CHAPTER

22

Late-Life Sleep and Sleep Disorders

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LATE-LIFE SLEEP AND SLEEP DISORDERS NORMAL LATE-LIFE SLEEP

In meta-analyses of cross-sectional studies, the sleep of relatively healthy older adults has been observed to be lighter (lower percentage of deep sleep stage N3 and higher percentage of light sleep stages N1 and N2) and more disrupted (longer time to fall asleep, increases in awakenings, greater difficulty in falling back to sleep) compared to younger adults (Ohayon, Carskadon, Guilleminault, & Vitiello, 2004). The profound reduction in deep sleep or stage N3 (i.e., delta [0–4 Hz] frequency EEG) is most prominent in men (Redline et al., 2004). This reduction is reflected in decreases both in the amplitude and prevalence of delta wave activity (Feinberg & Campbell, 2003). The shifts in sleep architecture tend to occur prior to 60 years of age and then reach a plateau; whereas, sleep continues to become more fragmented as older age progresses until late older adulthood (≥90 years). Older individuals also have been reported to have shorter sleep durations (Espiritu, 2008), and to be more phase advanced in the timing of their sleep such that they fall asleep earlier in the evening and wake earlier as well. However, the evidence is equivocal. The reasons for all of these age-related sleep changes are unclear. Changes in physical and psychological health as well as social rhythms may be contributory (Vitiello, Moe, & Prinz, 2002), and normal age-related changes in multiple neurobiologic substrates (e.g., growth hormone, cortisol, proinflammatory cytokines, hypocretin/orexin, serotonin, adenosine) may also play a substantial role in the quality, quantity, and timing of late-life sleep.

EPIDEMIOLOGY OF SLEEP DISORDERS IN LATE LIFE

Insomnia

Insomnia is defined as a complaint of one or more of the following: difficulty initiating sleep, maintaining sleep, waking up too early, and/or nonrestorative sleep (Bloom et al., 2009). These complaints are accompanied by daytime functioning impairments, and they occur in the context of sufficient opportunity to get the desired amount of sleep. The most commonly used classification systems used to diagnose insomnia are the Diagnostic Statistical Manual of Mental Disorders-5 (DSM-V; American Psychiatric Association [APA], 2013) and the International Classification of Sleep Disorders-2 (ICSD-2; American Academy of Sleep Medicine [AASM], 2005). Both systems define insomnia in concert with the previously described definition. The most recent edition of the DSM, the DSM-V, cross-references with the insomnia diagnoses in the ICSD-2.

TABLE 22.1 Studies Citing Prevalence of Insomnia and SDB in Older Adults

First author/ publication year	Country	Sample size	Age (years)	Method of sleep data collection	Prevalence estimate
INSOMNIA					
Bonanni/2010	Italy	1427	≥65	Reported complaint of insomnia symptoms	44.2%
Gureje/2009	Nigeria	2152	≥65	Reported complaint of insomnia symptoms	30.7%
Leger/2000	France	2456	≥65	Reported complaint of insomnia symptoms and related daytime impairment	20.4%
Lichstein/2004	United States	772	≥60	Reported complaint; sleep diaries with SOL or WASO ≥31 min, ≥3 nights per week, ≥6 months' duration; impaired daytime functioning	60–69: *M* = 9%, *W* = 17% 70–79: *M* = 23%, *W* = 26% 80–89 + : *M* = 23%, *W* = 41%
Liu/2005	China	1820	≥65	Reported complaint of insomnia symptoms and related daytime impairment	32.9% insomnia symptoms; 8.9% insomnia symptoms with daytime consequences

M, men; SOL, sleep-onset latency; W, women; WASO, wake after sleep onset.

The prevalence of insomnia in older adults varies across studies because of the different assessment methods used. Hence, the prevalence ranges from 9% to 44%, with higher rates obtained when insomnia was diagnosed without consideration of daytime impairments or the duration and frequency of the insomnia (see Table 22.1; Bonanni et al., 2010; Foley et al., 1995; Gureje, Kola, Ademola, & Olley, 2009; Leger, Guilleminault, Dreyfus, Delahaye, & Paillard, 2000; Liu & Liu, 2005). Across the lifespan, insomnia and general sleep disturbance are more prevalent among women than men, and this gender difference widens in older adulthood (Grandner et al., 2012).

Advanced Sleep Phase Disorder

Advanced Sleep Phase Disorder (ASPD) is a type of circadian rhythm sleep disorder. It is characterized by sleep and wake times that are typically more than 3 h earlier compared to societal norms, and often it is accompanied by excessive sleepiness during the afternoon and evening, and early morning awakenings (Reid & Zee, 2011). The quality and duration of the sleep during this time period is usually normal (Reid & Zee, 2011). Nonetheless, ASPD, according to both the DSM-V and the ICSD-2, is characterized by complaints of difficulty staying awake in the evening and inability to maintain sleep until a preferred time in the morning. The prevalence of ASPD among older adults is undetermined; however, it is commonly considered to be more prevalent in late-life because a fifth of older adults report early morning awakenings (Foley et al., 1995).

