophy, Prader-Willi syndrome, Alzheimer's disease, stroke, multiple sclerosis, hypothyroidism, and hepatic encephalopathy.^{223,226–234}

Patients with hypersomnia due to a drug or substance have a complaint of sleepiness or excessive sleep that is believed to be secondary to current use, recent discontinuation, or prior prolonged use of drugs or prescribed medications.^{90,235} Because older individuals often regularly take multiple medications, careful evaluation of an individual's drug regimen is an essential part of the assessment of hypersomnia in an older adult.

Risk Factors

Many hypersomnias have genetic and nongenetic risk factors, but usually they are not proven or definitively identified. For example, suggested precipitating factors for narcolepsy with or without cataplexy have included head

trauma, sustained sleep deprivation, and nonspecific viral illness.²³⁶

Morbidity and Mortality

The morbidity and mortality associated with the hypersomnias of central origin are primarily related to EDS. Cognitive impairment is a common feature, characterized by fatigue, tiredness, impaired memory and concentration, and coordination difficulties.^{237–240} Depression and problems at work (e.g., loss of employment due to sleep-related errors) or with social life (e.g., withdrawal from family and social activities because of sleepiness) are also common. Weight gain has also been linked to excessive sleep.²⁴¹ There is also a greater risk of traffic accidents or work-related injury due to sleepiness and inattentiveness.²⁴² Untreated narcolepsy with cataplexy can also be socially disabling, because the cataplexy attacks can lead to social withdrawal in addition to the risk of accidents.^{242,243}

Assessment

Specific studies are lacking regarding the assessment or treatment of older patients with hypersomnias of central origin. Therefore, the data collection tools used to assess patients are not specifically validated for the older adult population, although some extrapolations from existing studies can be made.

Key issues and questions to be addressed in obtaining a history:

- (1) If possible, obtain the history from the bed partner as well as the patient.
- (2) Questions should address EDS, cataplexy, symptom response to napping (if any), presence of dreaming during naps, hypnagogic hallucinations, sleep paralysis, and automatic behaviors.
- (3) Establish onset, frequency, and duration of the sleepiness as well as any episodes of remission.
- (4) Include questions about the patient's medical, neurological, and psychiatric illnesses, as well as use or recent discontinuation of recreational drugs, prescription drugs, or alcohol.
- (5) Questions about other comorbid sleep disorders such as OSA or RLS are also relevant.^{208,224,235,244–246} A number of subjective sleep questionnaires are available to assess sleep habits, sleep-wake schedules, and sleepiness (e.g., the ESS (Epworth Sleepiness Scale) and Karolinska Sleep Scales; sleep diaries are also useful assessment tools). The most commonly used questionnaire is the ESS. Such questionnaires should be part of the patient's evaluation.^{232,247}
- (6) What is the duration of nighttime sleep?

Important Areas to Include in the Physical Examination

For patients suspected of having a hypersomnia of central origin, a thorough physical examination, including a neurological evaluation, is important. An assessment of cognition is valuable and can be used to help make a diagnosis, as well as to monitor treatment response.

Appropriate Laboratory Tests

For diagnosis, patients suspected of having hypersomnias of central origin usually require an overnight PSG followed by a

multiple sleep latency test (MSLT).^{248–250} The MSLT is an electrophysiological test of sleep tendency that involves four or five daytime naps at 2-hour intervals with assessment of the latency to sleep onset and the type of sleep that occurs. A mean sleep latency of 8 minutes or less and the presence of REM sleep during two or more naps are indicative of narcolepsy. The MSLT is also required to support a diagnosis of one of the other hypersomnias of central origin. Common medications used to treat chronic conditions in older adults may complicate the interpretation of these studies.

A magnetic resonance image of the brain is useful in identifying causes of hypersomnia or narcolepsy due to a neurological disease (e.g., tumors, multiple sclerosis, intracranial bleeds, or strokes). Additionally, blood work can help identify suspected medical conditions that may cause the patient's excessive sleepiness (e.g., thyroid-stimulating hormone, liver function tests, complete blood count, serum chemistry). Cerebrospinal fluid hypocretin levels can confirm a diagnosis of narcolepsy with cataplexy in the absence of a MSLT.

