Cognitive-Behavioral Approaches to the Treatment of Insomnia

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Insomnia is a pervasive condition with various causes, manifestations, and health consequences. Regardless of the initial cause or event that precipitates insomnia, it is perpetuated into a chronic condition through learned behaviors and cognitions that foster sleeplessness. This article reviews the rationale and objectives of cognitive-behavioral therapy (CBT), a safe and effective treatment for insomnia that may be used to augment hypnotic drugs or as a monotherapy. Cognitive-behavioral management of insomnia includes 3 components—behavioral, cognitive, and educational modules—and is usually presented in a group or individual therapy setting. Each treatment procedure is detailed herein, and recommendations for implementation are given. The evidence supporting this behavioral approach shows that CBT is effective for 70% to 80% of patients and that it can significantly reduce several measures of insomnia, including sleep-onset latency and wake-after-sleep onset. Aside from the clinically measurable changes, this therapy system enables many patients to regain a feeling of control over their sleep, thereby reducing the emotional distress that sleep disturbances cause. Some clinical and practical issues that often arise when implementing this therapeutic approach for insomnia are also discussed. *(J Clin Psychiatry 2004;65[suppl 16]:33–40)*

Insomnia is a prevalent and costly health problem, both as a symptom and as a syndrome. In the general population, approximately 10% to 15% of people report chronic insomnia,[1–3] while 25% to 35% of people experience occasional or mild insomnia.[2,4] These rates are even higher among women and older patients.[3,5] In a 2001 National Sleep Foundation survey,[6] about half (51%) of the American adults polled experienced 1 or more symptoms of insomnia a few nights per week over the course of the year. Although few people seek treatment specifically for insomnia, 25% to 35% of patients seen in general practice report moderate or severe insomnia,[7] and the rate of regular pharmacologic hypnotic use ranges from 4% in the United States[2,6] to 11.7% in France.[8] Insomnia remains one of the most unrecognized and undertreated health conditions. Those who experience persistent insomnia tend to have greater medical and psychiatric morbidity[1,9] and a reduced quality of life, higher absenteeism, impaired job performance, higher health care utilization, and inferior general functioning and well-being compared with individuals without insomnia.[10,11] One report[12] estimated the total annual cost of insomnia to be between $92.5 and $107.5 billion, including medical and psychiatric treatment and medications, lost productivity, and costs associated with accidents and increased alcohol consumption resulting from insomnia.

In addition to the pharmacologic treatments available for insomnia, several nonpharmacologic and psychological treatments have shown efficacy in reducing insomnia.[13,14] Cognitive-behavioral therapy (CBT) provides a safe and effective treatment for insomnia, both as a monotherapy and as an augmentation of hypnotic drugs. CBT includes 3 components—behavioral, cognitive, and educational—that can be used to help patients modify the learned behaviors and cognitions that propagate sleep disturbances. This article provides a brief description of CBT and summarizes the research on CBT for insomnia. Clinical and practical issues involved in the implementation of this insomnia management system are also discussed. CBT, when administered correctly, has the potential to alleviate various forms of insomnia and, more importantly, to help patients understand and eliminate probable causes for this condition.

CONCEPTUAL MODEL OF INSOMNIA

Insomnia comprises various manifestations of sleep disturbance and has numerous origins. For some patients, insomnia consists of inadequate sleep duration at night, while others experience poor sleep efficiency (total sleep time divided by the total time in bed) or poor sleep quality.

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Table 1. Types of Primary Insomnia[a]

Type of Insomnia	Description
Psychophysiological	Evidence of conditioned sleep-preventing patterns Confirmation of increased body tension No indication that sleep disorder is caused by another medical or psychiatric disorder
Subjective (sleep state misperception)	Subjective complaint of insomnia is not corroborated by polysomnography Also called paradoxical insomnia May represent the transition between normal sleep and psychophysiological insomnia[b]
Idiopathic (childhood onset)	Lifelong inability to obtain adequate sleep Does not include temporal fluctuations seen in psychophysiological insomnia Patients may minimize the impact as they have adapted to the sleep loss, despite being the most objectively sleep impaired

[a]Based on Morin.[13]
[b]Based on Salin-Pascual et al.[17]

Table 2. Secondary Insomnia: Causes and Characteristics[a]

Causes of Insomnia	Description
Psychiatric disorders	Anxiety disorders Depression Bipolar disorder (manic phase) Dementia
Medical conditions	May be a symptom of medical conditions, including: Acute or chronic pain (back pain, arthritis, osteoporosis, headaches, cancer, musculoskeletal pain) Congestive heart failure Chronic pulmonary diseases Endocrine and gastrointestinal diseases Central nervous system disorders Alzheimer's disease Head or brain injury May be a side effect of medications or procedures used to treat medical conditions
Alcohol or drug dependence	Insomnia associated with substance (e.g., alcohol, cannabis) abuse or dependence. The substance may be used initially as a sleep-promoting agent
Other sleep disorders	Restless legs syndrome/periodic limb movements, sleep apnea, parasomnia, circadian rhythm sleep disorders

[a]Based on Morin[13] and Morin and Espie.[18]

Complaints from patients with insomnia include difficulty falling or staying asleep during the night, an inability to fall asleep again after nighttime arousals, and awaking too early in the morning.

