HHS Public Access

Author manuscript

Clin Geriatr Med. Author manuscript; available in PMC 2015 November 23.

Published in final edited form as:

Clin Geriatr Med. 2014 August ; 30(3): 591–627. doi:10.1016/j.cger.2014.04.007.

Sleep In Older Adults: Normative Changes, Sleep Disorders, and Treatment Options

Nalaka S. Gooneratne[1,2] and **Michael V. Vitiello**[3]

[1]Division of Geriatric Medicine, Department of Medicine, University of Pennsylvania, Philadelphia, Pennsylvania

[2]Center for Sleep and Circadian Neurobiology and Division of Sleep Medicine, School of Medicine, University of Pennsylvania, Philadelphia, Pennsylvania

[3]Department of Psychiatry and Behavioral Sciences, School of Medicine, University of Washington, Seattle, Washington

1 Synopsis

Sleep disorders are common in older adults: Approximately 5% of older adults meet criteria for clinically significant insomnia disorders and 20% for sleep apnea syndromes. When considering insomnia symptoms, it is important to distinguish age-appropriate changes in sleep from clinically significant insomnia, with the latter distinguished by the presence of significant daytime symptoms such as fatigue. Evaluation with a sleep diary and screening for comorbid conditions, especially mood disorders, is essential. Non-pharmacologic therapies, such as cognitive-behavioral therapy for insomnia, can be highly effective and have sustained benefit. A broad range of pharmacologic therapies are also available but can have unwanted psychomotor effects. If left untreated, insomnia can be associated with increased risk of depression and significant impairments in quality of life. In regards to sleep apnea, a high index of suspicion is crucial for effective diagnosis because symptoms commonly noted in younger patients, such as obesity or loud snoring, may not be present in older patients. Diagnosis and management is fairly similar across age groups, except that a more nuanced approach to weight loss is warranted in older adults. The increasing use of home-based portable polysomnography and auto-titrating positive-airway pressure therapy can reduce barriers to treatment.

Keywords

Sleep apnea; positive airway pressure; insomnia; cognitive-behavioral therapy (CBT); sedative-hypnotic; polysomnography; depression; dementia

Contact Author: Nalaka S. Gooneratne, MD, MSc, Associate Professor, Division of Geriatric Medicine, Center for Sleep and Circadian Neurobiology, University of Pennsylvania School of Medicine, 3615 Chestnut Street, Philadelphia, PA 19104, O: 215 573 2048, F: 215 573 8684, ngoonera@mail.med.upenn.edu.

Conflicts of Interest: There are no author conflicts of interest related to this manuscript

4 Introduction

4.1 Overview of sleep

With the advent of new technologies to measure sleep, such as nocturnal polysomnography and frequent biomarker sampling, the medical community has gained an increasing appreciation of the significant influence that sleep disorders have on a broad range of medical and psychiatric diseases. This is particularly relevant for older adults due to their increased prevalence of medical comorbidities and age-related changes in sleep. This review will provide an overview of sleep and its assessment, and then focus on two of the most common sleep disorders that geriatricians are likely to encounter, insomnia and sleep apnea, along with a brief discussions of less prevalent sleep disorders that may also be relevant for the clinical care of older adults.

4.1.1 Sleep stages—Sleep is divided into non-rapid eye movement sleep (NREM) and rapid eye movement sleep (REM). NREM sleep is further subdivided into stage 1, 2 and 3 based on unique electroencephalographic (EEG) and electromyelographic (EMG) criteria. Stage 3 is of particular importance since it is a period of slow-wave EEG activity that correlates with the release of growth hormone. Selective experimentally-induced deprivation of stage 3 sleep has been associated with increased insulin resistance.[1] REM sleep is characterized by the presence of rapid eye movements as noted on electrooculography (EOG) and is commonly referred to as dream sleep. Most individuals have a progression through these stages, starting with Stage 1, ultimately reaching REM sleep. Most individuals will cycle through this progression four to five times per night. Thus, they may have three to five REM (dream) episodes, but may not recall any of them. Nocturnal arousals, as defined by episodes lasting at least three seconds characterized by acute changes in EEG activity, are also common during sleep, with an average of 27 arousals/hour noted in healthy older adults without sleep complaints, as compared to 10–20 arousals/hour in younger age groups.[2]

Prior to the modern era, night time sleep generally occurred in two major intervals which were bridged by a period of wakefulness that may have lasted for up to an hour.[3] This natural sleep pattern has been described as “split sleep” and these sleep episodes were frequently referred to historically as “first sleep” and “second sleep”.[3] This has been confirmed in clinical research studies where study participants were allowed to sleep for longer than the typical contemporary seven to eight hour sleep period.[4] This research suggests that the loss of the natural split sleep pattern is in part a result of the condensed (restricted) sleep window that characterizes the modern era with its access to 24-hour lighting due to electricity.[3]

In addition to growth hormone release during slow wave sleep, there are several other key hormonal changes that occur during sleep which highlight the importance of sleep in physiologic homeostasis. Cortisol tends to be highest shortly before awakening.[5] Melatonin levels generally have a significant increase at sleep onset, with a drop in melatonin levels prior to awakening.[6] A number of other hormones related to energy balance and metabolism also vary considerably as a function of the sleep-wake cycle.[7]

4.1.2 Changes with sleep with age—As with many other physiologic processes, there are a number of age-related changes in sleep that occur across the lifespan. Total sleep time decreases considerably from 10–14 hours a night in the pediatric age range to 6.5–8.5 hours a night as a young adult, then decreases at a slower rate into older ages, where average values may range from 5–7 hours a night.[8] When provided up to 16 hours a day to sleep, maximal sleep capacity, as indicated by total sleep time, also decreases with age, averaging 8.9 hours in younger subjects and 7.4 hours in older subjects.[9] Interestingly, assuming good health is maintained, the trend for total sleep time to continue to decrease with age tends to cease after age 60, at which point total sleep time plateaus.[8]

Other elements of sleep architecture also change with age, including time spent awake at night. While anecdotally we might expect sleep latency (the time it takes to fall asleep at the beginning of the night) to also increase with age, this is generally not the case.[8] Instead, we see prominent increases in wakefulness after sleep onset (WASO, or the tendency to be awake in the middle of the night or early morning).[8] The sleep stages also tend to change with advancing age such that there is less time spent in slow wave sleep (stage 3 sleep).[8] Understanding these physiologic reductions in sleep need with aging are important, especially in the context of helping older adults with insomnia complaints to adjust their normative expectations of sleep.

4.1.2.1 Subjective changes: These objective sleep/wake changes give rise to the common perception that sleep becomes more difficult with age. Interestingly, while there is indeed an objective deterioration of sleep as measured using polysomnography, there is a decoupling of subjective perception of sleep quality such that many older adults are less likely to complain of sleep problems than younger age groups. One population-based study in the United States found that the generic complaint of sleep disturbances, after controlling for covariates such as general health and depression, was highest in the 18–24 year old age group, peaked again in the 45–54 year old age group (for women only), but otherwise declined with age with the lowest rates of subjective sleep disturbance in older adults.[10] The American Insomnia Survey of 10,094 individuals also noted that self-reported insomnia rates were lower in older adults relative to young/middle aged cohorts.[11]

What might explain this decoupling between objective and subjective sleep quality in older adults? The impact of general health status on sleep quality may be a key component. The odds of having sleep disturbance are markedly increased with poor health, suggesting that poor health is a major driver of subjective sleep problems and that older adults with good health are less likely to have subjective sleep problems.[10] Ohayon, in his landmark review of epidemiological research on insomnia, concluded that “healthy elderly sleep as well as younger subjects”.[12] In addition, there are profound changes in an older adult’s perception of acceptable health status compared to younger adults. That is, while many older adults note objective sleep problems, they nonetheless perceive their sleep as acceptable.[13] This age-related adjustment of health expectations was noted by Brouwer and colleagues in a study in which individuals across the age spectrum were asked if it would be acceptable to live with extreme pain or severe impairment.[14] More than 10 percent of adults age 75 or older felt this was acceptable, as compared to 5 percent or less of adults under the age of 65.[14] (see Figure 1). Similar findings have been noted for sleep complaints,[15] with only

weak correlations between objective and subjective sleep in older adults.[16] A final consideration is the phenomenon of sleep debt, that is, the difference between the actual amounts of sleep an individual obtains as compared to the amount of sleep that they need. Older adults may have fewer daytime symptoms from fragmented nocturnal sleep because increased flexibility due to retirement allows time for daytime naps. The fragmented nocturnal sleep may then be perceived as less of a problem.

4.2 Cultural Perspectives on Sleep

The multi-racial nature of the US population means that many older adults may be immigrants or have close ties to cultures outside of the US. This has important ramifications within the context of sleep medicine in terms of reporting sleep complaints. Members of certain cultures and ethnic groups may be more or less likely to complain of poor sleep. For example, one study comparing the prevalence of insomnia symptoms in Europe, the US and Japan noted that the prevalence was 37.2%, 27.1% and 6.6%, respectively.[17] Another study of older American women found that the prevalence of general insomnia symptoms also varied considerably by ethnic group, ranging from approximately 70% in African Americans and European Americans to 35% for English-speaking Caribbeans or Haitians; race/ethnicity explained up to 20% of the variance of insomnia symptom prevalence.[18] While there may be biological factors underlying this difference, the large range suggests that cultural aspects that influence symptom perception may lead patients with a specific cultural background to either report or not report sleep problems.

4.3 Coping with Sleep Problems

There is a general tendency to believe that sleep disruption in older adults results in significant cognitive and functional consequences. Research examining psychomotor performance, however, suggests that older adults may actually be more tolerant of sleep deprivation than younger age groups. Stenuit and colleagues measured psychomotor vigilance task (PVT) performance after several nights of sleep restriction in women aged 20–30 years compared to older women aged 55–65 years.[19] They noted that both groups had similar PVT performance initially, but that the younger women had more prominent impairments with sleep deprivation than the older women by the third night of sleep restriction.[19] Another sleep restriction study that examined performance on a driving simulator noted similar findings.[20] One limitation of this literature is that the research study participants were restricted to a fixed number of hours each night, such as five hours. For a younger adult, this could result in three to four hours of sleep debt, but for an older adult, who has a lower average total sleep time, the sleep debt may only be two to three hours. Further research is thus needed to clarify whether older adults are actually more resistant to the effects of sleep loss than younger adults and whether this underlies age differences in sleep complaints.

4.4 Measuring Sleep

The past several years have seen a significant growth in sleep assessment tools available for clinical practice. This has been largely the result of changes in Medicare guidelines in 2008 which now allow for reimbursement of portable, at-home sleep studies. A typical in-lab sleep study records EEG, EOG, EMG, chest/abdominal movement, airflow and oxygen

saturation, amongst other physiological parameters. Portable sleep studies generally record a more limited set of physiological signals, usually respiratory effort, airflow, heart rate and oxygen saturation. This approach is generally more convenient for the patient but has several important limitations. First, because many portable sleep studies do not include EEG, devices have limited ability to determine if the patient is awake or asleep. Patients with insomnia can thus have artificially low sleep apnea indexes because the sleep apnea severity is calculated as the number of apnea or hypopnea events divided by the time spent asleep. Most portable devices start recording from the time the patient presses the start button on the device and stop based on patient input in the morning. They generally count this entire period as 'asleep' and use it to calculate the apneahypopnea index for sleep apnea severity. If the patient was awake during much of this time, however, they will not have any sleep apnea and this will artificially lower their apnea-hypopnea index. Second, patients must demonstrate adequate cognitive capacity or have a caregiver available throughout the night to assist with the device. Third, the patient must have the physical capacity in terms of upper arm strength and mobility, as well as adequate visual acuity, to use the device. Fourth, portable sleep studies are generally only useful for diagnosing or excluding sleep apnea and are of minimal benefit for assessing other sleep disorders.

4.5 Sleep Disorders

Sleep disorders can be broadly categorized into insomnias, hypersomnias, circadian rhythm disorders, sleep-breathing disorders, narcolepsy, parasomnias, and sleep movement disorders. A symptom-based diagnostic algorithm for sleep complaints is shown in Figure 2. This review will focus on the two most common sleep disorders seen in older adults: insomnia and sleep-breathing disorders and provide a much briefer discussion of several other sleep disorders including; include Restless Legs Syndrome (RLS), Periodic Limb Movement disorder (PLMD), REM behavior disorder (RBD), and the hypersomnias.

5 Insomnia

5.1 Insomnia Disorder description

Insomnia is broadly defined as "dissatisfaction with sleep". Insomnia as a diagnostic entity appears in numerous nosologies, including the International Classification of Sleep Disorders 2nd edition (ICSD-2), the International Classification of Diseases 10th edition (ICD-10) and the Diagnostic and Statistical Manual, version 5 (DSM-V). Research examining the concordance across nosologies has found that the prevalence of insomnia varies considerably depending upon the diagnostic nosology: insomnia prevalence is approximately 15% (across all adult age groups) for the ICSD-2, while it is only 4% when using the ICD-10 criteria. This is largely due to differences in the definition. All of the diagnostic approaches generally include symptoms of difficulty falling asleep at bedtime, waking up in the middle of the night with difficulty going back to sleep, or waking up too early in the morning again with difficulty returning to sleep. Of note, the perception that sleep is "non-restorative" is no longer an accepted diagnostic symptom for the DSM-V definition because it is felt to be too broad a complaint, but it remains in the ICD-10 criteria. In addition, the nosologies usually require a frequency of at least three nights per week. Duration requirements vary, with the ICD-10 requiring a duration of one month, while the

recently revised DSM-V proposed a symptom duration of at least three months.[21] The major reason for the lower prevalence using the ICD-10 is the requirement that the diagnosis of insomnia be associated with a "preoccupation with the sleeplessness and excessive concern over its consequences at night and during the day and the unsatisfactory quantity and/or quality of sleep either causes marked distress or interferes with ordinary activities in daily living".[11, 22]

Since the ICD-10 is used commonly in clinical practice and will be mandatory for billing and reimbursement after October 2014, we will focus on this diagnostic schema. The ICD-10 defines insomnia as either "organic insomnia" (G47.--) or "non-organic insomnia" (F51.--), with the former referring to insomnia that occurs as a result of another medical condition/substance and the latter being independent of any known substance or medical condition. Within these categories, the most common ICD-10 diagnoses are shown in Table 1. It is important to highlight that the recently released DSM-V has collapsed several of these categories in favor of a more streamlined, symptom-based diagnostic approach.[21] In particular, the DSM-V emphasizes the concept of a more general insomnia disorder, as opposed to requiring delineation of causal attribution.[23] This was motivated in part due to low rates of diagnostic concordance when attempting to classify insomnia into specific categories, such as primary insomnia or insomnia due to other conditions.[24]

5.2 Scope of the problem

The overall prevalence of insomnia depends in large part on how it is defined. Most epidemiological studies note a prevalence of 20–40% for nocturnal insomnia complaints, such as difficulty initiating/maintaining sleep or unrefreshing sleep.[11, 12] When including daytime symptoms of fatigue or impaired concentration, the prevalence decreases to 10–20%.[12] When diagnostic nosologies such as the ICD-10 are applied, the prevalence is approximately 2–5%.[11] A key factor driving the prevalence of insomnia is the rate of incidence, remission and relapse. A study of 6,899 older adults observed an incidence rate for insomnia symptoms of approximately 5% per year.[25] Of note, this study extrapolated the one year incidence rate based on a three-year follow-up interview. Research using a one year follow-up noted a yearly incidence rate of 7.97% in older adults.[26] Recent research (across all age groups) that used a more frequent sampling period of 1 and 3 months, extrapolated an annual incidence rate of 31.2%.[27] Approximately half of the patients with insomnia symptoms will have remission during the follow-up period, with higher remission rates in older adult males relative to females.[26, 28] Another study across all age groups that used DSM-V criteria for insomnia noted that while the majority (78.6%) would have remission of their insomnia, a sizeable minority of 21.4% developed chronic insomnia[27] and that insomnia is more likely to be persistent in older adults.[29]

A commonly used framework for understanding the etiology and persistence of insomnia is the three factor model (see Figure 3), as conceptualized by Spielman and colleagues, which identifies predisposing, precipitating and perpetuating factors.[30] These factors combine to raise the likelihood of insomnia above the insomnia threshold as shown in the figure. Examples of predisposing factors are demographic characteristics. Women over the age of 45 are approximately 1.7 times more likely to have insomnia than men.[12] Individuals who

are divorced, separated or widowed are also more likely to have insomnia.[12] Lower levels of education attainment or income have also been linked to higher rates of insomnia in some, but not all studies.[12, 31] Smoking and alcohol use are also associated with higher rates of insomnia.[12] Reduced physical activity has been linked to higher rates of insomnia in older adults.[32] Recent research suggests that there may also be genetic variants in clock genes that can influence sleep parameters in older adults.[33]

Precipitating factors are generally acute life stressors or medical conditions that may disrupt sleep. Individuals with respiratory symptoms, physical disability and fair to poor perceived health are at increased risk of developing insomnia.[25] Medications may be one of the factors which contribute to developing insomnia in patients with multiple medical conditions. Examples of such medications include beta-blockers, glucocorticoids, NSAIDs, decongestants, and anti-androgen agents. Depression was also a major risk factor for incident and persistent insomnia.[25, 28] Another study of 1,814 patients (mean age of 57 years) that completed a 2-year follow-up assessment noted that the presence of major or sub threshold depression, even when adjusting for baseline insomnia, was associated with an odds ratio of 1.7 and 2.4, respectively, for developing insomnia.[34] High rates of insomnia also occur in patients with generalized anxiety disorder, with up to 90% reporting insomnia symptoms.[35] These studies, along with several others, highlight the profound role that mental illness and medical conditions have on disrupting sleep.