Sleep-Disordered Breathing

Sleep-Disordered Breathing (SDB) is defined as partial (i.e., hypopnea) to complete (i.e., apnea) cessations in breathing during sleep (Bloom et al., 2009). The most prevalent type of SDB is obstructive sleep apnea (OSA), which consists of apneas and hypopneas, caused by complete (apneas) or partial (hypopneas) obstructions in the upper airway (Bloom et al., 2009), occurring five or more times per hour of sleep (i.e., the apnea–hypopnea index, AHI),

with accompanying respiratory effort (AASM, 2005). The patient usually experiences excessive daytime sleepiness and unintentional sleep bouts as a consequence of interrupted sleep during the night due to these respiratory events. In the earliest large-scale study of sleep apnea in middle-aged adults, the prevalence of OSA was 2% in women and 4% in men (Young et al., 1993). However, in population-based studies of older adults the prevalence ranges from 15% to 24% when only the AHI is considered (Ancoli-Israel et al., 1991; Phillips et al., 1992).

ASSESSMENT

The essential parts of an initial sleep assessment given by a geropsychologist include a comprehensive clinical interview of the patient's sleep and daytime functioning as well as their medical, psychiatric, and substance use histories (Schutte-Rodin, Broch, Buysse, Dorsey, & Sateia, 2008). Once the initial retrospective assessment is administered, a prospective assessment is typically conducted by instructing the patient to complete 2-weeks' worth of sleep diaries. Depending on the nature of the patient's symptoms, current medical conditions, or ability to complete the diaries, objective measures of sleep such as polysomnography (PSG) or actigraphy, and medical laboratory testing may be needed. If their symptoms or current medical conditions indicate they may be high risk for sleep disorders, then objective measurement of sleep is recommended. For more detailed guidelines of the assessment of specific sleep disorders, please see the ICSD-2 (AASM, 2005).

Main Methods of Sleep Assessment

Both objective and subjective measurement approaches are important in assessing sleep in the older adult. The extent of use of either type depends on the specific sleep disorder in question.

Subjective Assessments

Sleep diaries are the best measure of subjective sleep because they are the least expensive and most time efficient (Lichstein, Durrence, Riedel, Taylor, & Bush, 2004). Recently, sleep diaries for adults were standardized to allow for direct comparison across research studies and clinical examinations (Carney et al., 2012). Sleep diaries do not alter the natural sleep environment or routines; thus, they are relatively accurate accounts of the patient's sleep from his or her own perspective. Sleep diaries also are less prone to recall bias in comparison to single-point retrospective accounts of sleep, which tend to overemphasize the poorest nights of sleep (Gorin & Stone, 2001). Sleep diaries provide prospective, albeit not in vivo, accounts of multiple sampling points that can be averaged to obtain a more representative sleep estimate than retrospective estimates. However, they are not without their disadvantages. Specifically, the three major disadvantages of sleep diaries are that they: (i) are prone to some measurement error because they are based on an individual's estimates of sleep patterns, (ii) cannot provide information on the presence and amount of sleep stages throughout the night, and (iii) do not record certain, unobserved sleep symptoms (e.g., apneic events, limb movements). Nonetheless, their accuracy relative to objective measures is similar (Lichstein et al., 2004).

Objective Assessments

PSG, the gold standard in the objective assessment of organic sleep disorders, traditionally lasts one to three nights in duration. These overnight visits can be expensive, inconvenient, and time-consuming. Older adults, who are suspected of having SDB, but have mobility issues, other safety concerns, or who are critically ill, may be good candidates for

portable PSG monitoring. However, a comprehensive sleep evaluation is still required to confirm the diagnosis. In-laboratory PSG is known to create the "first night effect," which means that sleep is substantially altered by the new environment and equipment (Kales & Kales, 1984). For older adults who have considerable difficulty overcoming the "first-night effect," portable PSG monitors may also be an option.

Wrist actigraphy is another measure of objective sleep. However, compared to PSG the number of measures it offers is limited (measures sleep continuity, duration, and timing only). However, its advantages include its ability to record sleep patterns and circadian rhythms over several weeks, and it is not as costly or as cumbersome as PSG (Ancoli-Israel et al., 2003). Actigraphy should always be used concurrently with sleep diaries to allow for verification of bedtimes and wake-times. In particular, actigraphy will help distinguish sleep from sedentary activity among older adults with a sedentary lifestyle (Ancoli-Israel et al., 2003). In cases of older adults who have difficulty recalling their sleep patterns, such as persons with dementia, actigraphy is a good option, particularly when combined with additional information from a proxy respondent.