Insomnia may be acute or chronic and primary or secondary.[14] Acute insomnia usually lasts less than 1 month and is often directly attributable to a known cause such as jet lag, medication, or poor sleep environment.[15] Insomnia that lasts between 1 and 6 months is considered short-term or subacute. Chronic insomnia generally lasts more than 6 months and may be recurrent over many years. Primary, or intrinsic, insomnia[16] is a condition autonomous from other disorders or conditions and may be psychophysiological, subjective, or idiopathic (Table 1).[13,17] Secondary, or extrinsic, insomnia is caused by other medical or psychiatric disorders, alcohol or drug dependence, sleep-induced respiratory disorders, movement disorders, circadian rhythm sleep disorders, environmental factors, or parasomnias (Table 2).[13,18] Most cases of chronic primary insomnia, the condition most discussed here, develop in response to a major life event or other stressor but continue after the patient has adapted to the stress or it has abated; the patient has become conditioned not to sleep, although the initial reason for wakefulness may be long past.[18]

The clinical significance of any case of insomnia is determined by the effects of the sleep loss on daily cognitive, physical, psychological, and social functioning. As defined by the DSM-IV-TR,[16] primary insomnia must be accompanied by daytime fatigue or other impairments in social and occupational functioning or cause significant emotional distress. While the other 2 classifications of sleep disorders (the *International Classification of Sleep Disorders*[15] and the 10th edition of the *International Classification of Diseases*[19]) differ slightly, the 3 classifications converge on a general definition of chronic, primary insomnia as a sleep disorder in which a patient subjectively complains of difficulties initiating or maintaining sleep 3 or more nights a week for 6 months or longer and, as a result of this sleep disturbance, experiences daytime fatigue, disturbed mood, and/or impaired function and marked emotional distress.

CURRENT TREATMENT PRACTICES AND BARRIERS TO TREATMENT

Despite the existence of several treatment options for insomnia, people who experience sleep difficulties usually start with passive strategies. When surveyed,[6] many insomnia patients reported that they read, watch television, or listen to the radio when they are unable to sleep. Many people with insomnia say that they "do nothing" except lie in bed, tossing and turning, waiting for sleep to come. The next step for most, the first level of active treatment, is self-medication with alcohol, over-the-counter sleep aids, or natural/herbal dietary supplements. If people with insomnia eventually seek medical help for their condition, they are generally prescribed hypnotic drugs as a second level of active treatment. The third level, and the focus of this article, includes nonpharmacologic treatments such as behavioral and psychological interventions.[13,18]

Several significant barriers to the treatment of insomnia exist, the first of which is the difficulty in identifying it. Insomnia often goes unrecognized and untreated due to several factors. Primarily, the impact of insomnia as a health problem is not acutely visible or well defined. Often, insomnia is not considered a high priority by health care providers, and many people with insomnia may be reluctant to seek treatment. Patients may feel that nothing can be done to alleviate their insomnia, so they do not re-

port the condition; others feel as if insomnia is not a legitimate health concern. For some people, admitting to sleep difficulties is equivalent to admitting a loss of control, so they may be unwilling to acknowledge and face the problem.

Pharmacotherapy, the most common therapy, is sometimes inadvisable as a monotherapy or is contraindicated. Also, patients may have various reasons for choosing not to use hypnotics.[20] For some patients, hypnotic drugs do not alleviate their insomnia. For others, pharmacotherapy may provide some initial relief but gradually loses efficacy. Hypnotics may be contraindicated by the use of other medications, existing medical conditions, or a patient's high susceptibility to substance abuse or addiction. In some situations, a patient is just not comfortable taking hypnotic drugs, or a physician is uncomfortable prescribing them.

Despite the significant advances in the CBT management of insomnia, the preference for nonpharmacologic treatments expressed by some patients, and evidence of its efficacy, CBT continues to be underutilized. Many health care providers are not familiar with the cognitive and behavioral interventions beneficial in treating insomnia. Even when this treatment is understood, barriers to its widespread use arise. CBT can be time consuming, for both patients and clinicians, as several counseling sessions are usually needed for the therapy to be effective. Nevertheless, CBT constitutes an important therapy option that can provide relief from chronic primary insomnia (and other insomnias) for patients who may not find relief by other methods.

