Insomnia

Behavioral Treatment in the Elderly

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KEYWORDS

• CBT-I • Elderly • Nonpharmacologic • Insomnia • Behavioral • Cognitive • Age

KEY POINTS

- Sleep issues in elderly individuals can often be attributed to increased mental and physical health problems associated with aging. Nonetheless, sleep drive decreases and fragmentation increases as we age.
- After the age of 20, circadian phase advances, causing many older patients to have an abnormally early sleep and wake time.
- Cognitive behavioral therapy for insomnia (CBT-I) is the preferred treatment approach for insomnia in all adults, including elderly individuals.
- Adding bright light therapy to CBT-I can help delay an advanced circadian phase.

INTRODUCTION

In this article, changes that occur in sleep with age, prevalence of insomnia, the most influential models explaining the development of insomnia, as well as nonpharmacologic treatment methods to improve poor-quality sleep are concisely reviewed within the context of older patients. In addition, other popular treatment techniques, such as sleep hygiene and melatonin use, are briefly mentioned.

AGE-RELATED CHANGES IN SLEEP ARCHITECTURE

Throughout the human life span, changes occur in the architecture, length, and quality of sleep.[1] During infancy and early childhood, we sleep more than any other point in postnatal life and spend a much larger percentage of time in both rapid-eye movement (REM) and slow-wave sleep (SWS).[2] Until the age of 60, total sleep time (TST), sleep efficiency, percentage of SWS, percentage of REM sleep, and REM latency all significantly decrease.[1] Wake after sleep onset, stage 1, and sleep latency are all increased

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in older compared with younger adults.[1,3] Many of the changes in sleep architecture, such as decreased REM and SWS, stabilize around the age of 60. Unfortunately, sleep efficiency continues to decline into old age.

INSOMNIA PREVALENCE

Insomnia is estimated to affect 6% to 15% of the population,[4] and is therefore one of the most prevalent health conditions worldwide. A large-scale epidemiologic study found that 23% to 34% of individuals older than 65 complained of symptoms consistent with insomnia, with more than 50% reporting difficulty sleeping (not reaching the level of insomnia).[5] Some have argued that changes in sleep architecture associated with aging are not indicative of reduced sleep need, but instead point toward reduced sleep ability, which is related to circadian rhythm alterations as well as psychiatric and health issues that tend to occur with age.[6] Moreover, once mental and medical health conditions are controlled for, the prevalence of insomnia in older adults is reduced and is similar to younger populations.[7] In addition, addressing mood and health conditions in elderly individuals often improves sleep quality. Nonetheless, insomnia remains a common complaint in elderly individuals and if not treated will decrease quality of life.

MODELS OF INSOMNIA

The 2-Process Model

Although not specifically a model of insomnia, the 2-Process model helps us to understand the factors that control alertness and sleep throughout the 24-hour day[8] and helps us to explain changes in sleep quality as we age. This model describes sleep propensity (homeostatic drive or process S) as a physiologic drive that builds in a monotonic fashion during periods of wakefulness. During sleep, this drive is relieved in the form of slow-wave activity (SWA) in the electroencephalogram. Periods of sleep deprivation result in a rebound of SWA during subsequent bouts of sleep, and naps during the day reduce SWA at night by reducing process S during the napping period.

Process C, the second part of the 2-Process model, describes the influence of circadian rhythms on sleep and alertness. This process is regulated by *zeitgebers* (German for time giver) that adapt various functions of the body to a 24-hour rhythm. In humans, light is a powerful zeitgeber.[9,10] Bright light exposure in the eyes of humans stimulates photosensitive retinal ganglion cells that transfer signals via the retinal hypothalamic pathway to the suprachiasmatic nucleus, which is the master clock of the body. The suprachiasmatic nucleus, through humoral and neuronal signals, regulates peripheral clocks throughout the body.[11]

As we age, changes in the circadian rhythm occur. In fact, Roenneberg and colleagues[12] proposed that the point of maximal delay and a subsequent shift to a more advanced circadian phase, which occurs at approximately 20 years of age, is an indication of a biological shift from adolescence to adulthood. Throughout adulthood and into old age, the circadian phase continues to advance.[12] Although the genetic properties of the circadian clock in cells throughout the body do not change in older adults, it has been hypothesized that a thermolabile factor in the blood serum of older individuals is responsible for a shortening of the circadian rhythms,[13] resulting in an advance of the sleep phase.