Treatment Options

Initial management of hypersomnias of central origin requires treatment optimization of any underlying medical, neurological, or psychiatric disorder. Furthermore, careful withdrawal of sedating medications or substances, if possible, is prudent. Ensuring an adequate opportunity for nighttime sleep is important to exclude sleep deprivation as a cause of excessive sleepiness.

Excessive sleepiness is treated with behavioral modification, modafinil, other stimulants, or a combination.^{251–256} Cataplexy is controlled with behavioral modification, antidepressants, or sodium oxybate.²⁵⁷

Behavioral

Some degree of behavioral modification is beneficial to most patients with excessive sleepiness. Good sleep hygiene techniques should be adopted, and a regular sleep-wake schedule allowing adequate time for nocturnal sleep should be maintained. Heavy meals throughout the day and alcohol use should be avoided. Two short 15- to 20-minute naps, one scheduled at approximately noon and the other at approximately 4:00 to 5:00 p.m., may alleviate some sleepiness.

The older patient who is still employed may benefit from occupation counseling. These individuals should avoid shiftwork, on-call schedules, jobs that involve driving, or any other job that demands continuous attention for long hours without breaks, especially under monotonous conditions. Healthcare workers should assist the patient with occupational and social accommodation for disabilities due to excessive sleepiness. Referral for support services and to support groups such as the Narcolepsy Institute or the National Sleep Foundation is helpful to many patients.^{210,229,248,258}

Pharmacological

Stimulant medications (amphetamines, methamphetamines, D-amphetamines, and methylphenidate) are traditionally used to treat EDS.²⁵⁹ Modafinil has recently gained favor for first-line use in the treatment of narcolepsy.²⁵⁹ Modafinil has also been increasingly used for the treatment of idiopathic hypersomnia, as well as hypersomnias due to a

medical or neurological condition. For elderly patients, a starting dose of modafinil at 100 mg upon awakening in the morning is recommended. This dose can be increased at weekly intervals as necessary. Typical doses range from 200 to 400 mg per day. The most common adverse reactions are nausea, headaches, and nervousness.

Other medications used to treat EDS in patients with narcolepsy include sodium oxybate, selegiline, and ritan-serin^{253,257,260–263}. Judicious use of caffeine may also be beneficial. In patients with drug- or medication-induced sedation, the treatment is to reduce or remove the drug or substance. This therapy should preferably be instituted under the guidance of a sleep specialist, who is familiar with these drugs, and the patient's primary care physician, who knows the patient's medical problems and the medications that he or she is taking.

Treatment of Cataplexy and REM Sleep Intrusion into Wakefulness

Sodium oxybate improves daytime sleepiness and cataplexy.^{260,261} In addition, it may be used to treat the other symptoms of narcolepsy, including disrupted nocturnal sleep, hypnagogic hallucinations, and sleep paralysis. Sodium oxybate is a liquid that is given in two divided doses at night. The first dose is given at bedtime and the second 2.5 to 4 hours later. Sodium oxybate can cause headaches, nausea, unexpected neuropsychiatric effects, and fluid retention. Selegiline, a monoamine oxidase inhibitor rarely used in narcolepsy because of the potential for side effects, not only improves daytime sleepiness, but can also treat cataplexy. Other REM sleep suppressant medications, such as tricyclic antidepressants, SSRIs, venlafaxine, and reboxetine, have all been used to treat cataplexy, hypnagogic hallucinations, and sleep paralysis, although adequate scientific evidence is lacking.

Follow-Up

Most of the hypersomnias of central origin are long-term or lifelong disorders and require ongoing management.

Monitoring Medications

As in most clinical scenarios, more-frequent follow-up is usually necessary when starting a medication or adjusting doses. For example, starting or adjusting the dose of a stimulant requires monitoring for adverse effects, including hypertension, palpitations or arrhythmias, irritability, or behavioral manifestations such as psychosis. Patients should be questioned about excessive stimulatory effects or nocturnal sleep disturbances.