COGNITIVE-BEHAVIORAL THERAPY FOR INSOMNIA

Objectives and Targets of Cognitive-Behavioral Therapy

CBT targets factors that perpetuate insomnia over time.[13,14,18,21] These factors specifically include excessive amounts of time spent in bed, routine daily napping, irregular sleep-wake schedules, excessive worry over sleep loss and rumination about the daytime consequences, and performance anxiety.

The first objective of CBT is to modify poor sleep habits. Since people with chronic insomnia often not only maintain maladaptive sleep habits but also engage in fairly irregular sleep schedules, the second objective is to regulate sleep-wake schedules. Third, faulty beliefs and attitudes about sleep must be corrected. Because autonomic arousal can be a significant factor in insomnia, CBT also attempts to reduce physiologic, cognitive, and emotional hyperarousal. Another important goal is to help the patient develop adaptive coping skills. Few cases of chronic insomnia are totally cured by any treatment, and most patients completing any type of therapy will still experience some lingering sleep disturbances. For these disturbances to remain residual and occasional, cognitive restructuring of expectations and beliefs about sleep helps patients to adapt to sleep loss and minimize its effects. This process also advances the final objective of CBT (indeed, of any insomnia treatment)—the prevention of relapse.

Table 3. Cognitive-Behavioral Treatments for Primary Insomnia

Activity	Description
Stimulus control	Instructions designed to reassociate the bed/bedroom with sleep and to reestablish a consistent sleep-wake schedule
Sleep restriction	A method for limiting time in bed to actual sleep time, thereby creating mild sleep deprivation, which results in more consolidated and more efficient sleep
Relaxation training	Methods aimed at reducing somatic tension or intrusive thoughts interfering with sleep
Cognitive therapy	Psychotherapeutic method aimed at changing faulty beliefs and attitudes about sleep and insomnia
Sleep hygiene education	General guidelines about health practices and environmental factors that may affect sleep

Cognitive-Behavioral Interventions

CBT comprises 3 components—behavioral, cognitive, and educational—each of which addresses a different aspect of insomnia (Table 3).

Stimulus control. Although sleep disturbances are usually caused by an initial stressor or illness, true insomnia is generally the result of maladaptive behaviors that continue beyond the original precipitating event. A person with insomnia no longer responds to the typical sleep stimuli with drowsiness and sleep but, instead, associates these stimuli with wakefulness.[22] Stimulus control consists of 5 simple instructions that help the patient reassociate sleep stimuli with the proper behavior—falling asleep—and encourage the establishment of a consistent sleep-wake schedule.

1. Go to bed only when sleepy. People with insomnia may often go to bed early to increase the possibility of being asleep by the desired time; however, extra time spent in bed only heightens arousal by causing more intrusive thoughts, worry, and frustration about the inability to sleep. Tiredness or fatigue should not be confused with sleepiness.

2. Use the bed or bedroom only for sleeping.When the bed or bedroom is used for reading, eating, watching television, or other activities, the setting becomes associated with wakefulness instead of sleep; using this area only for sleep will help to prompt sleepiness. The disassociation of stimulus and response (bed = sleep) may be particularly prevalent in older adults who spend considerable amounts of time in bed due to illness or physical restrictions.

3. Get out of bed when unable to sleep. If the patient is unable to fall asleep after 15 minutes spent in bed, he or she should go into another room and engage in some quiet activity (e.g., reading, watching television, listening to the radio). This instruction also holds true for awakenings in

the middle of the night. Although most people believe that getting out of bed when unable to fall asleep after nocturnal awakenings will cause them to become more aroused, this practice will generally hasten sleep onset and, in the long term, reassociate the bed with rapid sleep onset.

4. Arise at the same time every morning. Waking at a regular time will reestablish synchronized circadian rhythms. Regardless of the amount of sleep people with insomnia get during the night, they should maintain a regular waking time during weekdays and weekends.

5. Do not nap during the day. While napping may seem like a good way to catch up on sleep lost during the night (and may be appropriate for those who suffer from sleep deprivation rather than insomnia), it will only continue the cycle of irregular circadian rhythms and make it difficult for the patient to fall asleep at the desired bedtime.