SLEEP DISORDERS

Insomnia

Development

The development of insomnia has been conceptualized as following a progression from a premorbid or predisposing state to the experience of an acute, precipitating event that induces transient sleep disruption, and then in response, the adoption of behaviors and cognitions that are often intended to compensate for poor sleep but result in the perpetuation of the sleep disruption (Spielman & Glovinsky, 1991). Examples of predisposing factors that would increase the likelihood of responding to a precipitating event with subsequent sleep disruption are impaired plasticity, hyperarousal (discussed below), personality factors, and family history. Precipitating events vary in content, duration, and quality, but the core component of the precipitating event is that it is perceived as stressful. Perpetuating factors are behaviors and cognitions that in the short term appear to be logical solutions to eliminating sleep disruption (e.g., napping, extending time in bed, consuming sedating substances to fall asleep and stimulating substances to assist with wakefulness, etc.). Over the long term these behaviors and cognitions can create greater sleep pattern variability, a non-sleep-promoting environment, and unrealistic expectations of the sleep experience. Collectively, these behaviors and cognitions can perpetuate the sleep disruption beyond the duration of the original precipitating event.

Major Theories

Hyperarousal has been conceptualized as a predisposing factor for insomnia and a characterizing feature of the insomnia experience. A review on the hyperarousal model of insomnia by Riemann et al. (2010) stated that insomnia is actually a 24-h psychobiological disorder because its etiology is associated with alterations in neurobiological substrates as well as psychosocial stress, and maladaptive behaviors and cognitions. The evidence accumulated supports this model in younger adults, but little evidence is available in older adults, and the data available suggest the model does not fit older adults as well (Dzierzewski, O'Brien, Kay, & McCrae, 2010). The reason may be that late-life insomnia is generally characterized by comorbid insomnia. Thus, several interacting biological and medical factors may be competing with the influence of hyperarousal on the insomnia experience and presentation in older adults. A model of late-life insomnia should account for the effects of comorbid conditions,

normal age-related alterations in sleep, and an array of biopsychosocial factors, including hyperarousal.

Risk Factors for Late-Life Insomnia

Comorbidities

Multiple comorbid medical (e.g., cardiovascular and pulmonary diseases), psychological (e.g., depression, anxiety disorders), and pain-related conditions (e.g., osteoarthritis) are common precipitants to insomnia. However, insomnia is not regarded as "secondary" to the condition. Insomnia co-occurring with another medical or psychiatric condition is termed "comorbid" because the evidence suggests that insomnia, even if precipitated by an illness, can often become independent and self-sustaining as the patient responds to the sleep disturbance with well-meaning but self-defeating compensatory behaviors (sometimes called safety behaviors) and cognitions that ultimately perpetuate the disturbance (National Institutes of Health [NIH], 2005).

Social and Behavioral Changes

Many psychosocial and lifestyle factors tend to change in older adulthood, such as the transition to retirement, loss and bereavement, and decreased activity (Wolkove, Elkholy, Baltzan, & Palayew, 2007). The body's natural drive to maintain a balance between sleep and wake, also known as the sleep homeostat, can be negatively affected by these changes by reducing sleep drive.

Cognition

Within the definition of insomnia, the patient must perceive and report their sleep as disturbing. The perception and appraisal of one's sleep disturbance is a key cognitive factor in the transition from acute to chronic insomnia. Cognitive activity that is emotionally laden or highly arousing may perpetuate the insomnia. The normal age-related changes in the quality of cognitive processing (e.g., reduced processing speed and controlled attention) may increase the risk for perceptions and appraisals of sleep as disturbed. Dzierzewski et al. (2010) proposed that reductions in controlled attention specifically may alter the perception of initial and middle of the night sleep onset through disrupting the normal deactivation of sensory processing and consciousness centers. Furthermore, once a sleep disturbance is ongoing, the poor sleep may interact with current cognitive functioning and perpetuate the problem.

Socioeconomic Status

Poverty and low levels of educational attainment are related to greater sleep disturbance and insomnia complaints (Patel, Grandner, Xie, Branas, & Gooneratne, 2010). In a recent analysis of the National Health and Nutrition Examination Survey (2007–08) that examined the unique effects of various socioeconomic status (SES) indicators, lower educational attainment, no health insurance, and low food security were associated with increased likelihood of sleep complaints (Grandner et al., 2013). Although not thoroughly investigated, the pathways through which SES relates to insomnia symptoms may include low social support (Troxel, Buysse, Monk, Begley, & Hall, 2010), an unhealthy lifestyle (Gerber, Brand, Holsboer-Trachsler, & Puhse, 2010), and food insecurity (Grandner et al., 2013).

Hypnotic Dependence

Although not necessarily a traditional risk factor, hypnotic dependence is related to maintenance of chronic insomnia. Hypnotic-dependent insomnia is the presence of insomnia symptoms and complaints despite chronic use of hypnotic medications to treat the sleep disturbance. Often when a patient attempts to withdraw from the hypnotic, the insomnia temporarily worsens which incentivizes the patient to continue using hypnotics despite little to no efficacy (Dzierzewski, O'Brien, Kay, & McCrae,

2010). Because older adults seeking treatment for insomnia are likely to receive hypnotic medications as the first-line treatment, their risk of developing hypnotic-dependent insomnia is high.