Monitoring Symptoms

Because medications such as modafinil generally only improve sleepiness and do not eliminate it, frequent reassessment of impairments in functional ability due to residual sleepiness is necessary. The ESS is a useful tool for monitoring subjective sleepiness and its response to therapy at each patient visit. Once symptoms are stable, any future exacerbation of symptoms (sleepiness, cataplexy, sleep paralysis, hypnagogic hallucinations, or behavioral abnormalities) needs to be evaluated formally using a history, physical examination, repeat PSG, or a combination.

Healthcare workers should continue to assist the patient with occupational and social accommodation for disabilities due to excessive sleepiness.

Referral

Primary care physicians should refer a patient to a sleep specialist when narcolepsy or idiopathic hypersomnia is suspected or the cause of the sleepiness is unknown. In addition, complex patients who are unresponsive to initial or subsequent therapy may benefit from a sleep specialist consultation.

For recommendations, see Table 10.

Future Research

- (1) The basic pathophysiological mechanism(s) of excessive sleepiness due to idiopathic hypersomnia, narcolepsy, and Alzheimer's disease needs to be further investigated.
- (2) There is a need for standardization and validation of MSLT studies in older adults.
- (3) The efficacy of treatment modalities, especially pharmacotherapy (e.g., modafinil, lithium, sodium oxybate) in older adults for hypersomnia of central origin requires further study.
- (4) There is a need for better understanding of the comorbidities that may be associated with hypersomnias of central origin in older adults (e.g., cognitive deficits and obesity).
- (5) There is a need for better understanding of how older age and comorbidities may modify the symptoms and response to treatment in older adults.

SLEEP PROBLEMS IN LONG-TERM CARE FACILITIES

Background

Sleep disturbances are common in older people living in nursing homes, with many factors specific to this setting and population contributing to sleep difficulties. Medical conditions common to nursing home residents complicate these environmental factors. Residents frequently experience pain, paresthesias, nighttime cough and dyspnea, gastroesophageal reflux, and nocturia, all of which can interfere with sleep. Neurological illnesses are also common in this population, particularly neurodegenerative disorders such as dementia and Parkinson's disease, which are also associated with sleep disturbance. Medications prescribed for nursing home residents may also interfere with sleep, including diuretics, stimulating agents (e.g., sympathomimetics, bronchodilators, and stimulating antidepressants), anti-Parkinsonian agents, antihypertensives, and cholinesterase inhibitors taken near bedtime or sedating medications (e.g., antihistamines, anticholinergics, and sedating antidepressants) taken during the day. The latter group may contribute to daytime drowsiness and further disrupt the sleep-wake cycle.

Environmental factors may also play a role in sleep-wake problems in nursing homes. Many residents have limited interaction with the community outside of the nursing home due to physical or cognitive impairment. Most