CHANGE IN CIRCADIAN PHASE WITH AGE

The advance in the circadian phase means that older individuals tend to both fall asleep and wake up earlier than younger individuals (**Fig. 1**).[14] In some cases, sleep

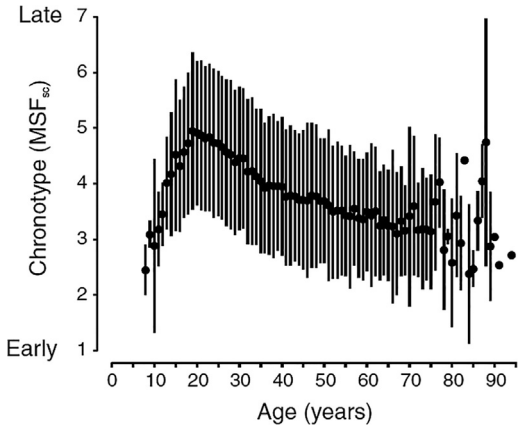

Fig. 1. Illustration of chronotype by age using the midpoint of sleep on free days derived from results on the Munich Chronotype Questionnaire. A higher value on the y-axis shows a more delayed circadian phase. (*From* Roenneberg T, Kuehnle T, Pramstaller PP, et al. A marker for the end of adolescence. *Curr Biol.* 2004;14(24):R1038-1039; with permission.)

can advance to the point of interfering with evening activities and can cause abnormally early morning wake-up times and may need to be treated. This is described in more detail later in this article.

The Spielman Model

The development of insomnia is complex, in part, because often the issues that led to the onset of disease are different from those that cause the difficulty sleeping to continue. Therefore, Spielman and colleagues[15] developed the 3-P (also known as the Spielman) model to help explain the change of insomnia over time from acute condition to chronic disease. The 3 P's in this model stand for predisposing, precipitating, and perpetuating factors.

Predisposing traits can be thought of as the underlying combination of both the personality makeup and the level of physiologic arousal of an individual and is relatively stable over time. The term hyperarousal is often used in the psychological literature to describe a specific predisposing trait that, if high, places a person at greater risk of developing insomnia. Folks with a high level of hyperarousal keep a consistently elevated level of vigilance. As a result, even small stressors can place these individuals at risk of acute difficulty sleeping.

Precipitating events are situations that create a high level of stress or change in lifestyle and can be either positive or negative in nature; for example, the death of someone close, changing jobs or careers, moving to a new home, or a serious conflict with someone, to name just a few instances. Nearly everyone encounters events that cause short-term difficulty sleeping. In most cases, once the stressful event dissipates, sleep returns to normal.

Perpetuating factors are behaviors that convert acute insomnia to a chronic condition. The single most damaging perpetuating activity to sleep is increasing time in bed (TIB). Often when people develop difficulty sleeping, they will increase TIB to help increase the chance that they will be able to recapture the lost sleep. However, if increased TIB is maintained for extended periods of time, it will almost certainly cause insomnia to become a chronic condition long after the original stressor has been

resolved. Other perpetuating activities can include maladaptive behaviors, such as working on the computer, reading, watching TV, or eating in bed.

Neurocognitive Model

Perlis and colleagues[16] elaborated on the Spielman model by describing how repeated episodes of insomnia, which result in a combination of cortical, cognitive, and somatic arousal, result in a classically conditioned hyperaroused response to sleep-related stimuli. Once conditioned, hyperarousal becomes a perpetuating factor that persists long after precipitating events cease to play a role in elevating stress. This theory is evidenced by studies showing increased cortical arousal in people with insomnia.[17–20]

Attention-Intention-Effort Pathway

Another framework to help understand insomnia has been proposed by Colin Espie and colleagues.[21] This model is called the Attention-Intention-Effort pathway. This theory describes how difficulty sleeping is worsened or perpetuated by direct attention on an activity that is normally an automatic process. In other words, good sleepers do not think about the process of falling asleep and maintaining sleep. However, as sleep quality degrades, attention becomes focused on the process of sleeping, which in turn causes increased arousal and turns an acute insomnia into a chronic condition.