Sleep restriction. This method limits the amount of time spent in bed to the amount of time spent sleeping.[23] By creating a temporary, mild state of sleep deprivation, the sleep restriction method helps to bring about a faster sleep onset and greater sleep continuity and quality. If someone with insomnia stays in bed for 8 hours per night but is only sleeping for 6 hours each night, he or she is asked to limit the time in bed to 6 hours, his or her "sleep window." This window of time is then altered weekly according to the sleep efficiency being achieved. For the purposes of sleep restriction, the sleep efficiency ([total sleep time/total time in bed] × 100%) goal for an insomnia patient should be around 85%. If sleep efficiency is greater than 90% in any week, the patient is given an additional 15 to 20 minutes of time in bed per night. Weekly sleep efficiency of less than 80% warrants a decrease in the sleep window by 15 to 20 minutes per night. Since daytime drowsiness can be a side effect of sleep restriction, the sleep window should not be reduced below 5 hours per night, regardless of sleep efficiency. While this drowsiness is normal and temporary, especially in the first week of sleep restriction, allowances must be made for those whose jobs require operation of motor vehicles or heavy machinery or entail other duties in which drowsiness may be a danger to the patient or to others.

Relaxation. The most commonly used nonpharmacologic therapy for insomnia, relaxation seeks to reduce 2 types of arousal that interfere with sleep—autonomic and cognitive.[14] Autonomic relaxation techniques, such as progressive muscle relaxation, autogenic training, and biofeedback, target muscle tension and other types of physical arousal. Imagery training, meditation, and thought stopping are a few of the methods used to counteract cognitive arousal. Relaxation therapy requires training and daily practice, so the patient should not expect immediate results.

Cognitive therapy. While maladaptive behaviors perpetuate insomnia, faulty beliefs and unrealistic expectations about sleep and insomnia bolster these behaviors. Cognitive therapy targets these erroneous beliefs and attempts to alter them.[24] First, the dysfunctional cognition is identified, which reveals the faulty underlying belief. Then, the clinician offers alternative interpretations, and the patient can begin to think about his or her insomnia in a different way. There are 5 main targets of cognitive therapy.

1. Misconceptions about causes of insomnia. Many patients attribute their insomnia to specific causes or precipitating factors, such as pain, allergies, age, or depression. The patient then believes that these conditions must be resolved before the insomnia will abate. Although such factors are often involved in sleep disturbances, attribution of insomnia to those factors alone is self-defeating because the patient may indeed have little control over them. Cognitive therapy shows the patient that chronic insomnia always involves behavioral and psychological factors over which the patient can exercise some control. By understanding that insomnia is not caused solely by external causes, the patient may then learn how to overcome or adapt to it.

2. Misattribution and amplification of consequences. While some patients may experience real daytime consequences of insomnia, the fear of these consequences and concerns about the health effects of sleep loss magnify the subjective report of these consequences and amplify the calamitous nature of insomnia. Tiredness, irritability, and inefficiency may be subjectively attributed to a poor night's sleep,[25] but objective measurements of daytime sleepiness and lack of vigilance in patients with insomnia are quite limited.[26–28] Reports of adverse effects are higher among worry-prone individuals.[29] In fact, people with subjective insomnia exhibit more daytime sequelae of adverse effects than those with psychophysiological insomnia, who have objective evidence of disturbed sleep.[28] The worry-prone personality of many patients with subjective insomnia may be the cause of this phenomenon, or it may confirm the tendency of psychophysiological insomnia patients toward physiologic hyperarousal, during both the night and the day.[27] In either case, a rational look at the objective consequences of sleep loss can help patients reattribute the presumed consequences of sleep loss and see that worry about insomnia may be more detrimental to their health than the actual sleep loss.

3. Unrealistic sleep expectations. Everyone sleeps differently. People with insomnia may become overly focused on achieving some "normal" sleep duration, onset, or quality by comparing their sleep patterns with those of friends or bed partners. While 8 hours may be the average sleep time for most adults, the self-imposed pressure of reaching this sleep duration may worsen sleep difficulties or even cause people without definable sleep disturbances to believe they have insomnia. In these cases, baseline clinical assessment of sleep parameters will

reveal the severity and significance of the sleep disturbance and determine whether it is pathologic (sleep-onset latency or wake time after sleep onset > 30 minutes; total sleep time < 6.5 hours)[13,18] or within normative ranges. For those patients with unrealistic expectations, cognitive therapy helps them change their expectations about sleep and reappraise the clinical importance of their sleep difficulties.