Special Populations

Certain neurological diseases that are more prevalent in the elderly (i.e., dementia, Alzheimer's disease, Parkinson's disease) tend to interfere with the sleep and wakefulness of these patients (Rose & Lorenz, 2010). Caregivers to these patients are not immune to sleep disturbances themselves and often have multiple risk factors for insomnia, including nighttime disruptive behaviors from their patients (i.e., sundowning in Alzheimer's), caregiving-related stress, and age-related comorbidities. Hypnotic medications are often prescribed to patients with neurological diseases (McCrae, Dzierzewski, & Kay, 2009). However, their efficacy is not well established, and hypnotics are associated with a slew of side effects known to affect cognitive functioning (Deschenes & McCurry, 2009).

Assessment

Because insomnia in older adults is often comorbid with other chronic conditions, thorough assessment of other diagnoses, including other sleep disorders, is necessary to determine all medical, substance-related, and psychiatric factors that may be contributing to the insomnia complaint. Further, the geropsychologist must determine whether the complaint matches that of normal age-related changes in sleep or is beyond these changes.

Interventions

Pharmacological

Older adults with insomnia are most commonly prescribed hypnotic medications (Dzierzewski et al., 2010). A range of medications are indicated for the treatment of insomnia (e.g., benzodiazepine receptor agonists, sedating antidepressants, melatonin); however, they are not without their side effects. Even the newer benzodiazepine receptor agonist drugs (e.g., zolpidem, zaleplon, zopiclone, eszopiclone), which were designed to reduce negative side effects, are associated with impaired cognitive functioning, daytime sleepiness, an increased risk of falls, and parasomnia-like symptoms, such as sleep walking and eating. Both the increased risk of falls and sleep walking are particularly salient to older adults. Most hypnotics are indicated for short-term use (e.g., a week or two). Beyond that timeframe the patient's potential for tolerance and dependence increases (McCrae, Nau, Taylor, & Lichstein, 2006). If psychological treatments are unsuccessful or the insomnia is in its acute phase, then hypnotics should be considered for the short term.

Several hypnotic medications have demonstrated efficacy for 3–6-month use from double-blind, randomized, placebo-controlled trials; however zaleplon is the only medication that was tested in older adults for those lengths of time (Ancoli-Israel et al., 2003). In a meta-analysis of 24 randomized controlled trials of various durations of hypnotic administration among older adults, hypnotic medications once again demonstrated efficacy in improving sleep quality and reducing nighttime awakenings (Glass, Lanctot, Herrmann, Sproule, & Busto, 2005), but the magnitude of the improvements was small to moderate (Cohen's d of 0.14 and 0.63, respectively). Additionally, there were more adverse events with the medications in comparison to placebo. The most commonly reported events were motor vehicle accidents, falls, daytime fatigue, and cognitive events. Glass and colleagues indicated that the benefit–risk ratio for hypnotic use among older adults with insomnia was not favorable.

An alternative pharmacologic treatment for older adults is melatonin. Melatonin is a common over-the-counter sleep aid that is not regulated in the United States though it is licensed in Europe and other countries. Prolonged-release melatonin

has been found to improve sleep and morning alertness among middle-aged to older adults with no evidence of withdrawal effects or rebound insomnia upon discontinuation (Lemoine, Nir, Laudon, & Zisapel, 2007), but here too, treatment effects are small (reduction in sleep-onset latency of 12 min; Buscemi et al., 2005).

Psychological

The symptoms and etiologies of late-life insomnia are often numerous and complex, requiring a multifaceted treatment approach. Cognitive and behavioral treatments for insomnia, such as sleep education, relaxation therapy, sleep hygiene, stimulus control, sleep restriction, and cognitive therapy (see Table 22.2; Carney & Edinger, 2010), have demonstrated efficacy, with a collective package of these treatments having the most impact. This package is termed cognitive-behavioral therapy for insomnia (CBT-I). CBT-I has demonstrated substantial treatment efficacy across many trials for both primary and comorbid insomnia (moderate-to-large effect sizes: 0.65–0.94) on outcomes of insomnia symptoms (Irwin, Cole, & Nicassio, 2006), as well as daytime and mood-related symptoms. Approximately 70–80% of patients exhibit improvements in sleep, post-treatment (Morin, Culbert, & Schwartz, 1994). Older adults with insomnia also have shown significant improvement in their insomnia symptoms (Edinger & Sampson, 2003; Pallesen et al., 2003). Because of the evidence, CBT-I is now regarded by national agencies as the first-line treatment for chronic insomnia above and beyond that of pharmacological interventions.