Perpetuating factors result in a persistence of insomnia. Once a patient with predisposing and precipitating factors has developed acute insomnia, this acute insomnia will not necessarily develop into chronic insomnia unless these perpetuating factors are present. These perpetuating factors are often behavioral or cognitive changes that arise as a result of the acute insomnia. For example, a patient may develop increased pre-sleep anxiety due to fear of having another night of insomnia (conditioning), or they may use TV or radio to help “relax” or occupy their time while waiting to fall asleep. Others may start to have variable bed times to deal with insomnia or take daytime naps; while naps are generally not recommended for patients with insomnia, for older adults without insomnia complaints, it is important to note that naps have minimal side effects[36] and improve vigilance/performance post-naps.[37] Addressing these perpetuating factors is an important goal of non-pharmacologic insomnia treatment as discussed further below.

5.3 Insomnia Clinical Correlations

As noted earlier, while the prevalence of objective changes in sleep with aging is high, many older adults do not complain of significant sleep problems. An important clinical ramification of this is that when older adults do complain of insomnia symptoms that are causing significant daytime distress and meet criteria for insomnia disorder, this complaint should be taken seriously by the clinician. Clinicians should therefore ask themselves what is prompting their patient to bring forward this complaint and seek assistance when the majority of older adults do not have significant distress from age-related objective changes in sleep.

If left untreated, insomnia can be associated with significant morbidity across a variety of domains. The strongest level of evidence entails mental illness. Older adults with insomnia

symptoms have a 23% increase in their risk of developing depression symptoms.[38] Several other studies have also suggested an increased risk for depression in older adults, especially those with persistent insomnia.[39] Cole and Dendukuri, in a meta-analysis of risk factors for depression in the elderly, noted that sleep disturbance had an OR of 2.6, higher than that of prior depression or disability.[40] Furthermore, data from the IMPACT study of older adults with depression noted that 44% of patients with persistent insomnia continued to have depression at the 6 month time point as compared to only 16% of those without insomnia.[41] Depression recurrence is also markedly increased in older adults with persistent sleep disturbances, with one study noting an adjusted hazards ratio of 16.1.[42] Of particular concern, insomnia may be a risk factor for suicide, however, research studies have had conflicting findings in this area, with some showing an increased risk of suicide,[43] while others found no increased risk in older adults.[44]

Understanding the relationship between insomnia and mental disorders is complex because they may have a bidirectional relationship.[45] That is, patients with insomnia at baseline have higher risks of developing anxiety or depression, and those with anxiety or depression at baseline have a higher risk of developing insomnia.[45] The insomnia symptoms most strongly associated with subsequent depression risk are poor sleep quality and difficulty initiating and maintaining sleep, while early morning awakening is not a risk factor.[38] When exploring the pathway through which insomnia may increase the risk of depression, Sadler and colleagues postulated that hopelessness and maladaptive sleep beliefs were important components.[46] Another aspect of the insomnia-depression association is that insomnia is one of the defining symptoms of depression, thus many of the assessment tools used to identify depression include questions related to insomnia. Furthermore, insomnia questionnaires that inquire about daytime fatigue or impairment will often be endorsed by patients with depression. Research studies that have removed sleep-related items from depression scales, however, continue to show evidence of bidirectional links between insomnia and depression.[42]

In addition to mental health effects, older adults with insomnia may be at higher risk for other medical conditions and impaired quality of life. A meta-analysis of insomnia symptoms and their association with heart disease, after adjusting for age and other cardiovascular risk factors, found that risk ratios for heart disease from insomnia symptoms ranged from 1.47 to 3.90.[47] Recent research suggests that the insomnia symptoms may lead to increased rates of cancer, such as prostate cancer.[48]

While many older adults with insomnia complain of next-day neurocognitive difficulties, research examining daytime neuropsychological or cognitive performance has shown no evidence of significant impairments.[49, 50] Long-term, insomnia complaints may be associated with increased risk of cognitive impairment,[51] although this has not been noted in all studies.[52] Interestingly, recent research using wrist-activity monitors to identify objective sleep fragmentation (as opposed to self-reported insomnia symptoms) observed that study participants in the highest 10% bracket of sleep fragmentation had a 50% increased risk of developing Alzheimer's Disease compared to those in the lowest 10% bracket over an average 3.3 years of follow-up.[53]

Older adults with insomnia also tend to have lower quality of life assessment scores.[54] However, this is generally not associated with significant impairments in self-reported overall functional status.[55] When considering objective measures, such as wrist-activity monitoring of activity, next-day increases in physical activity with reduced sleep fragmentation were noted, although the overall effects were small (the researchers estimated that a 10% increase in sleep efficiency (which is a large change) was associated with only a 3.2% increase in next day activity).[56] Furthermore, the data linking insomnia to mortality is equivocal, with several studies finding no strong link between insomnia symptoms and mortality.[57–59]

5.4 Diagnostic Standards and Dilemmas for Insomnia Disorder

As noted earlier, one of the main challenges in evaluating insomnia symptoms is determining if they are related to another condition, such as chronic pain or depression, or whether they exist as a primary diagnosis. To minimize this diagnostic dilemma, the revised DSM-V guidelines use the term “insomnia disorder” to avoid the need to differentiate insomnia into specific subtypes such as those in the ICD-10.[23] There remain, however, several important alternative diagnoses that should be considered. These include the presence of other sleep disorders, such as sleep apnea or periodic limb movement disorder (see Figure 2).[60, 61] Older adults with both insomnia and sleep apnea may be at a particularly increased risk of functional impairments.[62]

Another important category of sleep disorders that is often associated with insomnia complaints is that of circadian rhythm disorders. While there is a general perspective that older adults experience advanced sleep phase (i.e., a tendency to go to sleep early and wake early), this may not necessarily be the case for older adults with insomnia symptoms. Youngstedt and colleagues noted that older adults with insomnia complaints were more likely to have delayed circadian phase than advanced circadian phase, and had significant circadian dispersion and malsynchronization compared to young, healthy subjects.[63] Understanding this distinction is important because circadian rhythm disorders generally respond well to chronotherapy using appropriately timed melatonin and light therapy, thus avoiding the need for sedative-hypnotics; detailed clinical practice guidelines for circadian rhythm disorders are available.[64]

5.5 Insomnia Disorder Clinical Findings

It is important to emphasize that the diagnosis of insomnia is a clinical one that relies on history obtained from the patient. In cases where the patient is not a reliable informant, history may be obtained from a proxy caregiver. While specific cut-points for sleep onset latency (18–30 minutes) or wakefulness after sleep onset (21–60 minutes) have been proposed, they generally should not be used as diagnostic clinical criteria, but instead as general treatment goals.[65, 66] As is the case with many other mental health disorders, there is no objective test used to diagnose insomnia. Neither are there any specific physical exam findings.

5.5.1 Examination—When evaluating a patient with insomnia symptoms, a variety of tests may be useful to rule-out possible alternate diagnoses. Possible laboratory tests that

may be worthwhile depending upon the presence of other associated clinical symptoms are listed in Table 2.

5.5.2 Recommended rating scales—There are a number of insomnia rating scales available that are useful both for cataloging baseline symptomology and for gauging treatment progress. A list of the more useful appears in the adjacent box enclosure (Box 1).

5.5.3 Diagnostic modalities—In addition to the medical and sleep history, the essential diagnostic tool for evaluating insomnia is the sleep diary. This is a nightly record of sleep patterns and disruptions, as well as attempted treatments and sleep hygiene factors. Recently, a consensus sleep diary has been proposed that incorporates the recommended measures of bedtime, sleep onset latency (SOL), nocturnal awakenings, wakefulness after sleep onset (WASO), terminal wakefulness (TWAK), total sleep time (TST), final awakening time, and sleep efficiency (SE) as well as sleep medication use, and daytime napping, caffeine and alcohol consumption.[67, 68] The recommended duration of a sleep diary is ideally two weeks in order to provide a comprehensive perspective on a patient's sleep patterns due to day-to-day variability. Several organizations, such as the National Sleep Foundation, provide free on-line sleep diaries that can be printed and provided to patients (http://www.sleepfoundation.org/sleep-diary/SleepDiaryv6.pdf).

In addition to the sleep diary, other diagnostic modalities that can be used include wrist activity monitoring (actigraphy; a portable accelerometer worn on the wrist).[60, 68, 69] While not required for the diagnosis of insomnia, actigraphy can be used to monitor the response to therapy and to screen for circadian rhythm disorders.[69] Of note, the specific parameters used for the actigraphy analysis can significantly influence the findings, especially in older adults with intellectual impairments,[70] research attempting to define actigraphy cut points for insomnia diagnosis has been unsuccessful,[66] and there can be considerable discordance between self-report sleep diaries and actigraphy in older adults.[71]

Polysomnography is not required for the evaluation of insomnia and is generally not recommended for routine cases.[72] However, polysomnography is warranted in certain specific situations, such as when sleep apnea or another sleep disorder is suspected (i.e., a history of limb movements, loud snoring/witness apneas/nocturnal choking or violent behavior is obtained), or the insomnia persists despite treatment.[72] Other sleep diagnostic tests, such as the Multiple Sleep Latency Test (MSLT) and Maintenance of Wakefulness Test (MWT) are not indicated for the evaluation or management of insomnia patients unless there are potential occupational safety risks from daytime fatigue.[73]

5.6 Insomnia Disorder Management

Effective management of insomnia requires setting realistic goals with the patient. Insomnia can be a chronic condition which can be significantly improved, but patients may not ultimately achieve an idealized version of sleep characterized by rapid sleep onset and sustained sleep with the complete absence of next day fatigue. Furthermore, patients may relapse or have occasional nights of poor sleep. Helping patients to adjust their expectations to fit the natural course of sleep changes with aging can thus be crucial in coping with insomnia.

5.7 Interventions/ Current evidence base

A summary of the recommendations for the treatment of insomnia are presented in Table 3. Treatment can be divided into non-pharmacologic and pharmacologic approaches. Due to the risks of side effects with pharmacologic approaches, and the potential for benefits to attenuate over time in comparison to non-pharmacologic approaches, older adults should first receive non-pharmacologic treatment for several months prior to implementing pharmacologic therapy.[74, 75]

5.7.1 Nonpharmacologic—While there are numerous non-pharmacologic options, the most commonly used include sleep hygiene and cognitive-behavioral therapy for insomnia. Several of these behavior-based approaches have been found to be effective even in patients with cognitive-impairment.[76]

5.7.1.1 Sleep Hygiene: Sleep hygiene consists of several interventions that promote a stable sleep pattern and non-disruptive environment. It is not realistic to expect a patient to engage in all of these sleep hygiene practices at once—one approach is to prioritize the most relevant sleep hygiene practices based on the patient's sleep history and implement changes in a serial fashion. Sleep hygiene practices grouped by general category are summarized in the adjacent boxed text (Box 2). While sleep hygiene is often used as an initial step in the treatment of insomnia, the evidence suggesting that it is effective as monotherapy is limited.[77]

5.7.1.2 Cognitive-Behavioral Therapy for Insomnia (CBT-I): When sleep hygiene alone is not effective, cognitive-behavioral therapy for insomnia (CBT-I) is a reasonable next step. CBT-I generally consists of six to ten sessions with a trained therapist that address counterproductive behaviors and cognitive beliefs that perpetuate insomnia. It has been found to be effective in older adults across multiple studies.[78–80] Core elements of CBT-I are shown in the boxed text (Box 3).

While most research has been conducted with up to eight sessions of CBT-I, a growing body of evidence suggests that brief behavioral treatment (BBT) or computerized/internet-based CBT-I can also be effective.[81, 82] CBT-I can also be useful to taper patients off pharmacologic therapy, but maximal effects may take months to manifest.[83] In addition to primary insomnia, it may also be effective in patients with insomnia and chronic pain,[84] comorbid psychiatric conditions,[85] or cancer.[86]

Other non-pharmacologic interventions for insomnia with limited evidence include bright light therapy and exercise.[80]

5.7.2 Pharmacological—Pharmacotherapy options are benzodiazepine sedatives, nonbenzodiazepine sedatives, melatonin-receptor agonists, and anti-depressants. A listing of the available FDA-approved compounds, their doses, and key side effects are listed in Table 4. Both benzodiazepine and nonbenzodiazepine sedatives bind to GABA-A receptors, with the main difference being that the non-benzodiazepine sedatives are more selective for the alpha-1 subclass, which is closely linked to sedation, but has minimal anxiolytic effects. Risks associated with sedative-hypnotics include next-day sedation, confusion, falls and

worsening depression/suicide; these have been identified by the FDA as warnings/precautions on many of the package inserts. While concerns have been raised regarding long-term mortality risks from sedatives, a recently published 12-year observational study that controlled for multiple covariates found no evidence of an increased mortality rate in sedative users and noted that comorbid psychiatric disorders may be important confounders.[87] One option to reduce the risk of habituation or dependence is to adopt an intermittent dosing approach in which patients are given 10–15 tablets for the entire month and are allowed to use them on an as-needed basis.[88] Other FDA approved agents include melatonin-receptor agonists, such as ramelteon, which have shown efficacy in older adults for sleep initiation insomnia.[89] It is not associated with dependence or nocturnal gait instability, but there are conflicting results regarding next-day morning sedation.[90–92]

Diverse categories of antidepressants have also been used for insomnia, including tricyclic antidepressants (TCA), phenylpiperazine compounds (trazodone) and noradrenergic/specific-serotonergic antidepressants (NaSSA, i.e. mirtazapine); of these, only low-dose doxepin, a TCA, is FDA approved for insomnia, and uncertainty exists regarding the role of other agents due to the paucity of randomized clinical trials in older adults.[93] In general, antidepressants may be appropriate for insomnia symptoms when there is a diagnosis of depression, or sub-threshold depression.[93] Clinically, trazodone is widely used for insomnia in older adults at doses ranging from 50–100 mg at bedtime, which are below the dose range for antidepressant effects, despite limited clinical trial evidence.[94] It is also important to note that serotonin-specific reuptake inhibitors (SSRIs) tend to worsen insomnia initially.[95] However, antidepressants with strong 5-HT2 antagonism, such as mirtazapine or trazodone, may improve insomnia and may be preferred to agents that create sedative effects solely through histamine receptor antagonism.[95, 96] Mirtazapine should not be used solely for the treatment of insomnia in the absence of depression symptoms due to conflicting evidence.[97] Additional treatment options for insomnia in the setting of depression are available.[98] Potential future pharmacologic agents for insomnia include orexin-receptor agonists, although as of 2013, none have yet been FDA approved. For post-menopausal women, hormone replacement therapy can lead to objective reductions in nocturnal awakenings and wakefulness after sleep onset.[99]

A variety of alternative medicine options are also available for insomnia, but there is a relative paucity of data demonstrating clinical efficacy in older adults.[100] One exception is melatonin, where one randomized clinical trial demonstrated efficacy in older adults for up to six months.[101] An important aspect of this particular trial is that the melatonin was taken approximately two hours before bedtime, suggesting that circadian phase aspects of the melatonin may have led to observed treatment benefits rather than any direct effect on sleep, per se.