4. Performance anxiety and learned helplessness. For many people, insomnia constitutes a lack of control. The variability of sleep patterns from night to night only magnifies the representation of sleep as unpredictable and out of control. As the feeling of powerlessness increases, patients place more pressure on themselves to fall asleep, creating a sense of performance anxiety that can make matters worse. Cognitive strategies for this situation may include paradoxical recommendations (e.g., "Try not to fall asleep"), which increase the patient's perception of control. When insomnia is unpredictable and seemingly without cause, patients may fall into a state of learned helplessness, believing that hypnotics are the only source of predictability or that there will never be any control or predictability. Then, patients begin to position themselves as victims and catastrophize the power, effect, and duration of insomnia.

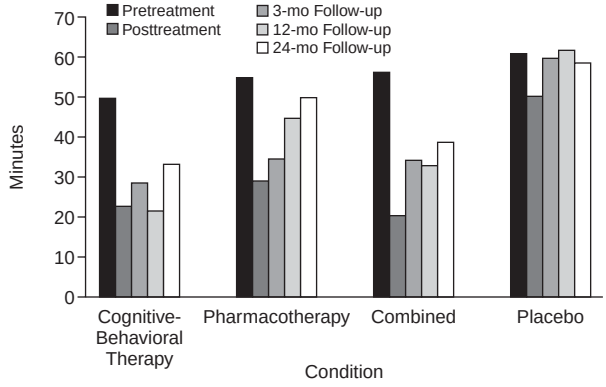
5. Faulty beliefs about sleep-promoting practices. Preconceived ideas about solutions for insomnia may only worsen the case. The counterproductive tactics most commonly practiced by people who are unable to fall asleep are to stay in bed and try harder or to sleep in later in the morning to compensate for poor sleep. Most erroneous practices are altered through the behavioral component of therapy, but patients also need to cognitively restructure the way they think about sleep promotion.

In general, cognitive therapy for insomnia helps to change the underlying ideas that perpetuate insomnia. People with insomnia should learn 6 basic cognitive strategies: (1) keep realistic expectations; (2) do not blame insomnia for all impairments; (3) never try to sleep; (4) do not give too much importance to sleep; (5) do not catastrophize after a poor night's sleep; and (6) develop tolerance to the effects of insomnia.

Sleep hygiene education. External factors can play a large role in sleep disturbances, and sleep hygiene education teaches insomnia patients to recognize and minimize these factors:

- Avoid caffeine and all stimulants after dinner.
- Avoid smoking near bedtime and upon night wakings.
- Do not drink alcohol in the late evening.
- Do not exercise too close to bedtime; regular exercise in the late afternoon or early evening may deepen sleep.
- Minimize noise, light, and excessive temperatures.

Figure 1. Reduction of Wake Time After Sleep Onset[a]



[a]Reprinted with permission from Morin et al.[33]

EFFICACY OF COGNITIVE-BEHAVIORAL THERAPY FOR INSOMNIA

CBT produces reliable clinical effects on several sleep and insomnia parameters. Studies[14,30–34] show an average symptom reduction of 50% to 60% on the main outcomes of sleep latency (measured in minutes until sleep onset) and wake-after-sleep onset (measured in minutes awake after sleep onset). After CBT, sleep-onset latency and wake-after-sleep onset are generally reduced below or near 30 minutes, the criterion typically used for defining sleep onset and sleep maintenance insomnia. These studies[14,30–34] have also shown that total sleep time after behavioral treatment increases by an average of 30 to 45 minutes, and sleep efficiency improves to approximately 85%.[33,34] In a study[35] of interventions used for insomnia secondary to chronic pain, CBT resulted in improvements in self-reported sleep-onset latency, wake-after-sleep onset, sleep efficiency, and sleep quality and maintenance of improvement at 3-month follow-up, confirming that CBT is also valuable in treating secondary insomnia.

These improvements are at least comparable to those shown by patients using pharmacotherapy alone.[33,34] Pharmacotherapy, which might include benzodiazepines, nonbenzodiazepine hypnotics, or antidepressants, is the most common method for management of insomnia.[5,11] Numerous studies[36,37] show pharmacotherapy to be effective in sleep promotion, but there are questions as to its long-term efficacy in treating chronic insomnia. Clinical comparison of pharmacotherapy and CBT[30] shows that drugs produce equivalent improvements but that the improvements from behavioral therapy are better sustained over time. In a recent meta-analysis[34] comparing the sleep benefits of behavioral therapy and pharmacotherapy, although greater sleep duration was achieved with pharmacotherapy, CBT showed greater improvement in all other measures, including self-reported sleep quality.