Psychological and Pharmacological Approaches Combined

A combined treatment approach with CBT-I and hypnotic medication may be beneficial for older adults. A four-condition study (CBT-I, CBT-I + temazepam, temazepam, and placebo) found that all treatments improved insomnia at similar magnitudes compared to placebo (Morin, Colecchi, Stone, Sood, & Brink, 1999). Though the participants reported they preferred CBT-I, and over 2-year follow-up, CBT-I alone was more efficacious than the other active treatment conditions. Variation in the efficacy of combined treatment (CBT-I + zolpidem) compared to CBT-I or zolpidem alone was also found in a study of older adults (Morin et al., 2009). At post-treatment, all treatment conditions produced equivalent improvements in sleep continuity as well as comparable rates of response and remission; though combined treatment had an earlier response. However, at follow-up, the efficacy of the combined therapy when participants continued to take zolpidem was not as optimal compared to participants that discontinued medication after initial treatment. This result led Morin and colleagues to recommend that hypnotic administration occur first followed by behavioral treatment and hypnotic discontinuation. Overall, combination therapy is not consistently better than CBT-I alone; therefore, the AASM recommended that, in practice, CBT-I alone should be used whenever possible (Schutte-Rodin et al., 2008). If combined therapy for older adults does occur in practice, then lower doses of hypnotic medications should be used as well as strategic timing of medication administration for other comorbid conditions.

CBT-I and Hypnotic Reduction

Even without discontinuation of hypnotic medication, CBT-I is effective in improving insomnia (Soeffing, Lichstein, & Nau, 2008). Simultaneous CBT-I and discontinuation of hypnotic medications among hypnotic-dependent participants has also been found to improve sleep, and treatment benefits were maintained at 1-year follow-up (Lichstein et al., 2013). CBT-I, as a strategic tool for hypnotic medication discontinuation among dependent patients, has also been found to be effective (Baillargeon et al., 2003). Overall, the evidence suggests that CBT-I can be used effectively for

TABLE 22.2 Multicomponent CBT-I

SLEEP HYGIENE
1. Eliminate or reduce caffeine use after 12 pm **2.** Do not drink alcohol within 2 h of bedtime **3.** Do not use tobacco within 2 h of bedtime **4.** Do not eat heavy meals within 2 h of bedtime **5.** Do not exercise within 2 h of bedtime (though routine exercise is encouraged)
STIMULUS CONTROL
1. Lie down to go to sleep only when you are sleepy **2.** Do not use the bed for anything except sleep and sex. Do not eat, read, watch television, or worry in bed **3.** If you cannot fall asleep within 10 min, get up and go to another room. Only return to bed when you feel sleepy again **4.** If you return to bed and still cannot fall asleep, repeat Step 3. Do this as often as necessary throughout the night **5.** Set your alarm and get up at the same time every morning regardless of how much you slept during the night. This will help your body acquire a constant sleep rhythm **6.** Do not nap during the day
SLEEP RESTRICTION/SLEEP COMPRESSION
• Aims to match the patient's time spent in bed to their actual time spent sleeping • Prescribe bed and wake times that more closely reflect time spent asleep • Sleep restriction abruptly tailors the time in bed to reflect sleep needs • Sleep compression gradually reduces time spent in bed to match sleep time
RELAXATION
• Diaphragmatic breathing, biofeedback, imagery, and meditation are all appropriate relaxation approaches for insomnia treatment • Progressive muscle relaxation (PMR) is an empirically supported treatment by the AASM • Leading patients through a deep breathing exercise, followed by alternatively tensing and relaxing muscle groups (e.g., arms, neck, back, legs) while attending to feelings of relaxation during and after the process
COGNITIVE THERAPY
• Identifying maladaptive beliefs about sleep and replacing them with more adaptive thoughts and attitudes • Integrates basic education about sleep; understanding normative sleep patterns and experiences can be helpful in addressing mistaken beliefs about sleep

sleep improvement in the case of simultaneous hypnotic use and medication tapering and withdrawal.

Advanced Sleep Phase Disorder

Development

Desynchronization between environmental factors (such as the light–dark cycle) and internal circadian timing mechanisms is at the root of circadian rhythm disorders. For shift work disorder or jet lag, the desynchronization occurs as a result of environmental factors such as working night shift. For circadian rhythm disorders such as ASPD, however, the desynchronization is a result of a disrupted internal timing system (Reid & Zee, 2011). As a result, the sleep–wake cycle of an individual with ASPD becomes out of sync with their external environment.

Major Theories

Several hypotheses have been proposed to explain the development of ASPD in older

adults including shortening of the endogenous length of the sleep period, alterations in response to light, and gene polymorphisms. First, researchers have investigated the hypothesis that the intrinsically driven circadian period shortens with age. That is, if the 24-h period encompassing sleep and wake was shortened in older adults, they may experience a phase advanced relative to younger adults. Conflicting results from both animal and human studies suggest that the circadian period is likely not shortened with age, and, therefore, not responsible for a higher prevalence of ASPD in older adults (Welsh & Ptacek, 2010). Second, age-related behavioral and physiological changes affecting exposure to light may contribute to ASPD in older adults. Older adults, especially those residing in long-term care settings, may receive less bright-light exposure or light exposure in the evening which could help to delay the circadian phase. Even when older adults are exposed to light, changes in vision with age such as the formation of cataracts and macular degeneration can diminish the amount and spectral composition of light absorbed (Kim et al., 2014). Third, genetic analyses using familial studies of ASPD have identified genetic factors implicated in ASPD (i.e., the circadian clock gene *hPer2* (Toh, Jones, He, Eide, & Vinz, 2001), CKI-delta mutation (Xu et al., 2005); Reid & Zee, 2011).