5.8 Combination Therapies

CBT-I can be used in combination with pharmacotherapy. In one study, concomitant CBT-I and temazepam use was associated with reduced temazepam use as compared to the temazepam only arm.[102] Another study examining combination therapy noted that the maximal sleep benefits were noticed in the combination therapy arm, but that patients who

completed combination therapy with CBT-I/pharmacotherapy and then tapered off pharmacotherapy had improved remission rates than those that did not taper off their pharmacotherapy and continued to use pharmacotherapy.[103]

5.9 Treating Insomnia Disorder in Institutionalized Settings and in Cognitively Impaired Patients

Institutionalized older adult patients are at risk of insomnia due to the increased prevalence of comorbid conditions and medications that can exacerbate insomnia, as well as environmental factors related to the institutional setting. Loud noises, for example, may be common in institutional environments. However, it is important to note that the majority of these loud noises are not from patient vocalizations, but instead from equipment, and staff and that incontinence care frequently contributes to nocturnal awakenings.[104] Interventions to reduce noise and other environmental disruptions, however, have had minimal benefit and should be combined with behavioral approaches.[105] Low levels of light exposure and other circadian rhythm cues in institutional settings can also increase the risk of developing circadian rhythm disorders that may manifest with symptoms suggestive of insomnia. Bright light therapy and melatonin have been effective in institutionalized settings for these circadian rhythm sleep disorders.[106] CBT-I can also be effective: an open-label, non-controlled study of CBT-I in nursing home elderly demonstrated improvements in sleep quality.[107] For community-dwelling older adults with cognitive impairment, a combined intervention that included sleep hygiene, increased activity (walking) and light therapy has been effective in reducing time awake at night and nighttime awakenings.[108] Pharmacotherapy, as discussed previously, can also be used for treating insomnia in institutionalized patients or cognitively impaired elders, however, there is little data regarding safety and efficacy in these populations, thus non-pharmacological approaches should be implemented first.[76, 109]

5.10 Caregiver perspectives for Insomnia Disorder

Caregivers for dementia patients frequently identify nocturnal awakenings by their care recipient to be significantly distressing behaviors that adversely impact the caregivers' quality of life.[110] These nocturnal sleep difficulties can be a contributing factor in the decision by a caregiver to institutionalize an older adult with cognitive impairment.[111] Caregivers' personal appraisal of their situation, which is influenced by their coping skills, affect and social support network, can be important mediators of the disruption the caregivers perceive from their care recipients' nocturnal awakenings.[110] CBT-I and exercise therapy for caregivers have both been shown to improve caregiver sleep quality.[112, 113]

6 Sleep Apnea

6.1 Sleep Apnea description

Sleep apnea, also referred to as sleep-disordered breathing and sleep-related breathing disorder, is a condition in which respiration ceases or decreases considerably in volume during sleep. It can be due to either obstruction of the upper airway (obstructive sleep apnea), dysfunction in the neurological drive to breath (central sleep apnea, Cheyne-Stokes breathing, medications/substance abuse) or their combination (mixed apnea, complex apnea,

or obesity-hypoventilation syndrome). It is generally defined by the apnea-hypopnea index (AHI) which measures the number of apneas (cessation of breathing for at least ten seconds) or hypopneas (reduction in the volume of breathing with associated evidence of neurological arousal or oxyhemoglobin desaturations).

The range for sleep apnea severity as a function of AHI is as follows: >=5 up to <15 events/hr. is mild; >=15 up to <30 events/hr. is moderate; and >=30 events per hour is severe.[114] Medicare guidelines will cover sleep apnea treatment if the AHI >=15 events/hr., or if it is >=5 events/hr. and the patient has clinical symptoms of excessive daytime sleepiness, impaired cognition, mood disorders or insomnia; or a medical history of hypertension, ischemic heart disease or stroke. The majority of clinical research studies on sleep apnea use an AHI>=15 events/hr. (independent of symptoms) as the diagnostic criteria; we will use this criteria for the remainder of this review unless otherwise mentioned.

6.2 Scope of the problem

The prevalence of sleep apnea can vary considerably based on the AHI criteria. Using a criteria of >=5 events/hr., the prevalence of sleep apnea in older adults is approximately 50%, while at a criteria of >=15, it is approximately 20%.[115] There is a clear age-related increase in the prevalence of sleep apnea since only 10% of young adults have an AHI >=15 events/hr.[115] The prevalence of sleep apnea is increased in certain medical conditions, most notably congestive heart failure, atrial fibrillation, cerebrovascular disease, and dementia, where prevalence rates for an AHI>=15 events/hr. may be as high as 50–80%.[116, 117] The likelihood of having central sleep apnea variants is also increased in cardiovascular/cerebrovascular disease.[116] Sleep apnea is more common in Asians than in Caucasians by an odds ratio of 2.1, while most data suggests that African-Americans have prevalence rates similar to Caucasians.[118] Thus, the overall prevalence of sleep apnea is high, and it should be considered in the evaluation of a broad range of conditions as discussed further below. Unfortunately, it is frequently under-diagnosed: for example, sleep apnea was diagnosed and treated in only 2% of incident congestive heart failure patients, though, as previously noted, the prevalence of sleep apnea in congestive heart failure is greater than 50%.[119]

6.3 Sleep Apnea Clinical Correlations

Risk factors for sleep apnea are shown in Table 5. In general, these risk factors are similar to those for younger age groups with four important differences. First, while obesity is a risk factor for older adults, it decreases in importance with age. This is likely due to the fact that older adults have increased upper airway collapsibility relative to younger subjects, and other factors that are not obesity-dependent play a role in older adults.[120, 121] Second, while sleep apnea is more common in young/middle aged males than females, for older adults there is an equal prevalence by gender which starts after the menopause transition.[122] Third, the diagnosis can be challenging because the clinical presentation can be subtle. For example, while snoring is a common complaint of partners of younger patients, many older adults will not endorse snoring as a symptom because they may not have a bed partner.[122] In these cases, it can be helpful to inquire if snoring was noted during vacations or when napping. Fourth, the edentulous state markedly increases the risk of having sleep apnea.[123]

As noted previously, sleep apnea is common in patients with cognitive impairment, with one study of institutionalized severe AD patients observing a 63% prevalence.[117] One mechanism for this is found in Alzheimer disease-related reductions in cholinergic neural activity: cholinergic neurons play a role in upper airway motor regulation and thalamic cholinergic neurons influence respiratory drive, thus damage to these neurons could increase the risk of sleep apnea in Alzheimer disease patients.[124]

In addition to cognitive impairment potentially increasing the likelihood of sleep apnea, it is also highly likely that sleep apnea increases the likelihood of cognitive impairment. A prospective observational study of 298 women, average age of 82.3 years and without dementia at baseline followed for an average of 4.4 years, found that those with sleep apnea had a striking 85% increase in the risk of developing mild cognitive impairment or dementia, even after adjusting for other risk factors.[125] Furthermore, oxyhemoglobin desaturation, likely related to sleep apnea, was a particularly important risk factor, suggesting that oxyhemoglobin desaturation is a major mediator of neurocognitive consequences of sleep apnea.[125] Further evidence of the importance of sleep apnea in the context of cognitive impairment can be seen in positive airway pressure treatment studies which have shown preliminary signs of improvements in cognitive status in older adults with dementia[126] and in neuro-imaging in non-demented/ non-elderly patients,[127] although many cognitive deficits persist and may be irreversible.[128]

Several research studies, mostly in younger cohorts, have shown links between sleep apnea and depression,[129, 130] with one longitudinal study demonstrating an odds ratio of 2.6 for developing depression in those with moderate or worse sleep apnea over four-year follow-up intervals.[131] Others have noted that it is not the severity of sleep apnea as measured by the apnea-hypopnea index, but rather daytime sleepiness and fatigue that are correlated with depression.[132–135] Gender and type of depression symptoms may also be a factor: somatic depression complaints were associated with sleep apnea severity in males only.[136] Interestingly, treatment with armodafinil, a stimulant, improved sleepiness but not depression.[137] CBT for depression in post-MI patients was less beneficial in those who had sleep apnea as compared to those without sleep apnea.[138] Attempts to use CPAP for treating sleep apnea in depression have had more success, with improvements in depression scores that may be attributed to reduced daytime sleepiness.[139] The relationship between anxiety and sleep apnea is unclear, with some research finding no significant associations.[140, 141]

Sleep apnea has also been found to increase the risk of other medical conditions. A longitudinal study of incident congestive heart failure found that each 10 unit increase in the AHI led to a 13% increase in heart failure in men, but not in women.[142] Similarly, the rate of incident stroke was increased in subjects with sleep apnea, especially at higher AHI levels.[143, 144] In one cross-sectional research study, sleep apnea was associated with the frailty syndrome only in older females, but not in older males.[145] Sleep apnea has also been linked to higher rates of postoperative delirium.[146] Due to the potential increased peri-operative risk, screening for sleep apnea by history and exam is recommended prior to surgery, with subsequent polysomnography evaluation and treatment if necessary.[147]

Despite the increased rate of various comorbidities as a result of sleep apnea, research examining sleep apnea and long-term mortality in older adults has had conflicting findings. One of the largest epidemiological studies, the Sleep Heart Health Study, noted an increased mortality rate for an AHI>=15 events/hr. only in study participants under the age of 70.[148] Several factors may explain this observation. First, it is possible that a higher AHI criteria, such as >30 or >40 events/hr., may be appropriate for older adults as several studies have noted an increased mortality rate in older adults in the setting of sleep apnea at these higher cut-points.[149, 150] Second, the inclusion of symptoms, such as sleepiness, as part of the diagnostic criteria for sleep apnea can markedly impact findings: one study noted that in the absence of symptoms of daytime sleepiness, older adults with only an elevated AHI (AHI>=20 events/hr.) had mortality rates similar to those with an AHI <20 events/hr. (approximately 35% at ten years) as compared to older adults with an AHI>=20 events/hr. and daytime sleepiness complaints, who had a markedly increased mortality rate of approximately 70% at ten years.[151] Third, since older adults are at increased risk for mortality from their other comorbid medical conditions, these competing risks for mortality may reduce the impact of sleep apnea alone.[152, 153] Of note, a recent long-term quasi-experimental study that compared older adults with treated sleep apnea to older adults with untreated sleep apnea noted markedly improved survival rates with treatment.[154]

6.4 Diagnostic Standards and Dilemmas for Sleep Apnea

The diagnosis of sleep apnea requires overnight polysomnography. As noted earlier, this previously necessitated an overnight stay at an in-lab sleep facility, however, the Centers for Medicare and Medicaid Services has approved portable, at-home testing. While portable testing provides several additional options for convenient diagnosis of suspected sleep apnea that can shorten the time to diagnosis and treatment, it is important to keep in mind the potential limitations discussed earlier (see “Measuring Sleep”).[155–157] Prior to ordering the polysomnography, a face-to-face physician evaluation is necessary during which key history and physical elements must be documents as outlined in “Clinical Findings” below; this is necessary to help ensure that Medicare will cover the costs of treatment. A repeat polysomnography may be warranted if there is a high clinical suspicion of sleep apnea in the setting of a negative initial polysomnography: AHI variability of >10 events/hr. has been noted in 18% of older adults.[158]

There are three major diagnoses within the category of sleep apnea. Obstructive sleep apnea is characterized primarily by obstructive apneas (complete cessation of breathing with evidence of upper thoracic respiratory effort as detected by abdominal or respiratory belts), whereas in central sleep apnea, there are at least five central apneas per hour (cessation of breathing with absence of thoracic effort indicating a central origin). Obesity-hypoventilation syndrome (also known as sleep related hypoventilation) is diagnosed by increasing hypercapnia with sleep.[159] There is often daytime hypercapnia ($PaCO_2$ > 45 mm Hg) and obstructive/central apneas as well.[159] It is generally felt to be due to extreme obesity and/or neuromuscular/chest wall disorders, such as kyphosis.

6.5 Sleep Apnea Clinical Findings

The clinical history physical exam for sleep apnea is similar for older adults as it is for younger age groups. Important questions include asking about the presence of snoring, witnessed apneas, or nocturnal choking, gasping or dyspnea. While many clinicians assess daytime sleepiness, less well recognized is the fact that insomnia is another possible diagnostic symptom of sleep apnea.[159] The physical exam should include a cardiopulmonary exam and a head and neck exam to confirm that there is no asymmetry suggestive of a mass effect that may compress the upper airway, such as a goiter or enlarged lymph nodes. For suspected central sleep apnea, the examination should also include a detailed cardiovascular, neurologic and chest/back musculoskeletal assessment as these may all contribute to the pathophysiology. Unique aspects relevant to older adults are presented in Table 6. At present, there is no laboratory or radiographic tests that can be used to diagnose sleep apnea.

6.6 Sleep Apnea Management 6.7 Interventions/ Current evidence base

The treatment of sleep apnea in older adults has several similarities to younger age groups. The major difference relates to the recommendation for weight loss, which is commonly suggested for younger subjects for whom obesity is often a major risk factor. For older adults, the link between obesity and sleep apnea is attenuated and thus it is possible to have older adults at ideal body weight who have sleep apnea. Furthermore, weight loss, even for overweight or obese older adults, can be problematic. Epidemiological studies have noted that overweight older adults have improved survival rates relative to those at ideal body weight, with one study noting that every unit increase in the BMI led to an improvement in the survival rate of 1–8% even after controlling for other mortality risk factors.[160] This is possibly due to increased amounts of physiologic reserve (in terms of nutrition, etc.) in overweight older adults that allow them to tolerate acute illnesses. Clinical trials are few, however, observational studies have noted improvement in sleep apnea in morbidly obese older adults post bariatric surgery.[161, 162] For these reasons, weight loss, while often a universal recommendation for younger patients with sleep apnea, may not be appropriate for many older adults and a more nuanced approach may be warranted.

Sleep apnea is frequently worse when supine because it is easier for the tongue to prolapse backwards and obstruct the upper airway in this position. As such, advising patients to sleep on their side (lateral position) can be effective in some cases. The potential utility of this “positional therapy” can be determined from polysomnography, which records sleep apnea severity in the supine and lateral positions. There are several commercial aids/pillows that can be used by the patient to help keep them in the lateral position while sleeping.

If positional therapy is not an option, positive airway pressure (PAP, also referred to as continuous positive airway pressure or CPAP) therapy is an appropriate initial treatment recommendation. It is highly effective when used consistently. One concern is that some older adults have upper extremity weakness that may interfere with their ability to apply a positive airway pressure mask.[153] Cognitive impairment can also limit adherence, however, the active assistance of a caregiver can help promote adherence even in those cases.[163] The appropriate PAP settings are usually determined either during an in-lab titration sleep study, or by an autotitrating PAP device used at home for several weeks. While auto-titrating PAP

can be more convenient, it is not recommended for patients with severe congestive heart failure, chronic obstructive pulmonary disease or central sleep apnea.[164] If a patient is initially treated with an auto-titrating PAP device, but does not experience symptom improvement, an in-lab titration study may be warranted.