Table 10. Hypersomnias

Recommendation	Quality of Evidence (Reference)	Strength of Evidence
A complaint of excessive sleepiness should be thoroughly evaluated using a detailed history and the appropriate use of subjective questionnaires.	III ^{16,221–224,230,231,244,245,247,264–267}	A
An accurate diagnosis should be established in older patients with hypersomnia using polysomnography and multiple sleep latency test.	II ^{210,248–250}	B
In patients with hypersomnia, management of any medical, neurological, and psychiatric disorders should be optimized.	III ^{16,208}	A
In patients with hypersomnia, medications or substances with sedating properties should be withdrawn when possible, or the timing of the medications should be changed to minimize sedation during waking hours.	III ^{208,235,246}	A
Behavioral modification of sleep–wake behavior is an effective treatment strategy that is useful for many patients with excessive sleepiness and should be advised.	II ^{208,229,246,259}	A
Scheduled naps can be beneficial to relieve sleepiness with or without the use of pharmacological agents.	II ^{259,268}	B
Pharmacological management should be considered for all patients who have a diagnosis of hypersomnia of central origin.	III ^{208,210,269}	A
There is little evidence that sleepiness due to medications is improved with other medications that are used to counteract sleepiness. If possible, discontinue the offending medication or change the timing or dose.	III	A
The following medications are effective treatments for narcolepsy with or without cataplexy.		
Modafinil is effective for excessive sleepiness due to narcolepsy.	I ^{210,251–253,269}	
Sodium oxybate:		B
May be effective for excessive daytime sleepiness due to narcolepsy in the older adult population.	I ^{257,261,269}	C
May be effective for cataplexy in the older adult.	I ^{257,260,261,269}	B
Methylphenidate and amphetamine derivatives may be effective for excessive sleepiness due to narcolepsy in the older adult.	II ²⁰⁸	C
Antidepressant medications may be an effective treatment for cataplexy.	II ^{208,210,262,263,269}	B
Modafinil, methylphenidate, and amphetamine derivatives may be effective for the treatment of excessive sleepiness due to idiopathic hypersomnia or recurrent hypersomnia, as well as hypersomnia due to a medical condition.	II ^{208,229,253–256}	C
Regular follow-up of patients with excessive sleepiness is necessary to monitor and ensure effective treatment.	III ^{210,230}	A
Referral to a sleep specialist should be undertaken when narcolepsy or idiopathic hypersomnia is suspected or if the cause of the sleepiness is unknown.	III	A

See Table 1 for quality and strength of evidence codes.

residents have little, if any, bright-light exposure, which impedes the coordination of the internal circadian clock to the external environment. Nursing home residents also spend extended periods in bed and are often physically inactive during the day, factors that contribute to sleep–wake and circadian rhythm abnormalities. Nighttime noise and light interruptions are also disruptive and are often caused by staff providing personal care to the resident or a roommate.

Assessment

A variety of measures have been used to assess sleep in nursing home residents. Most studies use wrist actigraphy for objective sleep measurement. PSG and portable sleep recording is used less frequently. Subjective sleep measures generally involve observations by research staff, resident questionnaires, nursing staff interviews or questionnaires, or medical record review.

Prevalence

Probably because of the different methodologies used for measurement, prevalence data regarding sleep problems in

nursing homes vary. The lowest estimate, 6.3% of residents, was estimated using Minimum Data Set (MDS) documentation from 34,000 nursing home residents in the state of Michigan,⁴³ but given concerns about the accuracy of the MDS data for insomnia, this is likely to be a significant underestimation.²⁷⁰

Residents have been reported to have significant sleep problems, including long sleep onset latency (time to fall asleep at night), long wake time after sleep onset (amount of time awake after initially falling asleep), low sleep efficiency (total sleep time as a percentage of total time spent in bed), and a high percentage of daytime napping.²⁷¹

Structured daytime observations have shown in one study that nearly three-quarters of residents sleep excessively during the day. Residents with excessive daytime sleeping were observed more often in bed, were less likely to have any time outdoors, had less social and physical activity, and required greater mean level of assistance for personal care activities than those without this pattern. Significantly less time asleep at night accompanied excessive daytime sleeping. Most residents also had evidence of

nighttime sleep disturbance, although this was rarely documented in their charts.²⁷²

Factors Associated with Sleep Disturbances in Nursing Home Residents

Studies using actigraphy in nursing home residents with dementia suggest an association between dementia and circadian rhythm disturbance, with residents with more severe dementia having more-disturbed circadian rest-activity rhythms.^{180,273} Older people on a rehabilitation ward or in a nursing home have also described pain, discomfort, and the need to go to the toilet as the most common causes of sleep disturbance.²⁷⁴

Cognitively intact nursing home residents have reported nocturia, noise and light disruption, and pain as the most common causes of subjective sleep disturbance. In addition, greater comorbidity and more depressive symptoms were significant independent predictors of worse Pittsburgh Sleep Quality Index scores.²⁷⁵