Assessment

ASPD may be under-diagnosed as symptoms of ASPD may appear similar to symptoms of other disorders. For example, older adults may complain of unwanted early morning awakenings similar to symptoms of depression. Also, older adults may identify the unwanted early wake time as insomnia, when, in fact, their sleep is good but just advanced. Lastly, complaints of excessive daytime sleepiness may: (i) reflect the advancing of the sleep phase rather than decreased alertness during the day (Reid & Zee, 2011) or (ii) could reflect a resistance to the ASPD that results in later bedtimes and less sleep time, resulting in daytime sleepiness (Neikrug & Ancoli-Israel, 2010). Unlike delayed sleep phase disorder, which can interfere with morning activities, the earlier bedtimes seen with ASPD are less likely to interfere with societal demands and, therefore, ASPD may be underreported (Welsh & Ptacek, 2010). Assessment for ASPD consists of monitoring sleep over an extended period (e.g., 14 days) using sleep diaries or actigraphy to identify a consistent advance of the sleep period (AASM, 2005).

Interventions

Two approaches are primarily recommended for the treatment of ASPD: chronotherapy and phototherapy (Morgenthaler et al., 2007). Chronotherapy uses sleep-scheduling to delay the timing of the sleep–wake cycle. Bedtime is delayed until a desired time to fall asleep is reached. For example, if the individual currently falls asleep at 6 pm, they would successively delay the onset of sleep, starting with 7 pm, followed by 8 pm the next night, 9 pm the next night, and so on until the desired bedtime is reached. As delaying sleep onset can be difficult due to excessive sleepiness, engaging in sufficiently stimulating activities can help to prolong wakefulness. Phototherapy is a second approach to treating ASPD. Generally, phototherapeutic approaches involve limiting exposure to morning light and increasing exposure to evening light (Morgenthaler et al., 2007). The older individual should sleep in a darkened room and limit early morning light exposure through the use of blackout curtains. Sunglasses can also be used to reduce morning light exposure while outside. They should seek outdoor light exposure in the late afternoon and early evening and use bright lights during the evening (Welsh & Ptacek, 2010).

Sleep-Disordered Breathing

Development

Upper airway collapse resulting in OSA events is often due to several anatomic and neuromuscular factors, such as age-related loss of muscle and muscle tone, excessive bulk of tissues, and/or abnormality in craniofacial structures (i.e., maxillomandibular malformation, enlarged adenoids or tonsils). Therefore, loss of airway patency with age increases risk for developing SDB (AASM, 2005).

Type—OSA Versus Central Sleep Apnea

In contrast to OSA, central sleep apnea (CSA), another major type of SDB, consists of recurrent episodes of apnea resulting from a loss of ventilatory drive (Bloom et al., 2009). No obstruction of the airway occurs in CSA. CSA is induced by one of two physiological mechanisms. The first is post-hypocapnia (reduction in arterial carbon dioxide [$PaCO_2$]) hyperventilation, and the second is hypoventilation due to instability in the neuromuscular output from the respiratory control system (Aurora et al., 2012). CSA events occur most often during wake to sleep transitions when $PaCO_2$ levels may be insufficient to maintain alveolar ventilation. Medical conditions associated with CSA events are neurological diseases, pulmonary diseases, congestive heart failure, stroke, and renal failure. All of these diseases are also more common in older adults. Persons with insomnia may also be susceptible because of the numerous transitions from wake to sleep that occur with sleep fragmentation.

Risk Factors

One of the major risk factors for OSA is older adulthood. Other risk factors include excess weight especially located at the neck, a family history of OSA, smoking, hypothyroidism, Down's syndrome, and menopause. Although menopause is a risk factor that raises women's incident rates to similar levels as men, men at all ages are at greater risk for OSA (Bixler et al., 2001). In a recent meta-analysis, African-Americans were also found to be at greater risk for both prevalent OSA and more severe OSA than non-Hispanic Whites; a result not moderated by age (Ruiter, DeCoster, Jacobs, & Lichstein, 2010). Patient factors that can exacerbate OSA include nasal congestion, alcohol, and use of other substances with sedative properties, such as sleep medications and anxiolytics (ICSD-2). Sleep medication use is significantly higher among older adults compared to younger adults (Espiritu, 2008); therefore, it is a particularly important contributory factor.

Assessment

To verify the presence of OSA, PSG recording is necessary. To meet clinical criteria for the diagnosis, the patient must have five or more scoreable apnea or hypopnea events per hour of sleep lasting at least 10 s with accompanying respiratory effort as evidenced by increased esophageal pressure. A recent update of the criteria for scoring respiratory events is outlined in Berry et al. (2012). Along with this objective evidence, the patient must complain of at least one of the following: excessive daytime sleepiness, unintentional sleep bouts, fatigue, insomnia, or nonrefreshing sleep (AASM, 2005). Both the ICSD-2 and the DSM-V require that OSA is not better explained by another disorder. Older adults may misattribute their symptoms to another ongoing medical or psychiatric conditions as well as appraise their symptoms as part of the aging process (e.g., snoring, nocturia, fatigue, cognitive dysfunction, unintentional napping, etc.). As a consequence, they may not seek medical attention readily. Geropsychologists ought to regularly screen for SDB in older adults.