The major limiting factor for PAP use is patient adherence: approximately 40–50% are adherent.[165] However, overall adherence to PAP in older patients is similar to younger ones.[166] PAP therapy can also be used effectively in dementia patients with good adherence rates, especially if there is caregiver involvement and patients do not have significant levels of depression.[163] Several strategies can be used to help patients adjust to PAP, including trying alternate masks, heated humidification, and short daytime trials to encourage habituation. Cost can also be an important consideration. The first year of PAP therapy obtained through a durable medical equipment (DME) provider can generate charges of up to $1,500–2,000; while the majority is covered by Medicare, for older adults with co-pays, the cost for the first year could still be several hundred dollars. For Medicare to cover PAP therapy long-term, the patient must use the PAP device for a minimum of four hours a night on 70% of the nights for at least a one month period during the initial three month trial. In addition, they must have a face-to-face encounter with the ordering physician to document clinical response at some time between months one and three.

Despite these challenges, when used consistently, PAP therapy can be highly effective in older adults, even in those with diminished cognitive capacity. A landmark quasi-experimental study that examined outcomes of PAP treatment in older adults found that those who were adherent had overall survival rates that were similar to patients without sleep apnea.[154] The mortality rate over a median follow-up of 5.75 years was 16.7% for older adults without significant sleep apnea, 16.1% for those with sleep apnea that were adherent to PAP, and 34.1% for those with severe, untreated sleep apnea.[154]

Oral appliances are another option for the treatment of sleep apnea that is primarily obstructive. These devices resemble a mouth-guard and move the lower mandible forward by several centimeters. Since the tongue is attached to the lower mandible, this has the effect of opening up the retroglossal space, a key site of obstructive apneas and hypopneas. While PAP is more effective than an oral appliance in lowering the AHI into the normal range, the improved adherence to oral appliances leads to similarities in average outcomes across all study participants.[167] For patients with more severe sleep apnea, though, PAP may be more effective.[168] Approximately 25% will experience temporomandibular joint (TMJ) discomfort in the initial treatment period; fortunately, these were not associated with any permanent consequences.[169] Oral appliances are custom-manufactured, usually by dentists, and adjusted over a several month period. They are generally not covered by Medicare, and costs can range from $1,000 to $2,500.

Additional treatment options for sleep apnea include Oral Pressure therapy, which creates a negative pressure in the mouth to move the tongue forward[170] and upper-airway surgery.[114] A limited amount of data is available on Oral Pressure therapy, and further research is necessary to examine long-term outcomes. Upper airway surgery, such as an Uvulopalatopharyngoplasty (UPPP), involves removal of the tonsils, adenoids, posterior

tongue and soft palate. One retrospective review found no evidence of increased risk with advancing age.[171] However, dysphagia remains a concern with up to 29% of patients having dysphagia at one-year post-op.[172] Supplemental oxygen via nasal cannula (not using positive airway pressure) has also been attempted. Since sleep apnea often has an obstructive component, delivering supplemental oxygen alone may not be able to overcome the upper airway obstruction without the presence of positive airway pressure. Research comparing supplemental oxygen to PAP suggests that the former does not reduce the AHI significantly but that supplemental oxygen resulted in similar average nocturnal oxygen levels to PAP.[173] Adherence rates were similar to PAP.[173] In addition, it may be particularly effective for patients with predominantly central sleep apnea and congestive heart failure where research has shown improvements in exercise capacity and cardiac function.[174] There are at present no pharmacologic options for the treatment of sleep apnea.

7 Other sleep disorders

In addition to insomnia and sleep apnea, other sleep disorders that may be encountered in clinical practice include Restless Legs Syndrome (RLS), Periodic Limb Movement disorder (PLMD), REM behavior disorder (RBD), and the hypersomnias. A detailed discussion of these conditions and others from a geriatric medicine perspective was recently conducted by Bloom and colleagues;[60] they will be briefly reviewed here.

Restless Legs Syndrome is characterized by significant discomfort in the legs or arms prior to sleep that progressively worsens over the course of the evening and is relieved by movement. It is distinct from arthritis pain or fibromyalgia in that pain is usually not present in RLS. It is present in approximately 10% of older adults and is twice as common in women as men.[175, 176] Furthermore, it is frequently underdiagnosed, with one study noting that only 9 of 103 patients who met criteria for RLS had been diagnosed.[175] Low ferritin levels and end-stage renal disease may exacerbate RLS.[175] RLS may be even more common in dementia, where up to 24% of community-dwelling patients with dementia were noted to have RLS in one study.[177] Of particular interest, the presence of RLS was associated with higher rates of nocturnal agitation in these community-dwelling older adults with dementia.[177] The diagnosis of RLS is based on clinical history. Treatment involves the use of dopaminergic agents such as levodopa.

Patients with Periodic Limb Movement Disorder experience frequent (at least five per hour) sleep-related extremity movements that are often associated with increased brain activity (arousals). Many patients are not aware of the limb movements, but their bed partner may endorse them. Patients may instead complain of frequent nocturnal awakenings and daytime sleepiness. The prevalence of PLMD is 4–11% in older adults, and is more common in dialysis patients.[176] PLMD often accompanies RLS. The diagnosis of PLMD requires a polysomnography with EEG and EMG signal acquisition (usually an in-lab PSG). Treatment frequently involves dopaminergic agents or sedative-hypnotics.

REM behavior disorder is a condition in which patients act out their dreams due to a loss of physiologic atonia during sleep. While rare, it can be associated with significant injuries because patients are not aware of their surroundings. In addition, there is a growing body of

evidence to suggest that many patients with REM behavior disorder will subsequently develop Parkinson's Disease or other neurologic disorders.[178] Diagnosis requires an in-lab polysomnography with EEG and EMG to confirm the presence of increased muscle tone during REM sleep. Treatment centers around patient education and safety, with pharmacotherapy consisting of clonazepam or melatonin.[179]

Insufficient sleep syndrome refers to habitual patient behaviors that result in self-induced sleep deprivation. This may either be work/social-related, or due to frequent use of electronics or TV watching at night. A sleep diary is often useful to elucidate this history. Effective treatment can be difficult as patients are often resistant to changing their lifestyle. It is imperative in these cases to avoid using stimulant therapy.

Other rare sleep disorders in older adults include narcolepsy and primary hypersomnia. Narcolepsy is characterized by the presence of significant hypersomnia as well as nocturnal sleep fragmentation. It may or may not be associated with sleep attacks (cataplexy). Generally, it is diagnosed in younger adults, but delayed diagnosis with initial identification in older adults can occur. Accurate diagnosis requires a Multiple Sleep Latency Test (MSLT) which will show rapid entry into REM sleep (sleep-onset REM). If the patient demonstrates rapid sleep onset without sleep-onset REM episodes, and they do not have any other underlying sleep disorder or medication etiology, then a diagnosis of primary hypersomnia may be appropriate. Treatment of either condition relies on the use of stimulant therapy with dopamine modulators, such as modafinil, or methamphetamines.

8 Conclusion

Significant changes occur in sleep with advancing age. There is a reduction in total sleep time from an average of 8.9 hours per day in young adults to 7.4 hours per day in older adults.[9] In addition, there is an increase in nocturnal awake time, with older adults spending 30–60 minutes awake after sleep onset.[8] Despite these objective changes and a 20–30% prevalence of insomnia complaints, the actual prevalence of clinically significant insomnia disorder is approximately 5%;[12] older adults may be less likely than younger adults to meet ICD-10 diagnostic criteria for insomnia disorder because it requires an associated daytime impairment.[11] The evaluation of insomnia relies on self-report history and a sleep diary. Treatment options include the following: sleep hygiene, which while helpful, has not been found to be effective when used as monotherapy;[180] cognitive-behavioral therapy for insomnia, which requires multiple sessions but can have sustained benefit; and pharmacotherapy with sedative-hypnotics, melatonin agonists, or anti-depressants. Patients with chronic insomnia that is resistant to treatment may benefit from a polysomnography to screen for other sleep disorders.[72] If left untreated, insomnia can increase the risk of depression.

Sleep apnea is characterized by recurrent cessation or reduction in breathing, with 5–15 events/hr. considered as mild, 15–30 as moderate and >=30 as severe. It may lead to hypoxia or increased nocturnal sleep arousal and fragmentation. It is present in approximately 20% of older adults, a high prevalence that is double that of young adults.[115] It can increase the risk of cardiovascular disease and may nearly double the risk of cognitive impairment over a

five year period.[125] Diagnosis requires a polysomnography, however, Medicare guidelines now allow for coverage of portable, at-home polysomnography in most cases. Effective diagnosis requires a high clinical suspicion because many older adults lack a bed partner to report classic symptoms such as snoring or witnessed apneas. Furthermore, an increased BMI is not as prominent a risk factor in older adults as in younger ones.[115] Treatment with positive airway pressure (PAP) can be highly effective if patients are adherent. Other treatment options include an oral appliance, weight loss or surgery in select patients, and oral pressure therapy. Treatment with PAP has been shown to improve survival in older adults.[154]

Finally in addition to insomnia and sleep apnea geriatricians need to be aware of the possibility of the presence of other sleep disorders in their patients, such as; Restless Legs Syndrome, Periodic Limb Movement disorder, REM behavior disorder, and the hypersomnias for all of which efficacious treatments are available.

References

1. Herzog N, Jauch-Chara K, Hyzy F, et al. Selective slow wave sleep but not rapid eye movement sleep suppression impairs morning glucose tolerance in healthy men. Psychoneuroendocrinology. 2013 Oct; 38(10):2075–2082. [PubMed: 23602132]
2. Boselli M, Parrino L, Smerieri A, Terzano MG. Effect of age on EEG arousals in normal sleep. Sleep. 1998 Jun 15; 21(4):351–357. [PubMed: 9646379]
3. Ekirch AR. Sleep we have lost: pre-industrial slumber in the British Isles. Am Hist Rev. 2001; 106(2):343–386. [PubMed: 18680884]
4. Wehr TA. In short photoperiods, human sleep is biphasic. J Sleep Res. 1992 Jun; 1(2):103–107. [PubMed: 10607034]
5. Elder GJ, Wetherell MA, Barclay NL, Ellis JG. The cortisol awakening response - Applications and implications for sleep medicine. Sleep Med Rev. 2013 Jul 5.
6. Gooneratne NS, Edwards AY, Zhou C, Cuellar N, Grandner MA, Barrett JS. Melatonin pharmacokinetics following two different oral surge-sustained release doses in older adults. J Pineal Res. 2012 May; 52(4):437–445. [PubMed: 22348451]
7. Kalsbeek A, Fliers E. Daily regulation of hormone profiles. Handb Exp Pharmacol. 2013; (217): 185–226. [PubMed: 23604480]
8. Ohayon MM, Carskadon MA, Guilleminault C, Vitiello MV. Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: developing normative sleep values across the human lifespan. Sleep. 2004 Nov 1; 27(7):1255–1273. [PubMed: 15586779]
9. Klerman EB, Dijk DJ. Age-related reduction in the maximal capacity for sleep--implications for insomnia. Curr Biol. 2008 Aug 5; 18(15):1118–1123. [PubMed: 18656358]
10. Grandner MA, Martin JL, Patel NP, et al. Age and sleep disturbances among American men and women: data from the U.S. Behavioral Risk Factor Surveillance System. Sleep. 2012 Mar 1; 35(3): 395–406. [PubMed: 22379246]
11. Roth T, Coulouvrat C, Hajak G, et al. Prevalence and Perceived Health Associated with Insomnia Based on DSM-IV-TR; International Statistical Classification of Diseases and Related Health Problems, Tenth Revision; and Research Diagnostic Criteria/International Classification of Sleep Disorders, Second Edition Criteria: Results from the America Insomnia Survey. Biol Psychiatry. 2010 Dec 31.
12. Ohayon MM. Epidemiology of insomnia: what we know and what we still need to learn. Sleep Med Rev. 2002 Apr; 6(2):97–111. [PubMed: 12531146]
13. Malakouti SK, Foroughan M, Nojomi M, Ghalebandi MF, Zandi T. Sleep patterns, sleep disturbances and sleepiness in retired Iranian elders. Int J Geriatr Psychiatry. 2009 Nov; 24(11): 1201–1208. [PubMed: 19343736]

14. Brouwer WB, van Exel NJ, Stolk EA. Acceptability of less than perfect health states. Soc Sci Med. 2005 Jan; 60(2):237–246. [PubMed: 15522481]
15. Vitiello MV, Larsen LH, Moe KE. Age-related sleep change: Gender and estrogen effects on the subjective-objective sleep quality relationships of healthy, noncomplaining older men and women. J Psychosom Res. 2004 May; 56(5):503–510. [PubMed: 15172206]
16. Gooneratne NS, Bellamy SL, Pack F, et al. Case-control study of subjective and objective differences in sleep patterns in older adults with insomnia symptoms. J Sleep Res. 2011 Sep; 20(3):434–444. [PubMed: 20887395]
17. Leger D, Poursain B. An international survey of insomnia: under-recognition and under-treatment of a ploysymptomatic condition. Curr Med Res Opin. 2005 Nov; 21(11):1785–1792. [PubMed: 16307699]
18. Jean-Louis G, Magai C, Casimir GJ, et al. Insomnia symptoms in a multiethnic sample of American women. J Womens Health (Larchmt). 2008 Jan-Feb;17(1):15–25. [PubMed: 18240978]
19. Stenuit P, Kerkhofs M. Age modulates the effects of sleep restriction in women. Sleep. 2005 Oct 1; 28(10):1283–1288. [PubMed: 16295213]
20. Filtness AJ, Reyner LA, Horne JA. Driver sleepiness-comparisons between young and older men during a monotonous afternoon simulated drive. Biol Psychol. 2012 Mar; 89(3):580–583. [PubMed: 22266164]
21. American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 5th edition: DSM-V. 5th ed.. Washington DC: American Psychiatric Association; 2013.
22. World Health Organization. The ICD-10 Classification of Mental and Behavioural Disorders: Diagnostic Criteria for Research. World Health Organization; 1993.
23. Reynolds CF 3rd, Redline S. Workgroup D-VS-WD, Advisors. The DSM-V sleep-wake disorders nosology: an update and an invitation to the sleep community. Sleep. 2010 Jan; 33(1):10–11. [PubMed: 20120613]
24. Buysse DJ, Reynolds CF 3rd, Hauri PJ, et al. Diagnostic concordance for DSM-IV sleep disorders: a report from the APA/NIMH DSM-IV field trial. Am J Psychiatry. 1994 Sep; 151(9):1351–1360. [PubMed: 8067492]
25. Foley DJ, Monjan A, Simonsick EM, Wallace RB, Blazer DG. Incidence and remission of insomnia among elderly adults: an epidemiologic study of 6,800 persons over three years. Sleep. 1999; 22(Suppl 2):S366–S372. [PubMed: 10394609]
26. Gureje O, Oladeji BD, Abiona T, Makanjuola V, Esan O. The natural history of insomnia in the Ibadan study of ageing. Sleep. 2011 Jul; 34(7):965–973. [PubMed: 21731147]
27. Ellis JG, Perlis ML, Neale LF, Espie CA, Bastien CH. The natural history of insomnia: focus on prevalence and incidence of acute insomnia. J Psychiatr Res. 2012 Oct; 46(10):1278–1285. [PubMed: 22800714]
28. Foley DJ, Monjan AA, Izmirlian G, Hays JC, Blazer DG. Incidence and remission of insomnia among elderly adults in a biracial cohort. Sleep. 1999; 22(Suppl 2):S373–S378. [PubMed: 10394610]
29. Morin CM, Belanger L, LeBlanc M, et al. The natural history of insomnia: a population-based 3-year longitudinal study. Arch Intern Med. 2009 Mar 9; 169(5):447–453. [PubMed: 19273774]
30. Spielman AJ, Caruso LS, Glovinsky PB. A behavioral perspective on insomnia treatment. Psychiatr Clin North Am. 1987 Dec; 10(4):541–553. [PubMed: 3332317]
31. Patel NP, Grandner MA, Xie D, Branas CC, Gooneratne N. "Sleep disparity" in the population: poor sleep quality is strongly associated with poverty and ethnicity. BMC Public Health. 2010; 10:475. [PubMed: 20701789]
32. Morgan K. Daytime activity and risk factors for late-life insomnia. J Sleep Res. 2003 Sep; 12(3): 231–238. [PubMed: 12941062]
33. Evans DS, Parimi N, Nievergelt CM, et al. Common genetic variants in ARNTL and NPAS2 and at chromosome 12p13 are associated with objectively measured sleep traits in the elderly. Sleep. 2013 Mar; 36(3):431–446. [PubMed: 23449886]
34. Katz DA, McHorney CA. Clinical correlates of insomnia in patients with chronic illness. Arch Intern Med. 1998; 158(10):1099–1107. [PubMed: 9605781]