Figure 2. Sample Sleep Diary[a]

Name:								
Week: _____ to _____	Example	Mon	Tues	Wed	Thurs	Fri	Sat	Sun
1. I napped from ___ to ___ (note the times of all naps).	2:00 to 2:45 p.m.							
2. I took ___ of ___ medication and/or ___oz of alcohol as a sleep aid.	5 mg Ambien							
3. I went to bed at ___ and turned out the lights at ___.	10:30 p.m. 11:10 p.m.							
4. After turning the lights out, I fell asleep in ___ minutes.	45							
5. My sleep was interrupted ___ times (specify number of nighttime awakenings).	3							
6. My sleep was interrupted for ___ minutes (specify duration of each awakening).	20 30 10							
7. I woke up at ___ (note time of last awakening).	6:15 a.m.							
8. I got out of bed at ___.	6:40 a.m.							
9. When I got up this morning, I felt ___. (1 = exhausted, 5 = refreshed)	2							
10. Overall, my sleep last night was ___. (1 = very restless, 5 = very sound)	1							

[a]Reprinted with permission from Morin.[13]

Of the cognitive-behavioral interventions discussed, a meta-analysis of the literature[14] shows stimulus control to be the most effective single therapy. While sleep restriction actually shows greater benefits, too few clinical trials have been conducted to date to confirm its superiority. Rates of effectiveness for nonpharmacologic interventions among insomniacs are promising, with 70% to 80% of patients benefiting from treatment.[14,30] Beyond the objectively measured improvements attributed to CBT, patients subjectively report an improved experience of sleep. They regain a sense of control over their sleep and experience a reduction in the emotional stress caused by their insomnia.

The combined therapy approach (pharmacotherapy and CBT) may produce greater sleep improvement than either therapy alone. In a clinical trial,[33] the percentage reductions of wake time after sleep onset were greatest with the combined therapy (63.5%), followed by CBT (55%), pharmacotherapy (46.5%), and placebo (16.9%) (Figure 1). Long-term improvement was sustained with CBT, while pharmacotherapy alone did not provide long-term efficacy. Combined treatment showed variable long-term outcomes. Although these results show potential for successful combined therapy insomnia management, more research is needed. As some patients may eventually discontinue their use of hypnotics when they pursue a CBT approach, their clinicians should play a role in the withdrawal process. The decision to discontinue medication will depend on the hypnotic use pattern, insomnia severity, and patient characteristics, and the withdrawal program should be structured to fit individual patients.

CLINICAL AND PRACTICAL ISSUES IN GENERAL PRACTICE

Indications for Cognitive-Behavioral Therapy

CBT is indicated, principally, for persistent, primary insomnia. It is also proving effective as an augmentation therapy for insomnia secondary to other medical or psychiatric disorders.[38] The CBT program (or individual behavioral, cognitive, or educational components) may be used in combination with hypnotics. Although most efficacious as a 3-pronged management system, the components also may be used individually when time or resources preclude the administration of the whole process. However, sleep restriction should not be used with patients presenting with bipolar disorder or excessive daytime sleepiness; caution is advised when using sleep restriction with patients operating motor vehicles or heavy machinery and those for whom vigilance cannot be compromised.

Initial Evaluation of Insomnia

Before CBT management of insomnia may begin, the patient should participate in a clinical interview to assess

Table 4. Essential Features of Cognitive-Behavioral Therapy Protocol[a]

Evaluation	Conduct a detailed sleep history addressing:
	Nature of sleep difficulties (initial, middle, late insomnia)
	Typical sleep schedule (bedtime, arising time)
	Onset, duration, and course of insomnia
	Exacerbating/facilitating factors
	Medical, psychiatric, and substance use factors
	Use of sleep-promoting medication
	Prior therapies and outcomes
	Obtain baseline sleep diary monitoring and psychological screening
Treatment	Explain the nature and rationale of behavioral treatment
	Provide a conceptual framework in which to explain insomnia in the context of the patient's historical variables
	Discuss the patient's expectations and provide realistic outcome expectations
	Introduce sleep restriction and prescribe an initial "sleep window" based on baseline diary data
	Introduce stimulus control procedures, emphasizing the need to implement all instructions in a systematic fashion
	Examine and assist patients in revising unrealistic sleep expectations, misattributions of insomnia causes, and misconceptions of daytime consequences
	Consider relaxation training
	Review basic sleep hygiene principles
	Conduct periodic follow-ups to address compliance issues and monitor treatment progress
	Continue daily sleep diary monitoring throughout treatment and use these data to evaluate insomnia severity, monitor treatment progress, and involve the patient in his or her treatment