Intervention

Continuous positive airway pressure (CPAP) devices are the gold standard treatment for

OSA in older adults. CPAP devices are portable machines that deliver positive pressure through the normally obstructed airway via facial mask. By maintaining an open airway, CPAP devices reduce the AHI, normalize blood oxygen saturation, reduce nighttime arousals associated with apnea or hypopnea events, increase deep sleep, and decrease self-reported symptoms, such as gasping for breath, snoring, and observed apneas (Sawyer et al., 2011). In older adults, CPAP has been shown to reduce self-reported daytime sleepiness and to improve cardiac functioning. Each patient may vary in the level of positive pressure they need to receive to maintain airway patency, so PSG is needed to adequately titrate the CPAP device.

Given the demands of use of the CPAP device and the higher prevalence of complicated, comorbid conditions, older adults may have difficulty with adhering to CPAP. The lowest recommended standard for adequate adherence is 4 h per night on 70% of nights (Sawyer et al., 2011). Greater duration and frequency is associated with better sleep and health-related outcomes. Factors that are associated with poor adherence in older adults include depression, nocturia, nasal irritation, smoking, claustrophobia, and mask intolerance (Weaver & Chasens, 2007; Weaver & Sawyer, 2010). Combined education on the benefits of CPAP use and risk of non-use, individualized treatment plans, and follow-up on any issues with CPAP that arise within the first few days of treatment are associated with high adherence rates in older adults (Sawyer et al., 2011; Weaver & Chasens, 2007; Weaver & Sawyer, 2010). A study on the efficacy of CBT for CPAP adherence (i.e., goal development, managing treatment expectations, and addressing negative cognitions associated with CPAP use) plus education about OSA and CPAP found that the intervention increased CPAP adherence within the first month of treatment compared to patients who received treatment as usual (Richards, Bartlett, Wong, Malouff, & Grunstein, 2007).

EXPECTANCIES REGARDING INTERVENTIONS AND OUTCOMES IN OLDER ADULTS

Evidence-Based Treatments

Does CBT-I Work for Older Adults?

The resounding answer to this question is "yes." Two reviews of the evidence found that CBT-I is appropriate and effective in older adults causing moderate to substantial improvements in insomnia among older adults (≥55 years) with effects lasting up to 2 years (Irwin et al., 2006; Morin et al., 1999). CBT-I for older adults is now deemed a standard, evidence-based treatment for older adults (NIH, 2005).

Treatment for Comorbid Insomnia in Older Adults?

Historically, insomnia comorbid with medical and psychological conditions was not directly treated. The common assumption was that treatment of the medical or psychological condition would result in the resolution of the insomnia (McCrae et al., 2009). However, evidence suggests insomnia can be considered as an independent disorder (as was recently recommended in the DSM-V), because the insomnia presentation can be perpetuated by maladaptive behaviors and cognitions even after the resolution of, or reduction in the severity of, the comorbid illness (Harvey, 2005). This is often the case with older adults, because comorbid insomnia is more common than primary insomnia in this population. Fortunately CBT-I is effective for insomnia comorbid with a wide array of medical and psychiatric conditions. There are indications that CBT-I in older adults improves sleep directly, *and* it may indirectly improve some of the comorbid conditions' symptomatology including psychological distress, quality of life, and various measures of daytime functioning (Bloom et al., 2009). Implementation of CBT-I in older adults as opposed to pharmacological treatments also reduces the risk of

polypharmacy that is often a potentially dangerous issue with comorbid insomnia.

Treatment of Comorbid Apnea and Insomnia?

Considering the high prevalence of sleep fragmentation and SDB in the elderly population, comorbid insomnia with SDB is a highly likely scenario (Luyster, Buysse, & Strollo, 2010). Proper treatment is crucial; however, there is very little research on treatment of concurrent apnea and insomnia. In a trial of the effect of CBT-I and SDB treatment (i.e., CPAP, oral appliances, surgery) for comorbid insomnia and SDB, sleep measures and daytime functioning improved, and there was a higher insomnia remission rate (Krakow et al., 2004). CBT-I alone was not as effective for remission or for sleep-related daytime functioning improvement. In a study comparing CBT-I with surgical treatment for OSA in patients with insomnia and mild OSA, results indicated that surgical treatment resolved insomnia symptoms in a third of patients; whereas, patients receiving CBT-I alone had no remission (Guilleminault, Davis, & Huynh, 2008). However, CBT-I received after surgical intervention improved sleep further than SDB treatment alone. These results imply that simultaneous treatment of SDB and insomnia may be the best treatment approach. This conclusion is further bolstered by results from an observational study of insomnia symptoms before and 2 years after the initiation of CPAP treatment for OSA (Björnsdóttir et al., 2012). Results indicate that complaints of difficulty maintaining sleep were reduced with CPAP treatment, but difficulty initiating sleep and early morning awakenings persisted.