35. Brenes GA, Miller ME, Stanley MA, Williamson JD, Knudson M, McCall WV. Insomnia in older adults with generalized anxiety disorder. Am J Geriatr Psychiatry. 2009 Jun; 17(6):465–472. [PubMed: 19472436]
36. Dautovich ND, McCrae CS, Rowe M. Subjective and objective napping and sleep in older adults: are evening naps"bad" for nighttime sleep? J Am Geriatr Soc. 2008 Sep; 56(9):1681–1686. [PubMed: 18691289]
37. Batejat DM, Lagarde DP. Naps and modafinil as countermeasures for the effects of sleep deprivation on cognitive performance. Aviat Space Environ Med. 1999 May; 70(5):493–498. [PubMed: 10332946]
38. Jaussent I, Bouyer J, Ancelin ML, et al. Insomnia and daytime sleepiness are risk factors for depressive symptoms in the elderly. Sleep. 2011 Aug; 34(8):1103–1110. [PubMed: 21804672]
39. Perlis ML, Smith LJ, Lyness JM, et al. Insomnia as a risk factor for onset of depression in the elderly. Behav Sleep Med. 2006; 4(2):104–113. [PubMed: 16579719]
40. Cole MG, Dendukuri N. Risk factors for depression among elderly community subjects: a systematic review and meta-analysis. Am J Psychiatry. 2003 Jun; 160(6):1147–1156. [PubMed: 12777274]
41. Pigeon WR, Hegel M, Unutzer J, et al. Is insomnia a perpetuating factor for late-life depression in the IMPACT cohort? Sleep. 2008 Apr; 31(4):481–488. [PubMed: 18457235]
42. Lee E, Cho HJ, Olmstead R, Levin MJ, Oxman MN, Irwin MR. Persistent sleep disturbance: a risk factor for recurrent depression in community-dwelling older adults. Sleep. 2013; 36(11):1685–1691. [PubMed: 24179302]
43. Suh S, Kim H, Yang HC, Cho ER, Lee SK, Shin C. Longitudinal course of depression scores with and without insomnia in non-depressed individuals: a 6-year follow-up longitudinal study in a Korean cohort. Sleep. 2013 Mar; 36(3):369–376. [PubMed: 23449814]
44. Bjorngaard JH, Bjerkeset O, Romundstad P, Gunnell D. Sleeping problems and suicide in 75,000 Norwegian adults: a 20 year follow-up of the HUNT I study. Sleep. 2011 Sep; 34(9):1155–1159. [PubMed: 21886352]
45. Jansson-Frojmark M, Lindblom K. A bidirectional relationship between anxiety and depression, and insomnia? A prospective study in the general population. J Psychosom Res. 2008 Apr; 64(4):443–449. [PubMed: 18374745]
46. Sadler P, McLaren S, Jenkins M. A psychological pathway from insomnia to depression among older adults. Int Psychogeriatr. 2013 Aug; 25(8):1375–1383. [PubMed: 23664086]
47. Schwartz S, McDowell Anderson W, Cole SR, Cornoni-Huntley J, Hays JC, Blazer D. Insomnia and heart disease: a review of epidemiologic studies. J Psychosom Res. 1999; 47(4):313–333. [PubMed: 10616226]
48. Sigurdardottir LG, Valdimarsdottir UA, Mucci LA, et al. Sleep disruption among older men and risk of prostate cancer. Cancer Epidemiol Biomarkers Prev. 2013 May; 22(5):872–879. [PubMed: 23652374]
49. Lovato N, Lack L, Wright H, Cant M, Humphreys J. Working memory performance of older adults with insomnia. J Sleep Res. 2013 Jun; 22(3):251–257. [PubMed: 23171327]
50. Sivertsen B, Hysing M, Wehling E, et al. Neuropsychological performance in older insomniacs. Neuropsychol Dev Cogn B Aging Neuropsychol Cogn. 2013; 20(1):34–48. [PubMed: 22490041]
51. Cricco M, Simonsick EM, Foley DJ. The impact of insomnia on cognitive functioning in older adults. J Am Geriatr Soc. 2001 Sep; 49(9):1185–1189. [PubMed: 11559377]
52. Tworoger SS, Lee S, Schernhammer ES, Grodstein F. The association of self-reported sleep duration, difficulty sleeping, and snoring with cognitive function in older women. Alzheimer Dis Assoc Disord. 2006 Jan-Mar;20(1):41–48. [PubMed: 16493235]
53. Lim AS, Kowgier M, Yu L, Buchman AS, Bennett DA. Sleep Fragmentation and the Risk of Incident Alzheimer's Disease and Cognitive Decline in Older Persons. Sleep. 2013; 36(7):1027–1032. [PubMed: 23814339]
54. Xiang YT, Weng YZ, Leung CM, Tang WK, Lai KY, Ungvari GS. Prevalence and correlates of insomnia and its impact on quality of life in Chinese schizophrenia patients. Sleep. 2009 Jan; 32(1):105–109. [PubMed: 19189785]

55. Hidalgo JL, Gras CB, Garcia YD, Lapeira JT, del Campo del Campo JM, Verdejo MA. Functional status in the elderly with insomnia. Qual Life Res. 2007 Mar; 16(2):279–286. [PubMed: 17033891]
56. Lambiase MJ, Pettee Gabriel K, Kuller LH, Matthews KA. Temporal relationships between physical activity and sleep in older women. Med Sci Sports Exerc. 2013 Jun 3.
57. Phillips B, Mannino DM. Does insomnia kill? Sleep. 2005 Aug 1; 28(8):965–971. [PubMed: 16218079]
58. Ohayon MM. Insomnia: a dangerous condition but not a killer? Sleep. 2005 Sep 1; 28(9):1043–1044. [PubMed: 16268370]
59. Mallon L, Broman JE, Hetta J. Relationship between insomnia, depression, and mortality: a 12-year follow-up of older adults in the community. Int Psychogeriatr. 2000 Sep; 12(3):295–306. [PubMed: 11081951]
60. Bloom HG, Ahmed I, Alessi CA, et al. Evidence-based recommendations for the assessment and management of sleep disorders in older persons. J Am Geriatr Soc. 2009 May; 57(5):761–789. [PubMed: 19484833]
61. Al-Jawder SE, Bahammam AS. Comorbid insomnia in sleep-related breathing disorders: an under-recognized association. Sleep Breath. 2011 Mar 29. epub (ahead of print).
62. Gooneratne NS, Gehrman PR, Nkwuo JE, et al. Consequences of comorbid insomnia symptoms and sleep-related breathing disorder in elderly subjects. Arch Intern Med. 2006 Sep 18; 166(16): 1732–1738. [PubMed: 16983051]
63. Youngstedt SD, Kripke DF, Elliott JA, Klauber MR. Circadian abnormalities in older adults. J Pineal Res. 2001 Oct; 31(3):264–272. [PubMed: 11589762]
64. Morgenthaler TI, Lee-Chiong T, Alessi C, et al. Practice parameters for the clinical evaluation and treatment of circadian rhythm sleep disorders. An American Academy of Sleep Medicine report. Sleep. 2007 Nov 1; 30(11):1445–1459. [PubMed: 18041479]
65. McCall WV. Setting quantitative thresholds for detecting insomnia in older persons. J Clin Sleep Med. 2013 Feb 1; 9(2):133–134. [PubMed: 23372465]
66. Levenson JC, Troxel WM, Begley A, et al. A quantitative approach to distinguishing older adults with insomnia from good sleeper controls. J Clin Sleep Med. 2013 Feb 1; 9(2):125–131. [PubMed: 23372464]
67. Carney CE, Buysse DJ, Ancoli-Israel S, et al. The consensus sleep diary: standardizing prospective sleep self-monitoring. Sleep. 2012 Feb; 35(2):287–302. [PubMed: 22294820]
68. Buysse DJ, Ancoli-Israel S, Edinger JD, Lichstein KL, Morin CM. Recommendations for a standard research assessment of insomnia. Sleep. 2006 Sep 1; 29(9):1155–1173. [PubMed: 17040003]
69. Morgenthaler T, Alessi C, Friedman L, et al. Practice parameters for the use of actigraphy in the assessment of sleep and sleep disorders: an update for 2007. Sleep. 2007 Apr 1; 30(4):519–529. [PubMed: 17520797]
70. van Dijk E, Hilgenkamp TI, Evenhuis HM, Echteld MA. Exploring the use of actigraphy to investigate sleep problems in older people with intellectual disability. J Intellect Disabil Res. 2012 Feb; 56(2):204–211. [PubMed: 21801264]
71. Van Den Berg JF, Van Rooij FJ, Vos H, et al. Disagreement between subjective and actigraphic measures of sleep duration in a population-based study of elderly persons. J Sleep Res. 2008 Sep; 17(3):295–302. [PubMed: 18321246]
72. Littner M, Hirshkowitz M, Kramer M, et al. Practice parameters for using polysomnography to evaluate insomnia: an update. Sleep. 2003 Sep; 26(6):754–760. [PubMed: 14572131]
73. Littner MR, Kushida C, Wise M, et al. Practice parameters for clinical use of the multiple sleep latency test and the maintenance of wakefulness test. Sleep. 2005 Jan 1; 28(1):113–121. [PubMed: 15700727]
74. Riemann D, Perlis ML. The treatments of chronic insomnia: a review of benzodiazepine receptor agonists and psychological and behavioral therapies. Sleep Med Rev. 2009 Jun; 13(3):205–214. [PubMed: 19201632]

75. National Institutes of Health State of the Science Conference statement on Manifestations and Management of Chronic Insomnia in Adults, June 13–15, 2005. Sleep. 2005 Sep 1; 28(9):1049–1057. [PubMed: 16268373]
76. Shub D, Darvishi R, Kunik ME. Non-pharmacologic treatment of insomnia in persons with dementia. Geriatrics. 2009 Feb; 64(2):22–26. [PubMed: 19256583]
77. McCurry SM, Logsdon RG, Teri L, Vitiello MV. Evidence-based psychological treatments for insomnia in older adults. Psychol Aging. 2007 Mar; 22(1):18–27. [PubMed: 17385979]
78. Montgomery P, Dennis J. A systematic review of non-pharmacological therapies for sleep problems in later life. Sleep Med Rev. 2004 Feb; 8(1):47–62. [PubMed: 15062210]
79. Morin CM, Colecchi C, Stone J, Sood R, Brink D. Behavioral and pharmacological therapies for late-life insomnia: a randomized controlled trial. Jama. 1999; 281(11):991–999. [PubMed: 10086433]
80. Alessi C, Vitiello MV. Insomnia (primary) in older people. Clin Evid (Online). 2011:2011.
81. Buysse DJ, Germain A, Moul DE, et al. Efficacy of brief behavioral treatment for chronic insomnia in older adults. Arch Intern Med. 2011 May 23; 171(10):887–895. [PubMed: 21263078]
82. Cheng SK, Dizon J. Computerised cognitive behavioural therapy for insomnia: a systematic review and meta-analysis. Psychother Psychosom. 2012; 81(4):206–216. [PubMed: 22585048]
83. Morin CM, Bastien C, Guay B, Radouco-Thomas M, Leblanc J, Vallieres A. Randomized clinical trial of supervised tapering and cognitive behavior therapy to facilitate benzodiazepine discontinuation in older adults with chronic insomnia. Am J Psychiatry. 2004 Feb; 161(2):332–342. [PubMed: 14754783]
84. Jungquist CR, O'Brien C, Matteson-Rusby S, et al. The efficacy of cognitive-behavioral therapy for insomnia in patients with chronic pain. Sleep Med. 2010 Mar; 11(3):302–309. [PubMed: 20133188]
85. Sanchez-Ortuno MM, Edinger JD. Cognitive-behavioral therapy for the management of insomnia comorbid with mental disorders. Curr Psychiatry Rep. 2012 Oct; 14(5):519–528. [PubMed: 22865156]
86. Espie CA, Fleming L, Cassidy J, et al. Randomized controlled clinical effectiveness trial of cognitive behavior therapy compared with treatment as usual for persistent insomnia in patients with cancer. J Clin Oncol. 2008 Oct 1; 26(28):4651–4658. [PubMed: 18591549]
87. Jaussent I, Ancelin ML, Berr C, et al. Hypnotics and mortality in an elderly general population: a 12-year prospective study. BMC Med. 2013 Sep 26.11(1):212. [PubMed: 24070457]
88. Perlis ML, McCall WV, Krystal AD, Walsh JK. Long-term, non-nightly administration of zolpidem in the treatment of patients with primary insomnia. J Clin Psychiatry. 2004 Aug; 65(8):1128–1137. [PubMed: 15323600]
89. Roth T, Seiden D, Sainati S, Wang-Weigand S, Zhang J, Zee P. Effects of ramelteon on patient-reported sleep latency in older adults with chronic insomnia. Sleep Med. 2006 Jun; 7(4):312–318. [PubMed: 16709464]
90. Zammit G, Wang-Weigand S, Rosenthal M, Peng X. Effect of ramelteon on middle-of-the-night balance in older adults with chronic insomnia. J Clin Sleep Med. 2009 Feb 15; 5(1):34–40. [PubMed: 19317379]
91. Zammit G, Erman M, Wang-Weigand S, Sainati S, Zhang J, Roth T. Evaluation of the efficacy and safety of ramelteon in subjects with chronic insomnia. J Clin Sleep Med. 2007 Aug 15; 3(5):495–504. [PubMed: 17803013]
92. Mets MA, de Vries JM, de Senerpont Domis LM, Volkerts ER, Olivier B, Verster JC. Next-day effects of ramelteon (8 mg), zopiclone (7.5 mg), and placebo on highway driving performance, memory functioning, psychomotor performance, and mood in healthy adult subjects. Sleep. 2011 Oct; 34(10):1327–1334. [PubMed: 21966064]
93. Wiegand MH. Antidepressants for the treatment of insomnia : a suitable approach? Drugs. 2008; 68(17):2411–2417. [PubMed: 19016570]
94. Walsh JK. Drugs used to treat insomnia in 2002: regulatory-based rather than evidence-based medicine. Sleep. 2004 Dec 15; 27(8):1441–1442. [PubMed: 15683131]
95. Wilson S, Nutt D. Management of insomnia: treatments and mechanisms. Br J Psychiatry. 2007 Sep.191:195–197. [PubMed: 17766757]

96. Winokur A, DeMartinis NA 3rd, McNally DP, Gary EM, Cormier JL, Gary KA. Comparative effects of mirtazapine and fluoxetine on sleep physiology measures in patients with major depression and insomnia. J Clin Psychiatry. 2003 Oct; 64(10):1224–1229. [PubMed: 14658972]

97. Vande Griend JP, Anderson SL. Histamine-1 receptor antagonism for treatment of insomnia. J Am Pharm Assoc (2003). 2012; 52(6):e210–e219. [PubMed: 23229983]

98. Buysse DJ. Insomnia, depression and aging. Assessing sleep and mood interactions in older adults. Geriatrics. 2004 Feb; 59(2):47–51. quiz 52. [PubMed: 14989593]

99. Tranah GJ, Parimi N, Blackwell T, et al. Postmenopausal hormones and sleep quality in the elderly: a population based study. BMC Womens Health. 2010; 10:15. [PubMed: 20441593]

100. Sarris J, Byrne GJ. A systematic review of insomnia and complementary medicine. Sleep Med Rev. 2010 Jun 5.

101. Wade AG, Ford I, Crawford G, et al. Nightly treatment of primary insomnia with prolonged release melatonin for 6 months: a randomized placebo controlled trial on age and endogenous melatonin as predictors of efficacy and safety. BMC Med. 2010; 8:51. [PubMed: 20712869]