Two-Factor Model

Developed by Bonnet and Arand in the late 1990s,[22] the 2-factor model describes insomnia as involving 2 competing drives. One is the physiologic arousal level of the individual and the other is the sleep drive. Having a low sleep drive or an elevated level of arousal predisposes a person to insomnia. This model is based, in part, on studies that show, despite inadequate sleep, people with insomnia are less sleepy than those with normal sleep when given a multiple sleep latency test,[23] which is an objective test of daytime sleepiness. Moreover, when normal sleepers are experimentally driven to higher levels of anxiety, they tend to develop poor-quality sleep.[24] This model has good face validity because it is easy to relate to having trouble sleeping after a stressful day, even if one does not consider oneself to be an insomniac. The 2-Factor model explains this phenomenon through a brief increase in arousal level due to the stressful day, which acutely surpasses the level of sleep drive.

The 2-Factor model is simple to understand and describes 2 important targets of treatment. Therefore, it is useful to use this model to describe insomnia to patients, particularly when focusing on a treatment approach such as sleep restriction therapy (SRT; described in more detail later in this article), which is designed to increase sleep drive to overcome the arousal level of the individual with insomnia. Describing SRT in terms of the 2-Factor model helps patients to understand the theory behind reducing TIB to help increase sleep drive, and how a high sleep drive can overcome elevated levels of anxiety without focusing directly on the anxiety.

Nonpharmacologic Treatment Models for Insomnia

According to the American College of Physicians, cognitive behavioral therapy for insomnia (CBT-I) should be used as the first-line treatment for primary insomnia.[25] Although hypnotic medication can be useful for acute insomnia, CBT-I is the preferred long-term treatment choice for difficulty sleeping because it addresses the factors that perpetuate the problem. CBT-I is a multi-element therapy that includes stimulus control, cognitive therapy, relaxation-based treatment, and sleep restriction (**Table 1** below).

Table 1
The first recommendation listed is the current guideline[26]

Therapeutic Approach	Focus of Intervention	AASM Level of Recommendation
Multicomponent cognitive behavioral therapy	Using all the techniques listed below in combination.	[a]Standard[26] (+Strong[27])
Stimulus control	Conditioning the bed and bedroom to be associated with sleep	[a]Standard[26] (++Conditional[27])
Cognitive therapy	Restructuring maladaptive thought processes about sleep	[a]Standard[26] (+++No recommendation[27])
Relaxation-based treatment	Reducing tension	[a]Standard[26] (Conditional[27])
Sleep restriction	Increasing sleep drive to overcome level of arousal	[b]Guideline[26] (Conditional[27])
Mindfulness	Teaching nonjudgmental awareness to help reduce anxiety about sleep	N/A[26] (+++No recommendation[27])
Sleep hygiene	A collection of behaviors or actions that should either be avoided or adopted to improve sleep quality	+++No recommendation[26] (Conditional recommendation not to use[27])

The American Academy of Sleep Medicine's (AASM) level of recommendation in brackets shows the recently released (2021) recommendations.[27]

+Strong: Indicates a treatment that will benefit most patients. ++Conditional: A recommended treatment that should be used based on clinical judgment. +++No recommendation: A treatment without adequate evidence of benefit. N/A: not assessed in review.

[a] Standard: This is a generally accepted patient-care strategy that reflects a high degree of clinical certainty.

[b] Legend: This is a patient-care strategy that reflects a moderate degree of clinical certainty.

More recently, mindfulness-based interventions also have been added to the armamentarium.

COGNITIVE BEHAVIORAL THERAPY FOR INSOMNIA TREATMENT MODALITY, FOCUS OF INTERVENTION, AND LEVEL OF AMERICAN ACADEMY OF SLEEP MEDICINE RECOMMENDATION

Stimulus Control Therapy

Originally developed by Bootzin[28] and based on operant conditioning, stimulus control therapy is the concept that pairing waking activities with the bed and bedroom prevents the conditioned association of the bed with sleep. For example, consistently pairing TV watching, reading, and/or eating in bed will result in the bed and bedroom being associated with activities related to wakefulness, whereas if the bed and bedroom are only used for sleep, it increases the likelihood that sleep will occur when getting into bed. Therefore, with repeated pairings, the bed becomes a discriminative stimulus to sleep onset. The goal of stimulus control treatment is to condition the bed and bedroom with sleep. To carry out this task, the following recommendations are made[29]:

1. Only get into bed when sleepy.
2. Get out of bed if unable to sleep (after 20 minutes).
3. Eliminate wake-promoting activities from the bed and bedroom.
4. Wake at the same time each morning.
5. Avoid napping.