Mild to moderate sleep apnea was reported in one study in 32% of residents, with another 38% having evidence of severe sleep apnea. There was a strong relationship between sleep apnea and dementia in this sample.²⁷⁶ A later study in nursing home residents with dementia found that more than half had evidence of severe sleep apnea.²⁷⁷

Evidence of a relationship between sleep and psychotropic medications in nursing home residents has been mixed. For example, one study found that sleep fragmentation (estimated using actigraphy) was not associated with sedative-hypnotic use, whereas another found that residents taking psychoactive medications had a dampening of the normal day-night variation in sleep and waking over 24 hours.^{278,279} Another study showed that use of psychotropic medications (e.g., antipsychotics, sedative-hypnotics, and antidepressant medications) was not associated with measures of daytime or nighttime sleep, although residents taking psychotropic medications had less in-bed body movement at night, which may increase risk of skin breakdown.²⁸⁰

Institutional factors contribute to sleep disturbance in the nursing home setting. One study that used a bedside monitor in residents' rooms to measure noise and light levels found that half of nighttime awakenings were associated with noise or light.²⁸¹

Many residents also have limited exposure to bright light, which is a key zeitgeber ("time-giver") to time intrinsic biological rhythms to the external clock. Studies have shown that residents do not get much, if any, exposure to bright light, with nearly half of residents having no bright-light exposure at all.^{170,273} Residents with higher light levels had fewer nighttime awakenings and a later rest-activity acrophase, a measure of circadian rhythm.¹⁷⁰

Sleep disturbances in nursing home residents are associated with significant negative consequences. Sleep disturbance has been identified as a significant predictor of mortality in nursing home residents.²⁸² Excessive daytime sleeping has also been associated with worse quality of life (less participation in social and physical activities, less social conversation) and more functional impairment (more nursing assistance for eating, drinking, bathing, dressing, grooming, and toileting).²⁷¹

Although little evidence of a relationship between psychoactive medications and sleep has been found in nursing home residents, there is clinical concern about a potential relationship between sedating medications and the risk of falls. One study found that use of benzodiazepines was associated with greater risk of daytime and nighttime falls.²⁸³ Risk of daytime and nighttime falls was the same regardless of use of benzodiazepines with intermediate or long half-lives, although use of benzodiazepines with short half-lives was predictive of falls only during the night. Another large study suggested that it was insomnia, rather than the use of hypnotics, that was associated with greater risk of falls, although the findings from that study remain controversial.^{43,271}

Interventional Studies

Studies of sleep in nursing home residents have included interventions involving bright-light therapy, exercise or physical activity, multicomponent nonpharmaceutical programs, changes to nighttime nursing care, social and individualized activities, client-centered nursing care, medications, and discontinuation of antipsychotic medication.

Beneficial effects of morning bright-light therapy have been reported.^{182,284-286} Nursing home residents with dementia receiving bright overhead lighting in the morning or all day long have been shown to have more total sleep time at night, with the effect most pronounced in participants with severe dementia.²⁸⁷ The most relevant dimensions of light therapy may be the timing, duration, and intensity of the intervention.

The beneficial effects of exercise and physical activity on sleep in nursing home residents has been demonstrated in a number of studies.^{280,288,289} Positive sleep effects have also been reported with use of a stationary bicycle and Tai Chi.^{290,291} The combination of daily social and physical activity in residents of a continuing care facility has also been shown to be associated with more slow-wave sleep (as assessed according to PSG) and improvement in memory-oriented tasks.¹⁶⁷

Efforts to improve the nighttime nursing home environment to make it more conducive to sleep have been difficult to implement. A nursing intervention that decreased nighttime noise and light disruption was shown to be associated with fewer nighttime arousals.¹⁹¹

Multicomponent nonpharmaceutical interventions have also had mixed results. A study that combined efforts to increase daytime physical activity and sunlight exposure, decrease time in bed during the day, provide a bedtime routine, and decrease nighttime noise and light levels found a decrease in duration of nighttime awakenings and daytime sleeping and greater participation in social activities, conversation, and physical activity during the day.¹⁹² A similar multicomponent nonpharmaceutical intervention found no significant effects on nighttime sleep but showed a modest but significant decrease in daytime sleeping.²⁹²