102. Morin CM, Bastien CH, Brink D, Brown TR. Adverse effects of temazepam in older adults with chronic insomnia. Hum Psychopharmacol. 2003 Jan; 18(1):75–82. [PubMed: 12532318]

103. Morin CM, Vallieres A, Guay B, et al. Cognitive behavioral therapy, singly and combined with medication, for persistent insomnia: a randomized controlled trial. JAMA. 2009 May 20; 301(19):2005–2015. [PubMed: 19454639]

104. Schnelle JF, Ouslander JG, Simmons SF, Alessi CA, Gravel MD. The nighttime environment, incontinence care, and sleep disruption in nursing homes. J Am Geriatr Soc. 1993; 41(9):910–914. [PubMed: 8409176]

105. Schnelle JF, Alessi CA, Al-Samarrai NR, Fricker RD Jr, Ouslander JG. The nursing home at night: effects of an intervention on noise, light, and sleep. J Am Geriatr Soc. 1999; 47(4):430–438. [PubMed: 10203118]

106. Mishima K, Okawa M, Hozumi S, Hishikawa Y. Supplementary administration of artificial bright light and melatonin as potent treatment for disorganized circadian rest-activity and dysfunctional autonomic and neuroendocrine systems in institutionalized demented elderly persons. Chronobiol Int. 2000 May; 17(3):419–432. [PubMed: 10841214]

107. El Kady HM, Ibrahim HK, Mohamed SG. Cognitive behavioral therapy for institutionalized elders complaining of sleep disturbance in Alexandria, Egypt. Sleep Breath. 2012 Dec; 16(4): 1173–1180. [PubMed: 22160931]

108. McCurry SM, Gibbons LE, Logsdon RG, Vitiello MV, Teri L. Nighttime insomnia treatment and education for Alzheimer's disease: a randomized, controlled trial. J Am Geriatr Soc. 2005 May; 53(5):793–802. [PubMed: 15877554]

109. Paniagua MA, Paniagua EW. The demented elder with insomnia. Clin Geriatr Med. 2008 Feb; 24(1):69–81. vii. [PubMed: 18035232]

110. McCurry SM, Gibbons LE, Logsdon RG, Vitiello MV, Teri L. Insomnia In Caregivers Of Persons With Dementia: Who Is At Risk And What Can Be Done About It? Sleep Med Clin. 2009 Dec 1; 4(4):519–526. [PubMed: 20046806]

111. Pollak C, Perlick D. Sleep problems and institutionalization of the elderly. J Geriatric Psych & Neurology. 1991 Oct-Dec;4(4):204–210.

112. McCurry SM, Logsdon RG, Vitiello MV, Teri L. Successful behavioral treatment for reported sleep problems in elderly caregivers of dementia patients: a controlled study. J Gerontol B Psychol Sci Soc Sci. 1998; 53(2):122–129.

113. King AC, Baumann K, O'Sullivan P, Wilcox S, Castro C. Effects of moderate-intensity exercise on physiological, behavioral, and emotional responses to family caregiving: a randomized controlled trial. J Gerontol A Biol Sci Med Sci. 2002 Jan; 57(1):M26–M36. [PubMed: 11773209]

114. Epstein LJ, Kristo D, Strollo PJ Jr, et al. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. J Clin Sleep Med. 2009 Jun 15; 5(3):263–276. [PubMed: 19960649]

115. Young T, Shahar E, Nieto FJ, et al. Predictors of sleep apnea in community-dwelling adults: the Sleep Heart Health Study. Arch Intern Med. 2002 Apr 22; 162(8):893–900. [PubMed: 11966340]

116. Herrscher TE, Akre H, Overland B, Sandvik L, Westheim AS. High prevalence of sleep apnea in heart failure outpatients: even in patients with preserved systolic function. J Card Fail. 2011 May; 17(5):420–425. [PubMed: 21549300]

117. Gehrman PR, Martin JL, Shochat T, Nolan S, Corey-Bloom J, Ancoli-Israel S. Sleep apnea and agitation in institutionalized adults with Alzheimer disease. Am J Geriatr Psychiatry. 2003 Jul-Aug;11(4):426–433. [PubMed: 12837671]

118. Mehra R, Stone KL, Blackwell T, et al. Prevalence and correlates of sleep apnea in older men: osteoporotic fractures in men sleep study. J Am Geriatr Soc. 2007 Sep; 55(9):1356–1364. [PubMed: 17767677]

119. Javaheri S, Caref EB, Chen E, Tong KB, Abraham WT. Sleep apnea testing and outcomes in a large cohort of Medicare beneficiaries with newly diagnosed heart failure. Am J Respir Crit Care Med. 2011 Feb 15; 183(4):539–546. [PubMed: 20656940]

120. Eikermann M, Jordan AS, Chamberlin NL, et al. The influence of aging on pharyngeal collapsibility during sleep. Chest. 2007 Jun; 131(6):1702–1709. [PubMed: 17413053]

121. Oliven A, Carmi N, Coleman R, Odeh M, Silbermann M. Age-related changes in upper airway muscles morphological and oxidative properties. Exp Gerontol. 2001 Nov; 36(10):1673–1686. [PubMed: 11672988]

122. Young T, Finn L, Austin D, Peterson A. Menopausal status and sleep apnea in the Wisconsin Sleep Cohort Study. Am J Respir Crit Care Med. 2003 May 1; 167(9):1181–1185. [PubMed: 12615621]

123. Bucca C, Cicolin A, Brussino L, et al. Tooth loss and obstructive sleep apnoea. Respir Res. 2006; 7:8. [PubMed: 16417639]

124. Gilman S, Chervin RD, Koeppe RA, et al. Obstructive sleep apnea is related to a thalamic cholinergic deficit in MSA. Neurology. 2003 Jul 8; 61(1):35–39. [PubMed: 12847153]

125. Yaffe K, Laffan AM, Harrison SL, et al. Sleep apnea, hypoxia, and risk of mild cognitive impairment and dementia in older women. JAMA. 2011 Aug 10; 306(6):613–619. [PubMed: 21828324]

126. Ancoli-Israel S, Palmer BW, Cooke JR, et al. Cognitive effects of treating obstructive sleep apnea in Alzheimer's disease: a randomized controlled study. J Am Geriatr Soc. 2008 Nov; 56(11): 2076–2081. [PubMed: 18795985]

127. Canessa N, Castronovo V, Cappa SF, et al. Obstructive sleep apnea: brain structural changes and neurocognitive function before and after treatment. Am J Respir Crit Care Med. 2011 May 15; 183(10):1419–1426. [PubMed: 21037021]

128. Kotterba S, Rasche K, Widdig W, et al. Neuropsychological investigations and event-related potentials in obstructive sleep apnea syndrome before and during CPAP-therapy. J Neurol Sci. 1998 Jul 15; 159(1):45–50. [PubMed: 9700702]

129. Wheaton AG, Perry GS, Chapman DP, Croft JB. Sleep disordered breathing and depression among U.S. adults: National Health and Nutrition Examination Survey, 2005–2008. Sleep. 2012 Apr; 35(4):461–467. [PubMed: 22467983]

130. Harris M, Glozier N, Ratnavadivel R, Grunstein RR. Obstructive sleep apnea and depression. Sleep Med Rev. 2009 Dec; 13(6):437–444. [PubMed: 19596599]

131. Peppard PE, Szklo-Coxe M, Hla KM, Young T. Longitudinal association of sleep-related breathing disorder and depression. Arch Intern Med. 2006 Sep 18; 166(16):1709–1715. [PubMed: 16983048]

132. Jackson ML, Stough C, Howard ME, Spong J, Downey LA, Thompson B. The contribution of fatigue and sleepiness to depression in patients attending the sleep laboratory for evaluation of obstructive sleep apnea. Sleep Breath. 2011 Sep; 15(3):439–445. [PubMed: 20446116]

133. Asghari A, Mohammadi F, Kamrava SK, Tavakoli S, Farhadi M. Severity of depression and anxiety in obstructive sleep apnea syndrome. Eur Arch Otorhinolaryngol. 2012 Dec; 269(12): 2549–2553. [PubMed: 22298252]

134. Ishman SL, Cavey RM, Mettel TL, Gourin CG. Depression, sleepiness, and disease severity in patients with obstructive sleep apnea. Laryngoscope. 2010 Nov; 120(11):2331–2335. [PubMed: 20939075]

135. Castro LS, Castro J, Hoexter MQ, et al. Depressive symptoms and sleep: A population-based polysomnographic study. Psychiatry Res. 2013 Dec 30; 210(3):906–912. [PubMed: 24041750]
136. Aloia MS, Arnedt JT, Smith L, Skrekas J, Stanchina M, Millman RP. Examining the construct of depression in obstructive sleep apnea syndrome. Sleep Med. 2005 Mar; 6(2):115–121. [PubMed: 15716215]
137. Krystal AD, Harsh JR, Yang R, Rippon GA, Lankford DA. A double-blind, placebo-controlled study of armodafinil for excessive sleepiness in patients with treated obstructive sleep apnea and comorbid depression. J Clin Psychiatry. 2010 Jan; 71(1):32–40. [PubMed: 20051221]
138. Freedland KE, Carney RM, Hayano J, Steinmeyer BC, Reese RL, Roest AM. Effect of obstructive sleep apnea on response to cognitive behavior therapy for depression after an acute myocardial infarction. J Psychosom Res. 2012 Apr; 72(4):276–281. [PubMed: 22405221]
139. Habukawa M, Uchimura N, Kakuma T, et al. Effect of CPAP treatment on residual depressive symptoms in patients with major depression and coexisting sleep apnea: Contribution of daytime sleepiness to residual depressive symptoms. Sleep Med. 2010 Jun; 11(6):552–557. [PubMed: 20488748]
140. Kjelsberg FN, Ruud EA, Stavem K. Predictors of symptoms of anxiety and depression in obstructive sleep apnea. Sleep Med. 2005 Jul; 6(4):341–346. [PubMed: 15946899]
141. Macey PM, Woo MA, Kumar R, Cross RL, Harper RM. Relationship between obstructive sleep apnea severity and sleep, depression and anxiety symptoms in newly-diagnosed patients. PLoS One. 2010; 5(4):e10211. [PubMed: 20419135]
142. Gottlieb DJ, Yenokyan G, Newman AB, et al. Prospective study of obstructive sleep apnea and incident coronary heart disease and heart failure: the sleep heart health study. Circulation. 2010 Jul 27; 122(4):352–360. [PubMed: 20625114]
143. Redline S, Yenokyan G, Gottlieb DJ, et al. Obstructive sleep apnea-hypopnea and incident stroke: the sleep heart health study. Am J Respir Crit Care Med. 2010 Jul 15; 182(2):269–277. [PubMed: 20339144]
144. Munoz R, Duran-Cantolla J, Martinez-Vila E, et al. Severe sleep apnea and risk of ischemic stroke in the elderly. Stroke. 2006 Sep; 37(9):2317–2321. [PubMed: 16888274]
145. Endeshaw YW, Unruh ML, Kutner M, Newman AB, Bliwise DL. Sleep apnea and frailty in the Cardiovascular Health Study Cohort. Am J Epidemiol. 2009 Jul 15; 170(2):193–202. [PubMed: 19465743]
146. Flink BJ, Rivelli SK, Cox EA, et al. Obstructive sleep apnea and incidence of postoperative delirium after elective knee replacement in the nondemented elderly. Anesthesiology. 2012 Apr; 116(4):788–796. [PubMed: 22337162]
147. Practice Guidelines for the Perioperative Management of Patients with Obstructive Sleep Apnea: An Updated Report by the American Society of Anesthesiologists Task Force on Perioperative Management of Patients with Obstructive Sleep Apnea. Anesthesiology. 2013 Dec 16.
148. Punjabi NM, Caffo BS, Goodwin JL, et al. Sleep apnea and mortality: a prospective cohort study. PLoS Med. 2009 Aug.6(8):e1000132. [PubMed: 19688045]
149. Ancoli-Israel S, Kripke DF, Klauber MR, et al. Morbidity, mortality and sleep apnea in community dwelling elderly. Sleep. 1996; 19(4):277–282. [PubMed: 8776783]
150. Gami AS, Howard DE, Olson EJ, Somers VK. Day-night pattern of sudden death in obstructive sleep apnea. N Engl J Med. 2005 Mar 24; 352(12):1206–1214. [PubMed: 15788497]
151. Gooneratne, N. Insomnia in the elderly. In: Forciea, MA.; Lavizzo-Mourey, R.; Schwab, EP., editors. Geriatric Secrets. 2nd ed.. Philadelphia: Hanley and Belfus; 2000. p. 44-49.
152. Welch HG, Albertsen PC, Nease RF, Bubolz TA, Wasson JH. Estimating treatment benefits for the elderly: the effect of competing risks. Ann Intern Med. 1996 Mar 15; 124(6):577–584. [PubMed: 8597322]
153. Gooneratne, NS.; Pien, G.; Gurubhagavatula, I. Sleep-related breathing disorders in the elderly. In: Kushida, CA., editor. Encyclopedia of Sleep. Vol. 3. Elsevier, Inc; 2013. p. 547-554.
154. Martinez-Garcia MA, Campos-Rodriguez F, Catalan-Serra P, et al. Cardiovascular mortality in obstructive sleep apnea in the elderly: role of long-term continuous positive airway pressure treatment: a prospective observational study. Am J Respir Crit Care Med. 2012 Nov 1; 186(9): 909–916. [PubMed: 22983957]

155. Polese JF, Santos-Silva R, de Oliveira Ferrari PM, Sartori DE, Tufik S, Bittencourt L. Is portable monitoring for diagnosing obstructive sleep apnea syndrome suitable in elderly population? Sleep Breath. 2013 May; 17(2):679–686. [PubMed: 22752758]

156. Agency for Healthcare Research and Quality. Obstructive Sleep Apnea-Hypopnea Syndrome: Modeling different diagnostic strategies. 2010. Available at: http://www.cms.hhs.gov/determinationprocess/downloads/id50TA.pdf

157. Centers for Medicare and Medicaid Services. [Accessed 2010] Decision Memo for Continuous Positive Airway Pressure (CPAP) Therapy for Obstructive Sleep Apnea (OSA) (CAG-00093R2) [HTML]. 2008 Mar. Available at: http://www.cms.hhs.gov/mcd/viewdecisionmemo.asp?id=204.

158. Bliwise DL, Benkert RE, Ingham RH. Factors associated with nightly variability in sleep apnea in the elderly. Chest. 1991; 100(4):973–976. [PubMed: 1914614]

159. American Academy of Sleep Medicine. International classification of sleep disorders, 2nd ed.: Diagnostic and coding manual. Westchester, Illinois: American Academy of Sleep Medicine; 2005.