Several studies have shown stimulus control to be an effective treatment for insomnia. Therefore, the American Academy of Sleep Medicine (AASM) considers this part of CBT-I to be a "standard" therapy, which is the Academy's highest recommendation.[26] As a result, elements of stimulus control are often used in the successful treatment of insomnia.

Cognitive Therapy

Cognitive therapy for insomnia focuses on irrational beliefs about sleep, unrealistic expectations, catastrophizing, and over valuing the need for sleep.[30] Those with insomnia can enter a negative thought loop where difficulty sleeping initiates a cascade of counterproductive thoughts about the insomnia. For example, while lying in bed not sleeping, one may begin looking at the clock to calculate how much sleep is possible before their required morning wake time. Feeling that the remaining sleep time is insufficient, even if they fell asleep at once, they may ruminate about being unable to perform at work, resulting in job loss, or they may worry about developing a catastrophic disease due to inadequate sleep. This downward spiral of thoughts decreases the likelihood that they will fall back to sleep and further worsens the insomnia.

Therefore, the focus of cognitive therapy is to break the cycle of negative thoughts and restructure thought processes to have more realistic expectations about sleep. To help control presleep rumination, the use of a worry list in conjunction with thought stopping may be helpful. A worry list (otherwise known as a Pennebaker writing task[31]) is a list constructed by the patient 1 to 2 hours before sleep. This list has all the issues the patient is worried about and how they intend to address these concerns the following day.[32] Issues that cannot be addressed will be deferred until the next day. Once the list has been written, the patient is instructed to avoid thinking about problems on the list. If they find themselves ruminating about an item on the worry list, they are advised to stop the thought at once and begin thinking about another

topic. Of note, the empirical data on the benefit of this approach are mixed; Harvey and Ferral[33] found it to significantly improve sleep onset latency, but in a more recent study this finding was not replicated.[34]

One of the more fascinating cognitive interventions for insomnia is called paradoxic intention (this method is often listed separately from cognitive therapy, but is essentially a specific type of cognitive approach). This strategy requires the patient to avoid trying to sleep and instead remain in bed awake as long as possible. The patient is advised to avoid extreme or painful techniques to stay awake, and instead simply lie in bed calmly with their eyes closed. By reducing the anxiety associated with trying to sleep, paradoxic intention has been shown to reduce sleep latency and improve overall sleep quality.[35]

Relaxation-Based Treatments

Relaxation-based methods to help improve sleep quality have also been investigated. These techniques focus on tension as the source of the insomnia. One of the most common relaxation therapies that focuses on somatic tension is progressive muscle relaxation. To use this method, patients are instructed to begin at the head, and systematically tense then relax each muscle group until they have reached their toes. Although effective in some patient populations, progressive muscle relaxation has been shown to worsen insomnia in those who do not report somatic tension.[36]

When rumination occurs during sleep, and a worry list with thought stopping is ineffective, creative visualization can be helpful. This process involves having the patient create a story that they visualize during sleep. The story can involve anything the patient finds relaxing, but must follow a few simple rules:

1. The story cannot include any other people.
2. It must be as detailed as possible.
3. The plot needs to have a beginning, middle, and end.

Visualizing this story when awake at night allows the patient to replace anxiety-producing thoughts with more relaxing mentations. This can hasten sleep onset.

Sleep Restriction Therapy

SRT, developed by Art Spielman and colleagues,[37] reduces TIB to match average sleep time, to improve nighttime sleep quality. To carry out this task, a patient is instructed to complete a sleep log (**Fig. 2**) each morning upon awakening. On this log, bedtime, periods of sleep, and time out of bed are documented typically for a period of at least 2 weeks. Average nightly TST is calculated from this log, and the patient is then asked to limit TIB to the TST from the previous 2 weeks. Once sleep quality improves, if sleepiness remains, TIB is slowly increased until daytime alertness improves.

When the proper sleep period is chosen in SRT, the result is an increase in homeostatic sleep drive. Over time, this increases the likelihood of sleep occurring during the prescribed sleep period. Although considered a guideline treatment for insomnia (1 step lower than a standard treatment) by AASM,[26] many consider SRT to be one of the most effective elements of CBT-I,[38] if not the most effective part. The reason this aspect of CBT-I is so effective, is because when performed correctly, SRT includes principal elements of stimulus control and cognitive therapy and helps align circadian rhythms. This statement may be surprising to some, but let us consider practical elements of implementing SRT.