Few studies have tested sleep medications specifically in the nursing home setting. A randomized trial of temazepam, diphenhydramine, or placebo in residents with sleep problems found that those who received diphenhydramine reported a shorter sleep latency than those given placebo, with some report of a longer duration of sleep than with

Table 11. Long-Term Care

Recommendation	Quality of Evidence (Reference)	Strength of Evidence
Given the high prevalence of sleep problems in nursing home residents, clinicians should consider sleep disturbance as a potential problem in every nursing home resident.	II ^{271,272}	A
Excessive daytime sleeping is common in nursing home residents and should be addressed.	II ²⁷²	B
Wrist actigraphy for the identification of sleeping problems in nursing home residents should be used if possible. Trained staff observations, resident self-report, and nursing staff report should be used if actigraphy is not available.	II ^{180,271–273,275,295}	B
Clinicians should have a high index of suspicion for sleep disordered breathing in nursing home residents, particularly those with dementia.	II ²⁷⁵	B
Careful review of medical conditions and medications that may be causing or contributing to sleep disturbance is warranted in every nursing home resident with evidence of a sleep problem.	III	A
Clinicians should encourage nursing home facilities to address nighttime environmental conditions and nursing care practices contributing to a nighttime environment that is not conducive to sleep.	II ²⁸¹	B
Clinicians should encourage nursing home facilities to implement measures to decrease amount of time residents spend in bed during the daytime and increase daytime physical activity.	III ²⁸	B
There is good evidence that daytime bright-light exposure improves sleep–wake patterns in nursing home residents. Clinicians should encourage facilities to implement measures that increase bright-light exposure using commercially available light boxes or sunlight exposure.	I ^{182,285–288}	A
There is essentially no evidence regarding the effectiveness of sedative–hypnotic medications in the nursing home population. Careful review of potential risks and benefits of these medications in nursing home residents is warranted, particularly concerns about risks of daytime sedation and falls.	III ²⁸⁴	B
Use of medications not Food and Drug Administration–approved for the treatment of insomnia (e.g., sedating antidepressants and sedating antipsychotics) should not be used in nursing home residents, except in those for whom other indications for use of these agents are present (e.g., depression, psychotic symptoms, agitation).	III	B
There is essentially no evidence to support the use of melatonin for sleep disturbance in nursing home residents with dementia.	I ¹⁸⁷	B
Any medication treatment for sleep disturbance in nursing home residents should be reviewed for effectiveness and adverse consequences, with frequent reevaluation to assess whether medication reduction or withdrawal is indicated.	III	A

See Table 1 for quality and strength of evidence codes.

temazepam treatment, but subjects taking medication performed worse on tests of neurological function and exhibited more daytime hypersomnolence.²⁹³ Another study in patients with Alzheimer's disease (including some participants in long-term care facilities) comparing melatonin with placebo found no significant differences between groups in objective sleep measures (wrist actigraphy) and only an isolated finding of better sleep quality with melatonin based on a caregiver rating.¹⁸⁷

The American Medical Directors Association has developed a clinical practice guideline that offers a 16-step approach to managing sleep problems in nursing home residents. They divide their approach into four different categories: recognition, assessment, treatment, and follow-up.²⁹⁴

For recommendations, see Table 11.

Recommendations for Future Research

- (1) Future research should clarify the consequences of sleep disturbance in nursing home residents, particularly in terms of effects on quality of life.
- (2) The effectiveness of pharmacological interventions to improve sleep in nursing home residents should be tested, with careful attention to the balance of potential risks and benefits in this vulnerable population.
- (3) More-effective behavioral and other nonpharmacological interventions must be identified that will help to

improve disturbed sleep patterns in nursing home residents.

- (4) The relationship between insomnia, sedative–hypnotic medications, and adverse events, including falls, in nursing home residents must be clarified.

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