160. Stessman J, Jacobs JM, Ein-Mor E, Bursztyn M. Normal body mass index rather than obesity predicts greater mortality in elderly people: the Jerusalem longitudinal study. J Am Geriatr Soc. 2009 Dec 1; 57(12):2232–2238. [PubMed: 19925614]

161. van Rutte PW, Smulders JF, de Zoete JP, Nienhuijs SW. Sleeve gastrectomy in older obese patients. Surg Endosc. 2013 Jun; 27(6):2014–2019. [PubMed: 23344504]

162. Busetto L, Angrisani L, Basso N, et al. Safety and efficacy of laparoscopic adjustable gastric banding in the elderly. Obesity (Silver Spring). 2008 Feb; 16(2):334–338. [PubMed: 18239641]

163. Ayalon L, Ancoli-Israel S, Stepnowsky C, et al. Adherence to continuous positive airway pressure treatment in patients with Alzheimer's disease and obstructive sleep apnea. Am J Geriatr Psychiatry. 2006 Feb; 14(2):176–180. [PubMed: 16473983]

164. Morgenthaler TI, Aurora RN, Brown T, et al. Practice parameters for the use of autotitrating continuous positive airway pressure devices for titrating pressures and treating adult patients with obstructive sleep apnea syndrome: an update for 2007. An American Academy of Sleep Medicine report. Sleep. 2008 Jan 1; 31(1):141–147. [PubMed: 18220088]

165. Sawyer AM, Gooneratne NS, Marcus CL, Ofer D, Richards KC, Weaver TE. A systematic review of CPAP adherence across age groups: Clinical and empiric insights for developing CPAP adherence interventions. Sleep Med Rev. 2011 Dec; 15(6):343–356. [PubMed: 21652236]

166. Russo-Magno P, O'Brien A, Panciera T, Rounds S. Compliance with CPAP therapy in older men with obstructive sleep apnea. J Am Geriatr Soc. 2001 Sep; 49(9):1205–1211. [PubMed: 11559380]

167. Aarab G, Lobbezoo F, Hamburger HL, Naeije M. Oral appliance therapy versus nasal continuous positive airway pressure in obstructive sleep apnea: a randomized, placebo-controlled trial. Respiration. 2011; 81(5):411–419. [PubMed: 20962502]

168. Holley AB, Lettieri CJ, Shah AA. Efficacy of an adjustable oral appliance and comparison with continuous positive airway pressure for the treatment of obstructive sleep apnea syndrome. Chest. 2011 Dec; 140(6):1511–1516. [PubMed: 21636666]

169. Doff MH, Veldhuis SK, Hoekema A, et al. Long-term oral appliance therapy in obstructive sleep apnea syndrome: a controlled study on temporomandibular side effects. Clin Oral Investig. 2012 Jun; 16(3):689–697.

170. Colrain IM, Black J, Siegel LC, et al. A multicenter evaluation of oral pressure therapy for the treatment of obstructive sleep apnea. Sleep Med. 2013 Sep; 14(9):830–837. [PubMed: 23871259]

171. Kezirian EJ, Weaver EM, Yueh B, et al. Incidence of serious complications after uvulopalatopharyngoplasty. Laryngoscope. 2004 Mar; 114(3):450–453. [PubMed: 15091217]

172. Jaghagen EL, Berggren D, Dahlqvist A, Isberg A. Prediction and risk of dysphagia after uvulopalatopharyngoplasty and uvulopalatoplasty. Acta Otolaryngol. 2004 Dec; 124(10):1197–1203. [PubMed: 15768818]

173. Loredo JS, Ancoli-Israel S, Kim EJ, Lim WJ, Dimsdale JE. Effect of continuous positive airway pressure versus supplemental oxygen on sleep quality in obstructive sleep apnea: a placebo-CPAP-controlled study. Sleep. 2006 Apr 1; 29(4):564–571. [PubMed: 16676791]

174. Toyama T, Seki R, Kasama S, et al. Effectiveness of nocturnal home oxygen therapy to improve exercise capacity, cardiac function and cardiac sympathetic nerve activity in patients with chronic heart failure and central sleep apnea. Circ J. 2009 Feb; 73(2):299–304. [PubMed: 19122308]

175. Curgunlu A, Doventas A, Karadeniz D, et al. Prevalence and characteristics of restless legs syndrome (RLS) in the elderly and the relation of serum ferritin levels with disease severity: hospital-based study from Istanbul, Turkey. Arch Gerontol Geriatr. 2012 Jul-Aug;55(1):73–76. [PubMed: 21722973]

176. Hornyak M, Trenkwalder C. Restless legs syndrome and periodic limb movement disorder in the elderly. J Psychosom Res. 2004 May; 56(5):543–548. [PubMed: 15172211]

177. Rose KM, Beck C, Tsai PF, et al. Sleep disturbances and nocturnal agitation behaviors in older adults with dementia. Sleep. 2011 Jun; 34(6):779–786. [PubMed: 21629366]

178. McCarter SJ, St Louis EK, Boeve BF. REM sleep behavior disorder and REM sleep without atonia as an early manifestation of degenerative neurological disease. Curr Neurol Neurosci Rep. 2012 Apr; 12(2):182–192. [PubMed: 22328094]

179. McCarter SJ, Boswell CL, St Louis EK, et al. Treatment outcomes in REM sleep behavior disorder. Sleep Med. 2013 Mar; 14(3):237–242. [PubMed: 23352028]

180. Morgenthaler T, Kramer M, Alessi C, et al. Practice parameters for the psychological and behavioral treatment of insomnia: an update. An american academy of sleep medicine report. Sleep. 2006 Nov 1; 29(11):1415–1419. [PubMed: 17162987]

181. National Institutes of Health Consensus Panel. NIH State-of-the-Science Conference Statement on manifestations and management of chronic insomnia in adults. NIH Consens State Sci Statements. 2005 Jun 13–15; 22(2):1–30.

182. Krystal AD, Durrence HH, Scharf M, et al. Efficacy and Safety of Doxepin 1 mg and 3 mg in a 12-week Sleep Laboratory and Outpatient Trial of Elderly Subjects with Chronic Primary Insomnia. Sleep. 2010 Nov; 33(11):1553–1561. [PubMed: 21102997]

183. Ancoli-Israel S, Kripke DF, Klauber MR, Mason WJ, Fell R, Kaplan O. Sleep apnea in community-dwelling elderly. Sleep. 1991; 14(6):486–495. [PubMed: 1798880]

Key Points

- Insomnia symptoms, while present in 20–40% of older adults, meet clinical criteria for insomnia diagnosis (i.e., significant daytime symptoms and duration criteria) in only approximately 5%.
- Sleep apnea, as defined by an apnea-hypopnea index >=15 events/hour, meeting Medicare treatment guidelines, is present in up to 20% of older adults, with a markedly increased prevalence in certain conditions, such as congestive heart failure or dementia (approximately 50–70%).
- A high index of suspicion is particularly important for the diagnosis of sleep apnea.
- Effective treatment exists for the majority of sleep disorders, such as positive airway pressure therapy for sleep apnea, and cognitive-behavioral therapy/ pharmacotherapy for insomnia.

Box 1

Recommended Insomnia and Sleep Apnea Evaluation Scales

Condition	Scale	Notes
Insomnia	Sleep diary	Consensus sleep diary available[67]
	Insomnia Severity Index	Values >=15 are consistent with “clinical insomnia”
	Pittsburgh Sleep Quality Index	A global measure of sleep quality that is not limited to insomnia symptoms
	Multidimensional Fatigue Inventory or Fatigue Severity Scale	Used to measure daytime fatigue, which is more common in insomnia patients than daytime sleepiness
Sleep Apnea	STOP-BANG	Primarily used for pre-operative screening for sleep apnea risk
	Epworth Sleepiness Scale	Measure of daytime sleepiness; older adults may not respond to all items thus under-reporting sleepiness severity

Box 2

Sleep Hygiene interventions for insomnia

Create a stable sleep pattern

1. Maintain a regular sleep/wake schedule. It is most important to keep the same rise time every day; bedtime is also important but it can be more difficult for patients to have a consistent bedtime due to day-to-day demands of work/family. Advise the patient to set their alarm to get up at the same time each morning, regardless of how much sleep they got during the night, in order to maintain a consistent sleep/wake schedule.
2. Do not attempt to "make up for lost sleep" on weekends or holidays.
3. Refrain from taking naps during the day.

Encourage a non-disruptive sleep environment

4. Keep the bedroom dark and at a temperature that is comfortable.
5. Block out noises that can disturb sleep. Sponge earplugs or "white noise" made by fans, air conditioners or a "white noise" machine.

Reduce pre-sleep tension

6. Do not watch the alarm clock and worry about the time or lost sleep.
7. Develop a sleep ritual: do the same things each evening before retiring for the night to give your body cues (signals) that it is time to settle down.
8. Plan evening activities that promote relaxation. Before going to the bedroom, make a list of things to deal with tomorrow and make a list of things to do before bedtime.

Dietary/Lifestyle Modifications

9. Maintain a healthy diet. Going to bed hungry or eating a large meal before bedtime can worsen sleep. If hungry at bedtime, eat a light snack. Eat meals at approximately the same time each day, every day.
10. Avoid or minimize the use of caffeine. It is recommended not to drink coffee, tea or soda after lunch. If the patient continues to have difficulty falling asleep, avoid drinking caffeinated beverages after breakfast.
11. Avoid alcohol. While it may temporarily lead to somnolence, for most people it causes awakenings as well as poor sleep later in the night. Alcohol can make snoring and sleep apnea worse.
12. Maintain a regular exercise schedule. Walking is an excellent form of exercise. The best time is in the late morning or mid-day (9 AM – 1 PM). For some people, "strenuous exercise" before bedtime can be too stimulating and

may prevent them from falling asleep. Light stretching can be done on rainy days.

Box 3

Components of Cognitive-Behavioral Therapy for Insomnia

Address maladaptive sleep cognitions: Insomnia patients may have exaggerated perceptions of how sleep impacts their life and how much sleep they need. These inaccurate beliefs lead to increased worry and unrealistic expectations.

Sleep Hygiene: promote regular sleep-wake patterns and minimize nocturnal disruptions

Stimulus Control Therapy: Chronic insomnia can condition negative associations between the bed and sleep such that patients find it difficult to relax in bed; stimulus control therapy seeks to re-associate the bed with sleep. Patient instructions include avoiding sleep-incompatible behaviors (do not use the bed as a place to read, watch television, or catch up on work), go to bed only when sleepy, and get out of the bed if unable to sleep and the patient is beginning to feel anxious.

Sleep Restriction Therapy: Many insomnia patients attempt to over-compensate for their insomnia by spending excessive time in bed. While seeming counter-intuitive, sleep restriction induces partial sleep deprivation which in turn increases the likelihood that an insomnia patient will actually sleep when they are in bed. The ultimate goal is to break the cycle of insomnia.

Relaxation Techniques: These include progressive muscle relaxation or guided imagery.

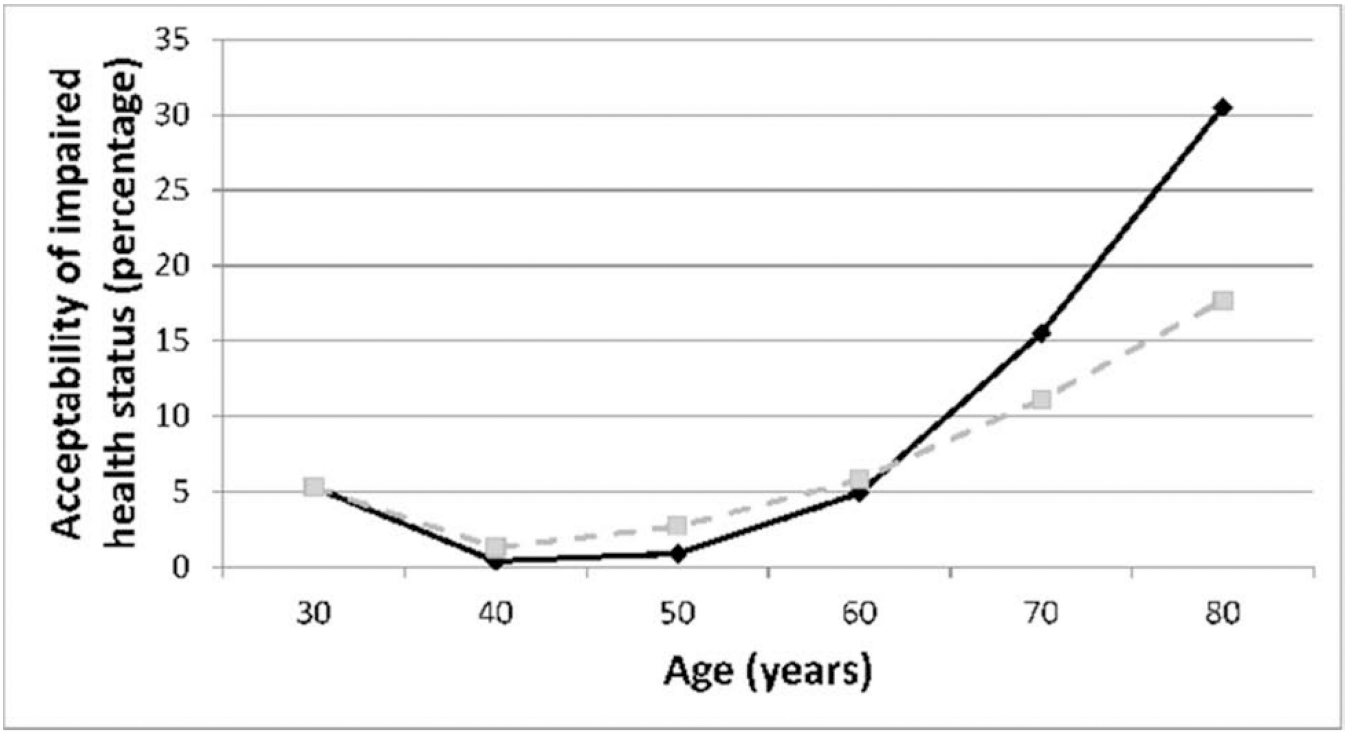

Figure 1.

Adjustment of perception of "acceptable" health with aging. Black line: Acceptability of severe impairment with usual activities. Grey line: Acceptability of extreme pain or discomfort.

Data from Brouwer WB, van Exel NJ, Stolk EA. Acceptability of less than perfect health states. *Soc Sci Med.* Jan 2005;60(2):237–246.

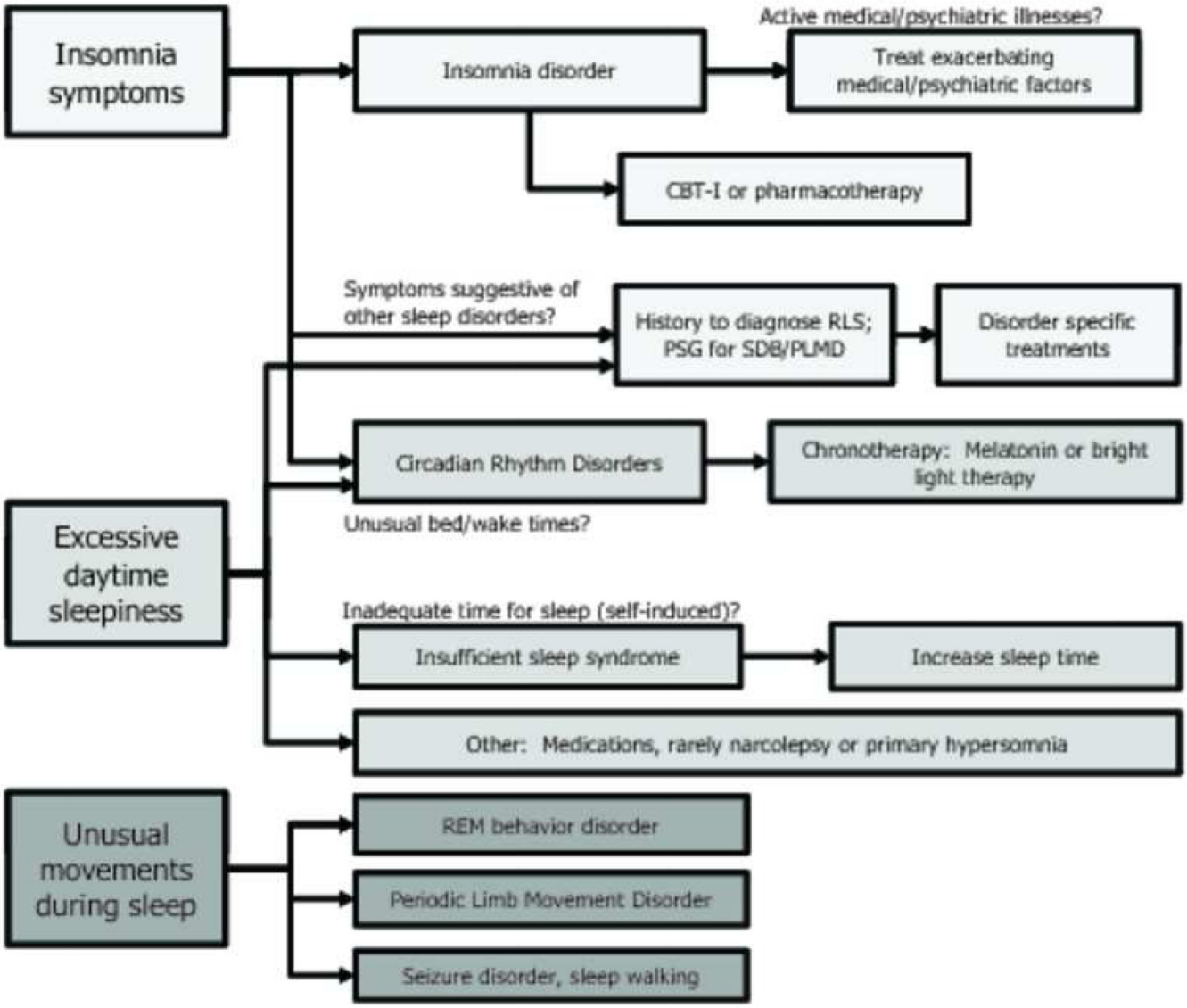

Figure 2.