[a]Based on Morin.[39]

the scope, severity, and characteristics of his or her insomnia and the personal characteristics that may affect treatment. A thorough evaluation will include a sleep history, medical history (including medication use), review of general health concerns (e.g., diet, smoking, exercise), and screening for other sleep disorders. The sleep history should focus on the nature of the complaint (e.g., sleep-onset latency, early morning awakenings, poor quality of sleep), the patient's sleep-wake schedule, the daytime sequelae of insomnia, and the natural history of the insomnia. It is also important to inquire into the patient's prebedtime routine, his or her typical responses to sleep difficulties, and secondary gains that the patient may get from his or her insomnia (e.g., sympathy from friends or family, time off work), which are all potential factors in the perpetuation of insomnia.[13,18]

Self-report sleep diaries play a vital role in the assessment and treatment of insomnia, providing valuable information to both the patient and physician (Figure 2). Patients should complete at least 1 week of sleep diaries—recording each night's bedtime, arising time, sleep-onset latency, number and duration of nighttime awakenings, time of last awakening, naps, medication intake, and some measurement of sleep quality[18]—before therapy begins and should continue the diaries throughout treatment. While polysomnography may be useful to determine actual physiologic measures, sleep diaries are an economical and simple way to track patients' sleep patterns. Polysomnography is indicated only when there are symptoms of other sleep disorders (e.g., sleep apnea, periodic limb movements, narcolepsy), the presenting complaint is excessive daytime sleepiness, or the patient with insomnia is unresponsive to treatment.

Therapy Format and Content

CBT for insomnia may be administered in an individual or group setting. The session content and protocol are basically the same for both formats. Group therapy is advantageous in that it is cost-effective and patients are able to support each other in confronting their sleep difficulties and adhering to treatment. The typical treatment protocol lasts an average of 5 hours, spread over 4 to 6 weekly consultation sessions. The specific number of consultation sessions is likely to vary as a function of several factors, including the insomnia severity, presence of comorbid medical and psychiatric disorders, and the patient's motivation. Treatment sessions focus on the different components of CBT (Table 4).[39] While each session differs in specific content, all sessions center around the following main activities: assessing progress from the last week and reviewing sleep diaries, ascertaining problems in home practice and levels of adherence to treatment, negotiating plans for better adherence, launching the new treatment component and explaining its rationale, and distributing self-help educational material.

CONCLUSION

CBT offers a safe and effective alternative to pharmacologic treatment of insomnia and has also shown success as an augmentation of hypnotic drug treatment. CBT has specific limitations—mainly that it is little known, is not widely available, and is more time-consuming than pharmacotherapy. Nevertheless, CBT shows promise as a valuable method that could be used more widely and to greater effectiveness in clinical settings to reduce various types, severities, and manifestations of insomnia. More research is vital to clarify the full clinical and economic implications of CBT and to determine the true potential of this emerging treatment system.

Drug name: zolpidem (Ambien).

Disclosure of off-label usage: The author has determined that, to the best of his knowledge, no investigational information about pharmaceutical agents has been presented in this article that is outside U.S. Food and Drug Administration–approved labeling.

REFERENCES

1. Ford DE, Kamerow DB. Epidemiologic study of sleep disturbances and psychiatric disorders: an opportunity for prevention? JAMA