After having a patient complete sleep logs and selecting a new sleep-wake schedule, the patient is advised to maintain a limited TIB with a fixed sleep and

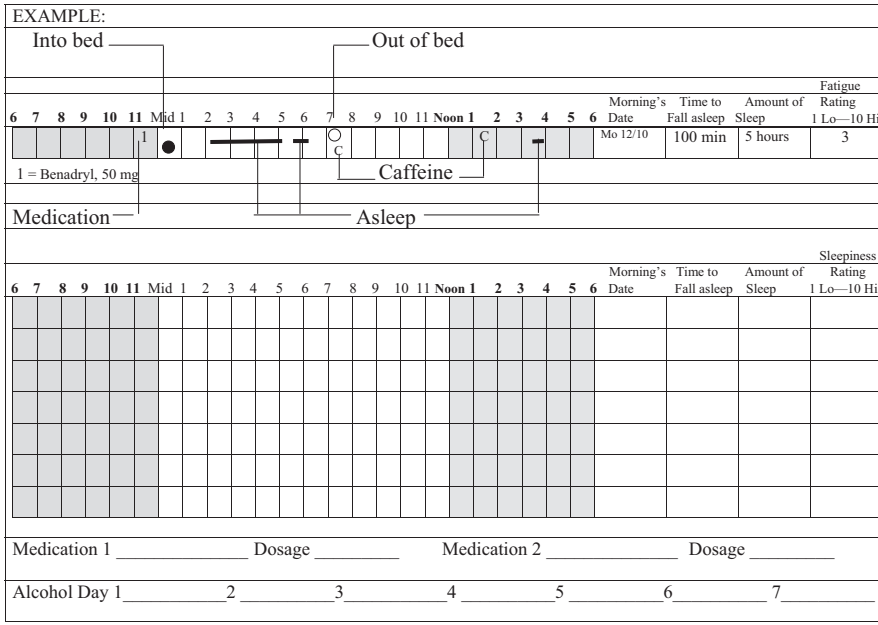

Fig. 2. A version of the City College of New York sleep log. Patients are instructed to complete this each morning. The black dot indicates the time the patient got into bed, and the black lines represent periods of sleep. The black circle shows time out of bed. The numeral before the black dot indicates when medications were taken (if any were taken before bedtime). Medications taken at other times of the day are listed below the chart. c indicates time of caffeine consumption. Daily alcohol consumption for each day is listed below medications. (*From* Ebben MR, Spielman AJ. Non-pharmacological treatments for insomnia. J Behav Med. 2009;32(3):244-254; with permission.)

wake time. Moreover, patients are asked to avoid napping. Therefore, 2 elements of stimulus control, avoiding napping and waking up at the same time each day, are part of the SRT instructions. In addition, keeping a fixed TIB helps to entrain circadian rhythms over time. The cognitive aspects of SRT are required to convince patients to follow the restricted schedule. Patients often complain about the risks of reducing TIB and the resulting sleep loss, and how this may negatively affect their long-term health and daytime functioning. This allows the practitioner an opportunity to discuss realistic expectations about sleep, and address catastrophizing and overvaluing the need for sleep.

Mindfulness-Based Interventions

Mindfulness is the primary focus of Buddhist psychology, which teaches patients nonjudgmental awareness. In the field of behavioral sleep medicine, mindfulness-based stress reduction (MBSR) has received growing attention in the literature.[39] The MBSR program teaches meditation, breathing techniques, and body scanning as tools of self-awareness and is often taught in a group setting. This technique can be helpful in patients who tend to catastrophize when unable to sleep. Teaching acceptance of the act of not sleeping can help to reduce anxiety during awakenings at night, or when having difficulty falling asleep. Often MBSR is combined with behavioral techniques such as SRT to improve efficacy.

Sleep Hygiene Education

The term sleep hygiene was originally coined by Peter Hauri[40] to describe a collection of behaviors that should be either avoided or adopted to improve sleep quality. Although these recommendations vary based on the source, they tend to have excellent face validity. As a result, over time they have become imprinted in the consciousness of the general public as the path to superior quality sleep. Unfortunately, sleep hygiene has not been consistently shown to be an effective standalone therapy for insomnia and is therefore not recognized by AASM as a validated treatment technique.[26] Regrettably, many people with insomnia conflate sleep hygiene with effective CBT-I methods. Consequently, after rigidly adhering to sleep hygiene and seeing no appreciable benefit, some individuals become disenchanted with nonpharmacologic interventions for insomnia before even trying them.