Diagnostic and treatment approach for sleep disorders in older adults. Adapted from: Bloom HG, Ahmed I, Alessi CA, et al. Evidence-based recommendations for the assessment and management of sleep disorders in older persons. *J Am Geriatr Soc.* May 2009;57(5):761–789.

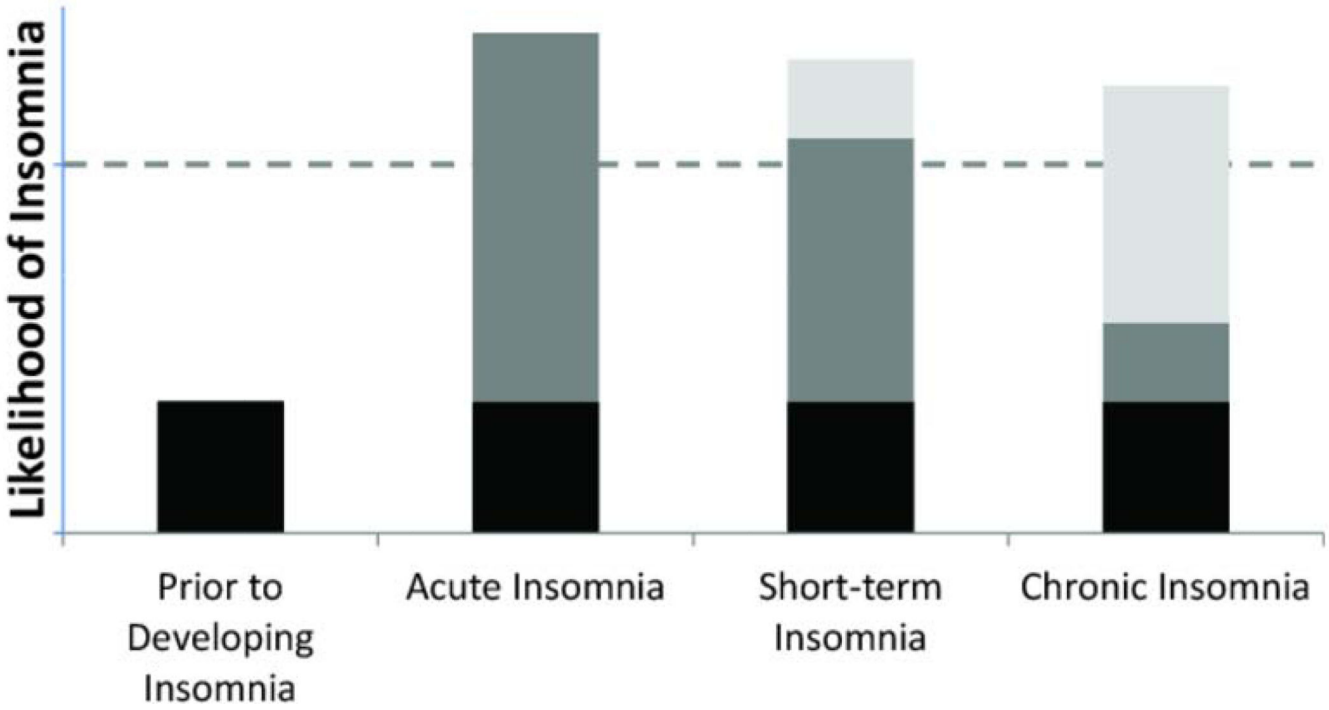

Figure 3.

This figure displays the three factor model of insomnia.[30] The dashed line indicates the threshold above which clinically significant insomnia occurs.

Data From: Spielman AJ, Caruso LS, Glovinsky PB. A behavioral perspective on insomnia treatment. *Psychiatr Clin North Am.* Dec 1987;10(4):541–553.

Table 1

Common used ICD-10 codes for insomnia

Diagnosis	ICD-10 code	Notes
Primary insomnia	F51.01	Does not occur in the context of any other sleep disorder and is not linked to any other substance or medical condition; often has a childhood or young-adult onset
Adjustment insomnia	F51.02	Transient insomnia, usually less than one month in duration
Paradoxical insomnia	F51.03	Also known as sleep-state misperception
Psychophysiological insomnia	F51.04	Chronic, conditioned insomnia
Insomnia due to other mental disorder	F51.05	Often related to anxiety or depression
Other insomnia not due to a substance or known physiological condition	F51.09	Generally refers to persistent insomnia
Insomnia, unspecified	G47.00	Suspected insomnia due to other conditions, but not definitive (NOS or not otherwise specified)
Insomnia due to medical condition	G47.01	Attempt to code the medical condition if possible
Alcohol-related insomnia	F10.182	While alcohol has an initial soporific effect, it can increase the rate of arousals as blood alcohol levels decrease, usually after about 2 hours
Drug-related insomnia	Multiple	Different ICD-10 codes depending upon drug

Table 2
Additional Tests and Evaluations for an Insomnia Work-Up

Condition	Test	Other Clinical Symptoms
Autoimmune disorders	Erythrocyte Sedimentation Rate (ESR), Anti-Nuclear Antibody (ANA), Rheumatoid Factor (RF)	Joint pain, rash, neurologic symptoms
Fibromyalgia	Presence of at least 11 tender points on exam and history of at least three months of chronic pain	Muscle/joint pain, fatigue
Hyperthyroid	Thyroid Stimulating Hormone (TSH)	Palpitations, weight loss, diaphoresis
Parkinson's Disease	Clinical exam	Tremor, gait instability
Circadian Rhythm Disorders / Insufficient Sleep Opportunity	Wrist-activity monitor (actigraphy) + Sleep Diary	Abnormal sleep times
Periodic Limb Movement Disorder/ Restless Legs Syndrome	Polysomnography	Leg or arm movement that interferes with sleep onset (RLS) or that wakes patient from sleep (PLMD)
Sleep Apnea	Polysomnography	Snoring, witnessed apneas, nocturnal choking/ gasping

Table 3
Evidence-Based Recommendations for Insomnia Evaluation and Treatment

Quality of evidence: I: At least one properly designed randomized controlled trial; II: Substantial evidence from non-randomized trials; III: Expert committee reports. Strength of evidence: A: Good evidence to support the use of a recommendation; B: Moderate evidence; C Poor evidence--clinicians may elect to not follow the recommendation. Adapted from Bloom HG, Ahmed I, Alessi CA, et al. Evidence-based recommendations for the assessment and management of sleep disorders in older persons. *J Am Geriatr Soc.* May 2009;57(5):761–789.

Recommendation	Quality of Evidence	Strength of Evidence
A Sleep Diary is an essential component of an insomnia evaluation.[68]	III	A
Polysomnography is not routinely required for insomnia evaluation.[72]	III	A
Cognitive-Behavioral Therapy for Insomnia (CBT-I) is effective in older adults and is associated with minimal side effects.[60, 80, 181]	I	A
Nonbenzodiazepines hypnotics can improve insomnia symptoms, but can be associated with side effects such as tolerance and neurocognitive effects.[60, 80]	I	A
Melatonin-receptor agonists can improve sleep onset insomnia symptoms.[60]	I	A
Concurrent CBT-I and pharmacotherapy can be efficacious.[79]	II	A
Doxepin at sub-antidepressant doses, can be efficacious.[137]	I	B
Antihistamines, anticonvulsants, and antipsychotics are not recommended for the chronic treatment of insomnia.[60]	II	B
Exercise and bright light therapy may improve sleep.[80]	II	B

Table 4

FDA approved medications for insomnia

Data obtained from FDA Prescribing Information (package inserts) and literature review.

Category	Generic (Trade) Name	Indication	Geriatric Dose (mg)	Half-Life in Older Adults (Hours)	Comments
Benzodiazepines[1]					
	Temazepam (Restoril)	Short-term treatment of insomnia (7–10 days)	7.5–15.0	10–15	Long half-life carries increased risk of next-day drowsiness. AE 5–10%: Drowsiness, headache*, nervousness*.[2]
	Triazolam (Halcion)	Short-term treatment of insomnia (7–10 days)	0.0625–0.25	1.7–5	Risk of confusion, daytime anxiety, anterograde amnesia. AE 10–15%: Drowsiness, headache. AE 5–9%: Dizziness, nervousness, light-headedness, ataxia, nausea [2]
Nonbenzodiazepine					
	Eszopiclone	No short-term limitation for use	1–2	9	AE 10–15%: Headache*, unpleasant taste. AE 5–9%: Dry mouth, dyspepsia, pain, dizziness [2]
	Zolpidem (Ambien, Edluar (sub-lingual form))	Short-term treatment of insomnia (up to 35 days)	5	2.9–3.7	AE 5–10%: Drowsiness, dizziness.[2,4]
	Zolpidem ER (Ambien CR)	Sleep initiation or sleep maintenance insomnia	6.25	1.9–7.3	AE 10–20%: Headache. AE 5–9%: Dizziness, nausea, somnolence, nasopharyngitis.[2,4]
	Zolpidem, low dose sub-lingual (Intermezzo)	sleep maintenance with at least 4 hours of sleep time remaining	1.75	1.4–3.6	AE 2–5%: headache.[2,4]
	Zolpidem, oral spray (Zolpimist)	sleep initiation insomnia	5	2.5–3.1	AE 5–10%: Drowsiness, dizziness.[2,4]
	Zaleplon (Sonata)	Short-term treatment of insomnia (up to 30 days)	5	1	AE 30–40%: Headache*. AE 5–9%: Dizziness*, nausea*, asthenia*, abdominal pain*, somnolence*.[2]
Melatonin receptor agonist					
	Ramelteon (Rozerem)	Sleep imitation insomnia. No short-term limitation for use	8	1–2.6	AE 2–5%: Dizziness, somnolence, nausea, insomnia exacerbation.[2] Not a Schedule IV drug.
Antidepressants					
	Doxepin	Sleep maintenance insomnia	3	15.3	CI: Untreated narrow angle glaucoma or severe urinary retention. AE 5–10%: Somnolence. AE 2–4%: upper respiratory tract infection, nausea/emesis.[3]

Data From: Bloom HG, Ahmed I, Alessi CA, et al. Evidence-based recommendations for the assessment and management of sleep disorders in older persons. *J Am Geriatr Soc*. May 2009;57(5):761–789.

Krystal AD, Durrence HH, Scharf M, et al. Efficacy and Safety of Doxepin 1 mg and 3 mg in a 12-week Sleep Laboratory and Outpatient Trial of Elderly Subjects with Chronic Primary Insomnia. *Sleep*. Nov 2010;33(11):1553–1561.

All side effects listed were more common in the medication group than in the placebo group except when indicated with an asterisk (in this case, the occurrence of the side effect was similar in both the medication and placebo arm).

[1]The following benzodiazepines are FDA approved for insomnia, but should generally be avoided in older adults due to their long half-life: flurazepam (Dalmane), quazepam (Doral), and estazolam (ProSom).

[2]Warnings/precautions: Anaphylaxis, abnormal thinking/behavior changes, complex behaviors while not fully awake (sleep-driving, etc.), CNS depressant effects, worsening of depression or suicidal thinking.

[3]Warnings/precautions: Abnormal thinking/behavior changes, complex behaviors while not fully awake (sleep-driving, etc.), CNS depressant effects, worsening of depression or suicidal thinking.

[4]Concomitant administration with anti-depressants, such as sertraline or fluoxetine, may increase zolpidem levels.

Table 5
Risk factors for sleep apnea in older adults

Risk factor	Mehra et al., Osteoporotic Fractures in Men Sleep Study[118]*	Ancoli-Israel et al., San Diego Cohort Community-Dwelling Elderly[183]**
	Odds Ratio (confidence intervals)	Coefficient (p-value)
Female gender (reference: male)	Study sample all male	0.52 (0.025)
Snoring (>3–5 times/week)	2.01 (1.62–2.49)	
Age (per 5-year increase)	1.24 (1.15–1.34)	
Race (reference: Caucasian)		
African American	1.05 (0.66–1.68)	
Asian	2.14 (1.33–3.45)	
Hispanic	1.38 (0.85–2.22)	
Obesity (BMI >30 kg/m^2)	2.54 (2.09–3.09)	0.087 (0.0067)
Neck circumference (per 5-cm increase)	2.19 (1.88–2.56)	
Waist circumference (per 5-cm increase)	1.24 (1.19–1.29)	
Socioeconomic status (range 1–10, per 1 unit increase)	0.95 (0.90–1.00)	
Current smoker	0.63 (0.31–1.29)	
Alcohol (per category increase)	1.01 (0.96–1.06)	
Daytime Sleepiness		
Excessive daytime somnolence (Epworth Sleepiness Scale score >10)	1.41 (1.11–1.79)	
Napping		0.41 (0.033)
Falling asleep reading		0.102 (0.046)
Comorbid factors (self-report)		
Hypertension	1.26 (1.06–1.50)	
Diabetes mellitus	1.18 (0.93–1.51)	
Cardiovascular disease	1.24 (1.03–1.48)	
Heart failure	1.81 (1.31–2.51)	
Stroke	0.86 (0.55–1.37)	

* Logistic Regression with Respiratory Disturbance Index>15 events/hr. as outcome. Study sample male. Data presented as adjusted Odds Ratios (confidence intervals), with models adjusted for race, age, and body mass index (BMI) except for race, age, and obesity indices--these were adjusted for respective other subject characteristics. Alcohol categories were drinks per week defined as follows: 0, <1, 1–2, 3–5, 6–13, >=14.

** Logistic regression coefficients of dependent variables with Respiratory Disturbance Index>=10 events/hr. as outcome.

Table 6

Unique aspects of sleep apnea assessment for older adults

Component	Domain	Specific Considerations
History	Daytime Symptoms of Sleepiness	Existing scales, such as the Epworth Sleepiness Scale, may under-report sleepiness in older adults because they may not drive, etc. Naps: Voluntary or involuntary? Increased voluntary napping may be a result of loss of daily social rhythms and not necessarily a sign of excessive sleepiness
	Sleep signs and symptoms when visiting family/friends	Witnessed apneas, nocturnal choking or gasping, loud snoring: symptom-reporting may under-report symptoms in older adults who do not have a bed partner
	Additional hx suggestive of sleep apnea	Nocturia, gait problems/falls at night, nocturnal confusion
	Caregiver Aspects	Concerns of the patient's caregiver, or if the patient is a caregiver themselves, how feasible is diagnosis/treatment of their own sleep disorder in the context of their caregiving duties
	Medical History	Atrial fibrillation, congestive heart failure, cerebrovascular events, dementia, hypertension, COPD/asthma may influence pre-test probability of sleep apnea or complicate management.
	Differential Considerations	Rule-out atypical depression in an older adult, polypharmacy effects, hypothyroidism
Physical Exam	Dentition	Edentulous/missing teeth: Sleep apnea may worsen when dentures removed
	Upper extremity function	Arthritis may affect hand dexterity also. Can they apply portable polysomnography sensors?
		Drop arm test to screen for rotator cuff tears, assess shoulder range of motion. Can they apply a CPAP mask themselves?
	Cognitive status	Screen for memory impairment (e.g.: Animal Naming test: patient should be able to name at least 14 different animals in one minute)