1989;262:1479–1484
2. Mellinger GD, Balter MB, Uhlenhuth EH. Insomnia and its treatment: prevalence and correlates. Arch Gen Psychiatry 1985;42:225–232
3. Ohayon M. Epidemiology of insomnia: what we know and what we still need to learn. Sleep Med Rev 2002;6:97–111
4. Ancoli-Israel S, Roth T. Characteristics of insomnia in the United States: results of the 1991 National Sleep Foundation Survey. Sleep 1999; 22(suppl 2):S347–S353
5. Ohayon M. Epidemiological study on insomnia in the general population. Sleep 1996;19(suppl 3):7–15
6. National Sleep Foundation. 2001 Sleep in America Poll. Available at: http://www.sleepfoundation.org/publications/2001poll.cfm. Accessed June 23, 2004
7. Hohagen F, Rink K, Kappler C, et al. Prevalence and treatment of insomnia in general practice: a longitudinal study. Eur Arch Psychiatry Clin Neurosci 1993;242:329–336
8. Ohayon MM, Caulet M. Psychotropic medication and insomnia complaints in two epidemiological studies. Can J Psychiatry 1996; 41:457–464
9. Foley DJ, Monjan AA, Brown SL, et al. Sleep complaints among elderly persons: an epidemiologic study of three communities. Sleep 1995;18: 425–432
10. Kuppermann M, Lubeck DP, Mazonson PD, et al. Sleep problems and their correlates in a working population. J Gen Intern Med 1995;10:25–32
11. Simon GE, VonKorff M. Prevalence, burden, and treatment of insomnia in primary care. Am J Psychiatry 1997;154:1417–1423
12. Stoller MK. Economic effects of insomnia. Clin Ther 1994;16:873–897
13. Morin CM. Insomnia: Psychological Assessment and Management. New York, NY: The Guilford Press; 1993
14. Morin CM, Culbert JP, Schwartz MS. Nonpharmacological interventions for insomnia: a meta-analysis of treatment efficacy. Am J Psychiatry 1994;151:1172–1180
15. American Sleep Disorders Association. International Classification of Sleep Disorders: Diagnostic and Coding Manual. Rochester, Minn: American Sleep Disorders Association; 1990
16. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision. Washington, DC: American Psychiatric Association; 2000
17. Salin-Pascual RJ, Roehrs TA, Merlotti LA, et al. Long-term study of the sleep of insomnia patients with sleep state misperception and other insomnia patients. Am J Psychiatry 1992;149:904–908
18. Morin CM, Espie CA. Insomnia: A Clinical Guide to Assessment and Treatment. New York, NY: Kluwer Academic/Plenum Publishers; 2003
19. World Health Organization. International Classification of Diseases, Tenth Revision. Geneva, Switzerland: World Health Organization; 1992
20. Morin CM, Gaulier B, Barry T, et al. Patients' acceptance of psychological and pharmacological therapies for insomnia. Sleep 1992; 15:302–305
21. Bootzin RR, Perlis ML. Nonpharmacologic treatments of insomnia. J Clin Psychiatry 1992;53(suppl, 6):37–41
22. Bootzin RR, Epstein D, Wood JM. Stimulus control instructions. In: Hauri PJ, ed. Case Studies in Insomnia. New York, NY: Plenum Medical Book Co; 1991:19–28
23. Spielman AJ, Saskin P, Thorpy MJ. Treatment of chronic insomnia by restriction of time in bed. Sleep 1987;10:45–56
24. Morin CM, Blais F, Savard J. Are changes in beliefs and attitudes about sleep related to sleep improvements in the treatment of insomnia? Behav Res Ther 2002;40:741–752
25. Zammit GK. Subjective ratings of the characteristics and sequelae of good and poor sleep in normals. J Clin Psychol 1988;44:123–130
26. Lichstein KL, Wilson NM, Noe SL, et al. Daytime sleepiness in insomnia: behavioral, biological and subjective indices. Sleep 1994;17: 693–702
27. Stepanski E, Zorick F, Roehrs T, et al. Daytime alertness in patients with chronic insomnia compared with asymptomatic control subjects. Sleep 1988;11:54–60
28. Sugerman JL, Stern JA, Walsh JK. Daytime alertness in subjective and objective insomnia: some preliminary findings. Biol Psychiatry 1985;20: 741–750
29. Coyle K, Watts FN. The factorial structure of sleep dissatisfaction. Behav Res Ther 1991;29:513–520
30. Morin CM, Hauri PJ, Espie CA, et al. Nonpharmacologic treatment of chronic insomnia: an American Academy of Sleep Medicine review. Sleep 1999;22:1134–1156
31. Espie CA, Inglis SJ, Tessier S, et al. The clinical effectiveness of cognitive behavioral therapy for chronic insomnia: implementation and evaluation of a sleep clinic in general medical practice. Behav Res Ther 2001;39:45–60
32. Edinger JD, Wohlgemuth WK, Radtke RA, et al. Cognitive behavioral therapy for treatment of chronic primary insomnia: a randomized controlled trial. JAMA 2001;285:1856–1864
33. Morin CM, Colecchi C, Stone J, et al. Behavioral and pharmacological therapies for late-life insomnia: a randomized clinical trial. JAMA 1999; 281:991–999
34. Smith MT, Perlis ML, Park A, et al. Comparative meta-analysis of pharmacotherapy and behavior therapy for persistent insomnia. Am J Psychiatry 2002;159:5–11
35. Currie SR, Wilson KG, Pontefract AJ, et al. Cognitive-behavioral treatment of insomnia secondary to chronic pain. J Consult Clin Psychol 2000;68:407–416
36. Nowell PD, Mazumdar S, Buysse DJ, et al. Benzodiazepines and zolpidem for chronic insomnia: a meta-analysis of treatment efficacy. JAMA 1997;278:2170–2177
37. Kupfer DJ, Reynolds CF. Management of insomnia. N Engl J Med 1997; 336:341–346
38. Lichstein KL, Wilson NM, Johnson CT. Psychological treatment of secondary insomnia. Psychol Aging 2000;15:232–240
39. Morin CM. Psychological and behavioral treatments for primary insomnia. In: Kryger M, Roth T, Dement W, eds. Principles and Practice of Sleep Medicine. 4th ed. Philadelphia, Pa: WB Saunders Co. In press