Considerations for Treating Insomnia in Older Populations

In older adults, often the onset of insomnia is more gradual in nature. This is due to lifestyle changes associated with retirement (or reduced work hours) and a decrease in social and/or physical activities. Frequently reduced daytime activities and a more flexible schedule result in increased TIB. For those who kept a chronic partial sleep-deprived state during their working years, the increased TIB may initially result in increased TST and feeling more alert during the day. However, once their sleep debt has been discharged, the added TIB results in a fragmentation of sleep over the longer period in bed. When insomnia develops in this fashion, the precipitating event is the change in lifestyle and not an acute increase in stress due to a temporary event. As a result, when conceptualized in the framework of the 3-P model, both the precipitating and perpetuating factors are one and the same.

There is also evidence that the homeostatic sleep drive changes with age. Studies have shown that when compared with younger individuals, homeostatic drive in response to sleep deprivation is reduced in elderly individuals, particularly in the frontal regions of the brain.[41] Moreover, the discharge of SWA becomes less pronounced with age,[3] suggesting that older patients have different treatment needs than younger individuals. Meta-analytic studies have shown CBT-I to be effective in elderly individuals.[42,43] In addition, when investigated alone, SRT has been shown to be an effective treatment method in older patients, and may be more effective than a purely cognitive approach.[43] This suggests that older patients may benefit more from treatment techniques that strengthen the homeostatic drive.

As mentioned in the introduction, beginning in the late teens to early 20s, circadian rhythms shift from a more delayed to a more advanced phase.[12] In some, the advance is so pronounced that it interferes with social activities in the evening and can result in persistent early morning awakenings. This condition is referred to as advanced sleep phase syndrome, and is more common in older patients.[44] This may be due to a combination of changes in the eye that occur with age, which can lead to less light intake,[45] and a reduction in both period length and amplitude of the circadian rhythm.[46] Systematic exposure to bright light at the proper circadian time, a treatment approach called bright light therapy, can help treat this condition.[47] A recent study in institutionalized elderly patients found that bright light exposure in the evening significantly improved sleep quality, cognitive function, and delayed circadian rhythms.[48]

Using Melatonin in Elderly Patients

One of the most common over-the-counter sleep aids is melatonin. Often, this medication is taken at bedtime in the hopes that it will hasten the onset of sleep, or,

particularly in elderly individuals, delay terminal awakenings in the morning. However, the use of melatonin in aged patients presents unique problems. To understand why melatonin is problematic in older patients, we need to appreciate the phase response curve of melatonin in humans. A phase response curve is a term used in the field of circadian rhythm research to describe the change in circadian phase caused by a *zeitgeber*. We have already described the importance of bright light exposure in humans, which is a powerful *zeitgeber*.[9,10] However, exogenous melatonin can also act as a phase shifting agent in humans.[49] Whereas bright light exposure in the evening causes a delay of circadian rhythms, taking melatonin in the evening causes an advance.[50] Therefore, taking melatonin at bedtime can worsen early morning awakenings, which is a frequent problem in elderly patients, because of a tendency of the circadian rhythm to advance with age.[12,14] Therefore, melatonin should be used with caution in older patients and not for early morning awakenings.

SUMMARY

As we age, homeostatic sleep drive becomes weaker, and dissipates at a slower rate over the course of the night. Stage 1 sleep is increased as is sleep latency and wake after sleep onset, resulting in lighter more fragmented sleep. In addition, lifestyle changes that often result in a more flexible daytime schedule due to retirement, either resulting in or combined with circadian phase advances cause a significant decrease in overall sleep quality. CBT-I has been shown to improve insomnia in elderly individuals and should be used to blunt the effects of aging on sleep, particularly elements of therapy that focus on increasing homeostatic sleep drive, such as SRT. In addition, bright light therapy, when used in conjunction with CBT-I can help delay the circadian phase, which helps entrain sleep to occur at a more socially acceptable time.

CLINICS CARE POINTS

- Successful treatment of insomnia should focus on increasing sleep drive to overcome hyperarousal and recondition the patient to sleep in their bedroom environment.
- Using Sleep Restriction Therapy in conjunction with Stimulus Control and Cognitive Therapy is an effective way to increase sleep drive and recondition the patient to associate the bed with sleep.
- It is important to understand the circadian phase of each patient to ensure that the correct treatment approach is utilized.

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