Benzodiazepines for the treatment of concurrent insomnia and apnea have not been thoroughly studied, most likely because these medications have respiratory suppressant effects that could worsen OSA severity (Luyster et al., 2010). On the other hand, the more recently developed GABAergic non-benzodiazepine drugs, such as zaleplon, zolpidem, and eszopiclone, do not appear to affect respiration or upper airway muscle tone (Berry & Patel, 2006; Rosenberg, Roach, Scharf, & Amato, 2007). However, these drugs are still not without their side effects. Close monitoring and great care in the dosing of these hypnotics must be taken, particularly when they are used in conjunction with other medications. Additionally, physiological habituation, dependency, and rebound insomnia upon withdrawal are concerns that must be taken into account.

Caregiver Involvement and Treatment

Dementia Patients and Caregivers

Approximately two-thirds of older adult caregivers of patients with forms of dementia report sleep disturbances (McCurry, Logsdon, Teri, & Vitiello, 2007). The chronic need to be vigilant both day and night to their patient's needs, and frequent nighttime awakenings by the patient can create a context ripe for insomnia manifestation in caregivers who are predisposed and vulnerable. Given that many caregivers are older adults who may have medical conditions themselves, and the need to be functional during the night if necessary, pharmacological interventions are less preferable. Two non-pharmacological options are more desirable: behavioral treatment programs to improve the patient's sleep and programs to improve the caregiver's sleep. A randomized trial of a sleep hygiene and behavior management program for caregivers to deliver to patients with dementia led to greater reductions in nighttime awakenings and duration compared to general education and caregiver support (McCurry, Gibbons, Logsdon, Vitiello, & Teri, 2005). A brief CBT-I protocol has also demonstrated improvements in sleep quality and sleep efficiency in elderly caregivers of dementia patients (McCurry, Logsdon, Vitiello, & Teri, 1998).

CONCLUSIONS AND FUTURE DIRECTIONS

Education, assessment and/or diagnosis, and treatment represent several ways that geropsychologists can contribute to the care of sleep and sleep disorders in older adults. Geropsychologists can provide needed education to patients, their families, and other health care professionals who work with elderly patients. Education about sleep, sleep disorders in old age, the importance of sleep, and adaptive expectations of sleep in later life are all important topics that geropsychologists can raise. A large proportion of the patients that geropsychologists treat are likely to be experiencing some form of sleep disturbance. Hence, geropsychologists are in a prime position to routinely screen for sleep disorders in their patients and to refer them for further assessment and treatment. Lastly, geropsychologists may want to treat sleep disturbances in their patients themselves. To do so, specialized training in sleep psychology, a newly recognized specialty in psychology, is needed. To become certified in behavioral sleep medicine, providers should visit the American Board of Sleep Medicine website to determine what type of training is required for certification (www.absm.org). Most of the providers that are certified are psychologists; however, the overall number of certified providers is only a couple of hundred. Clearly, there is great need for the dissemination of these evidence-based psychological interventions for sleep. Although sleep psychology was recently recognized as a formal specialty by the American Psychological Association, it is still in its infancy. With time and further growth of this new specialty, more opportunities will be available for geropsychologists to receive training in assessment and treatment of sleep disorders in older adults. If this training is not feasible for the provider, then a referral list of sleep psychologists and local sleep clinics is recommended. A list of certified behavioral sleep medicine providers and their locations can be found on the American Board of Sleep Medicine website (http://www.absm.org/BSMSpecialists.aspx).

Future research on the dissemination and implementation of evidence-based interventions for late-life sleep disorders is crucial given projected increases in the proportion of older individuals in the US and worldwide populations. Although the efficacy of these interventions has been established for some time, dissemination has not occurred rapidly. This is mainly because of the lack of geropsychologists and other psychologists trained to provide such services as mentioned earlier. Other reasons may be barriers to the implementation of these interventions in current health care systems. Researchers are actively investigating ways to overcome these barriers to foster greater recognition of late-life sleep disturbances and treatment at the point of patient presentation—primary care. This research has focused on the fidelity and disseminability of briefer versions of CBT-I (less than eight sessions), and motivational interviewing for CPAP adherence that are adapted for primary care settings. Investigations of different modalities for delivering treatment are also underway with particular emphasis on mobile and online delivery systems (i.e., telephone, video-conferencing, online programs, smart phone applications). As more geropsychologists become trained in behavioral sleep medicine, additional research will be needed to investigate the best methods for integrating geropsychologists in primary care settings to increase treatment access for older adults afflicted with disordered sleep. Other important lines of future research are exploring community-based and public health interventions for sleep screening and treatment, implementing interventions in full-time care facilities, developing and testing interventions for high-risk and highly comorbid older adult patients, and testing the effectiveness of treatments designed to improve the sleep of both caregivers and patients alike.

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